

**HYDRATION OF COLONIC INGESTA AND FECES IN HORSES
FED LARGE GRAIN MEALS OR TREATED WITH ENTERAL FLUID THERAPY,
SALINE CATHARTICS AND INTRAVENOUS FLUID THERAPY**

by

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Dissertation submitted to the Faculty of the Virginia Polytechnic Institute and
State University in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

IN

VETERINARY MEDICAL SCIENCES

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September 30, 2002
Blacksburg, Virginia

Key words: Equine, colic, gastrointestinal physiology, gastrointestinal disease.

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HYDRATION OF COLONIC INGESTA AND FECES IN HORSES FED LARGE GRAIN MEALS OR TREATED WITH ENTERAL FLUID THERAPY, SALINE CATHARTICS AND INTRAVENOUS FLUID THERAPY

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(ABSTRACT)

Systemic hydration, plasma electrolytes, ingesta and fecal hydration and gastrointestinal passage of cobalt (after CoEDTA administration via nasogastric tube) in horses fed large grain meals or treated with enteral fluid therapy, IV fluid therapy and enteral laxatives were investigated. In the first study, 0.9% NaCl (10 L/h/8h) was administered slowly via a small-bore nasogastric tube or as 10 L boluses via a large-bore nasogastric tube to four normal horses. In the other studies, horses with a right dorsal colon fistula were used. To create the right dorsal colon fistula, a cannula with 5 cm internal diameter was implanted 2 to 6 weeks after a right dorsal colopexy had been created. Six horses with the right dorsal colostomy were alternately used to test three feeding regimes for 48 h: 1- hay free choice; 2- hay free choice plus 4.5 kg of sweet feed twice daily after a period of 5 days of adaptation; 3- sudden change from hay to hay plus sweet feed. Seven horses with the right dorsal colostomy were alternately used to test 6 experimental conditions while fasted for 24 h: 1- control (no treatment), 2- enteral MgSO₄ (1 g/kg), 3- enteral Na₂SO₄ (1 g/kg), 4- IV lactated Ringer's solution (5 L/h/12 h), 5- enteral water (5 L/h/12 h), 6- enteral electrolyte solution (5 L/h/12 h). In the last study, four horses with the right dorsal colostomy were alternately treated with enteral electrolyte solution (10 L/h/6h) and enteral MgSO₄ (1 g/kg) plus IV fluid therapy (10 L/h/6h). Despite the administration regimen, enteral administration of 0.9% NaCl produced diarrhea, hypernatremia and hyperchloremia. Colostomy allowed serial collection of large ingesta samples. Grain ingestion did not change PCV or plasma protein, but affected plasma electrolytes and produced dehydration of ingesta and formation of frothy ingesta. Fasting delayed gastrointestinal transit. Enteral fluid therapy was the most effective treatment in promoting ingesta hydration. Enteral water, MgSO₄, Na₂SO₄, IV fluid therapy and enteral MgSO₄ plus IV fluid therapy were either ineffective in promoting ingesta hydration or produced marked plasma electrolyte imbalance. These findings support the use of enteral fluid therapy in horses with gastrointestinal impaction.

Dedication

To my parents

To my aunt Dorinha

To my brothers Luis, Paulo, Sergio and Eduardo and to my sister-in-law Eliana

To Brettania

Acknowledgments

Dr. Nathaniel White for his support and for his inspiring example of dedication to equine surgery and his continual quest for knowledge.

Dr. Mark Crisman, Daniel Ward and Dr. Lydia Donaldson who were always sources of good ideas, constructive criticism and friendly support.

The other members of my Advisory Committee: Dr. David Kronfeld, Dr. William Swecker, Dr. Michael Murray, Dr. Anthony Blikslager and Dr. Daniel Boon for their valuable contributions.

Barbara Wise, Athena Tilley, Carolyn Sink, Karen Perry, Hannah Anderson, John Strauss, and Delbert Jones for their assistance with laboratory work.

Shirley Johnson and Elizabeth Hullet for their many hours of enthusiastic volunteer work.

All other members of the Equine Medical Center who were supportive and willing to help.

Our sponsors: CNPq – Brasil, Grayson Jockey Club Research Foundation, the Animal Health and Disease and Hatch Grant, the Patricia Bonsall Stuart Award, Abbott Laboratories, Southern States Cooperative and Mila International.

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Chapter 1

Introduction

Despite the great progress during the last 3 decades, the information available on equine medicine is still limited when compared to human medicine and other fields of veterinary medicine. Though knowledge obtained in other species has been useful in equine medicine, this approach is limited by many factors such as differences in morphology and physiology. The fact that the horse is a monogastric herbivore is one of the main morphophysiological peculiarities of this species. Thus many aspects of gastrointestinal diseases in horses cannot be compared to what is seen in humans, carnivores or ruminants.

Although colic is among the main causes of death of domestic horses, many basic aspects of the etiopathogenesis and treatment of colic are still unknown. Anecdotal evidence and a few epidemiological and experimental studies have incriminated grain intake as a risk factor for gastrointestinal diseases, but the mechanisms to explain this association are not completely understood. Furthermore many aspects of the pathogenesis of common gastrointestinal diseases such as large colon impaction have not been investigated, and anecdotal information is still being used to guide treatment. Even routinely used treatments for colic and other diseases such as fluid therapy and laxatives have not been extensively studied in horses.

This dissertation presents some new insights on the etiopathogenesis and treatment of gastrointestinal disease in horses: it provides new information on the systemic and gastrointestinal effects of large grain meals and it proposes the use of enteral fluid therapy for large colon impaction and other diseases. The dissertation starts with a review of the literature on the association between feeding practices and colic, the effects of large grain meals on the equine gastrointestinal tract, the etiopathogenesis of gastrointestinal impaction, current treatments for large colon impaction and the principles of enteral fluid therapy. In chapter 3 the surgical technique and the design of the cannula used to create a large right dorsal colon fistula are described. Although a technique to create a small diameter colostomy had been described by others that type of fistula would not be suitable for collection of large samples of ingesta. Chapter 4 presents an experiment of the effects of large grain meals administered twice daily on ingesta and fecal hydration, plasma electrolytes, PCV and plasma protein. This study was conducted to assess the previously proposed theory that large meals can lead to ingesta dehydration and large colon impaction. In chapter 5, a study comparing enteral administration of 0.9% NaCl via nasogastric tubes of 2 different gauges is presented. Although large bore nasogastric tubes had been previously used for enteral fluid therapy, there was no information on whether small bore enteral feeding tubes would be appropriate for this use. Furthermore a controlled study of the administration of large volumes of 0.9% NaCl via nasogastric tube to horses had not been described. Chapter 6 presents a study comparing enteral administration of an electrolyte solution, enteral administration of water, enteral administration of saline cathartics (magnesium sulfate and sodium sulfate), and intravenous fluid therapy. The effects on ingesta hydration, fecal hydration, plasma electrolytes, PCV and plasma protein were assessed. Chapter 7 describes a study comparing enteral administration of an electrolyte solution with a currently recommended treatment for large colon impaction: magnesium sulfate via nasogastric tube plus IV fluid therapy. In chapter 8 the general conclusions of this series of studies are summarized.

Chapter 2

Literature Review

1- Equine Colic

Colic is a paroxysm of acute abdominal pain localized in a hollow organ and often caused by spasm, obstruction, or twisting (Merriam-Webster Medical Dictionary 1997). Gastrointestinal diseases are the most common causes of colic in horses, and in equine medicine, colic is almost synonymous with gastrointestinal disease (White 1990). However other diseases such as conditions of the liver and the genitourinary tract may also cause abdominal pain (Johnston et al. 1989, Laverty et al. 1992, Turner et al. 1993). Diseases of other systems distant from the abdominal cavity such as neurologic (e.g., rabies) and musculo-skeletal diseases (e.g., rhabdomyolysis) can also be associated with clinical signs that resemble colic (Harris 1991, Green et al. 1992). It is also important to emphasize that even limiting the term colic to gastrointestinal dysfunction, colic can not be considered as a single disease, but as a group of diseases.

Impact on Horse Welfare and on the Equine Industry

Colic has been reported to have a high incidence (from 3.5 to 26 colic cases/100 horse-years) (Uhlinger 1992, Kaneene et al. 1997, Tinker et al. 1997a, Hillyer et al. 2001, Traub-Dargatz et al. 2001), to be the leading group of diseases requiring veterinary care (Traub-Dargatz et al. 1991), to have a high mortality rate (6.3 to 15.3%) (Proudman 1991, Kaneene et al. 1997, Tinker et al. 1997a, Cohen et al. 1999, Hillyer et al. 2001, Traub-Dargatz et al. 2001), and to be the leading group of diseases causing death of domestic horses (17.5 to 28% of all deaths) (Tinker et al. 1997a, NAHMS 1998). Horses with a history of colic have an increased risk of other colic episodes, which has been suggested to result from damage to the gastrointestinal tract caused by previous colic or due to a sequela from gastrointestinal surgery (Baxter et al. 1989, Cohen et al. 1995, Reeves 1996, Schusser and White 1997, Tinker et al. 1997b, van den Boom and van der Velden 2001). Treatment of horses with colic is often expensive and the economic impact is considerable even when the horses survive (Traub-Dargatz et al. 2001). In many cases, due to the expense of the treatment, horses with considerable chance of recovery are subjected to euthanasia. Colic has also been found to be the second main cause of days of lost use (16.6%) after wounds, trauma and lameness combined (53.4%) (NAHMS 1998). In pregnant mares the losses due to abortion also have to be considered, since high incidences of post-colic abortion have been reported (Santschi et al. 1991, Boening and Leendertse 1993). Thus better understanding of the mechanisms involved in the pathogenesis of colic is needed in order to guide effective prophylactic measures and develop more effective therapies. Furthermore, considering the high cost of the current treatments, development of less expensive therapies may have a significant impact on the losses caused by colic.

Conclusions

Gastrointestinal diseases have a relatively high incidence in horses. These diseases often cause severe pain, and are frequently associated with significant derangements of essential physiological mechanisms, and may be fatal. Besides the concerns with horse welfare, colic is a cause of substantial financial losses for the equine industry. To establish effective prophylactic measures and to develop more effective and less expensive therapies more studies are needed.

2- Feeding Practices and Colic

The association between feeding practices and colic has been investigated in a series of large epidemiological studies (Cohen et al. 1995, Cohen and Peloso 1996, Reeves 1996, Tinker et al. 1997b, Cohen et al. 1999, Hudson et al. 2001). However these studies had several limitations: 1- All gastrointestinal diseases causing colic were analyzed together and cases of non-gastrointestinal diseases could not be excluded; 2- There were several potential sources of bias (e.g., recall bias) as acknowledged by the authors of those epidemiological studies; 3- Many times it was difficult to find a reasonable biologic explanation for the factors found to be associated with colic, suggesting that those factors might be just confounders. Furthermore it is difficult to compare the results of these studies because of the differences in the methods used, and conflicting results were obtained.

In a case control-study of 821 colic cases and 821 non-colic emergencies treated by veterinarians in the field, recent changes in diet were found to be associated with colic episodes. A change in diet within 2 weeks of the veterinary assistance had been reported in 19.7% of the colic cases and in only 4.9% of the non-colic cases. The other difference found to be significant was the presence of coastal grass hay in the diet of 79.2% of the horses with no history of previous colic, 87% of the horses with history of previous colic and 91.2% of horses with chronic intermittent colic. Although no association was found between feeding coastal grass hay and any specific type of colic, it was suggested that the lower digestibility of this type of hay may predispose horses to impactions (Cohen et al. 1995, Cohen and Peloso 1996).

In a case-control study of 406 colic cases and 406 non-colic cases referred to veterinary hospitals, horses with colic consumed less total non-roughage concentrate feed than control horses. According to the statistical model a 1 kg increase in the amount of non-roughage feed consumed was associated with a 12% reduction in the colic risk. Horses affected by colic consumed more whole-grain corn than control horses, and for each 1 kg increase in the amount of whole-grain corn consumed, the risk of colic increased over 3 fold (Reeves 1996).

In a one year prospective study on 31 horse farms with 1427 horses and 104 colic episodes, feeding larger amounts of concentrate was associated with a higher colic risk, while feeding whole grain reduced the colic risk. Changes in concentrate feeding amount or type and more than one change per year of hay were also identified as risk factors for colic (Tinker et al. 1997b).

In a case-control study of 1030 colic cases and 1030 non-colic emergencies treated by veterinarians in the field, access to pasture was found to reduce the risk of colic. Furthermore feeding hay other than coastal grass hay, Bermuda grass hay and alfalfa hay was significantly associated with colic, which was interpreted as the result of the lower digestibility of those other types of hay. Recent diet change (within 2 weeks) increased the risk of colic. Of the types of diet change, only change of hay was associated with colic (Cohen et al. 1999).

In a case-control study of 182 colic cases and 182 horses examined because of illnesses other than colic or for routine procedures, no pasture time or a recent decrease in acreage or pasture time and feeding grain were associated with an increased risk of colic. Feeding more than 2.7 kg of oats was associated with an increased risk of colic. Recent change (within 2 weeks) in the type of concentrate and abnormal feeding incidents (accidental overfeeding, irregular feeding times, feeding moldy feed and lack of access to water for 24 h) were also associated with an increased risk of colic. Feeding more than 2 flakes per day of hay other than coastal grass hay, Bermuda grass hay and alfalfa hay and feeding hay from round bales were associated with colic.

Changes in the amount of hay fed (increase or decrease), feeding a new batch of hay and recent (within 2 weeks) introduction of hay or grain was also associated with colic (Hudson et al. 2001).

Feeding practices and gastrointestinal diseases causing colic

Based on clinical evidence and a few experimental studies, associations between feeding practices and some gastrointestinal diseases that cause colic have been found. There is strong evidence that feeding alfalfa and bran is a risk factor for enterolithiasis. In a retrospective study of 715 cases of enterolithiasis in equids with reliable information about the amount of alfalfa in the diet, alfalfa represented more than 50% of the diet in 710 cases (99.3%) and was the sole feed in 472 cases (66%). Among the 688 equids with reliable information about the use of grain in the diet, grain was fed in 356 cases (51.7%) and bran was offered regularly in 197 cases (28.6%) (Hassel et al. 1999). Based on information on the chemical composition of the enteroliths and on experimental studies in fistulated horses, it has been suggested that the high magnesium and protein content of alfalfa and its ability to produce alkalization of colonic contents contribute to the formation of struvite, which is the predominant component of enteroliths in horses. The high phosphorus and magnesium concentrations of bran have also been suggested to predispose to struvite formation (Lloyd et al. 1987, Hintz et al. 1988, Hassel et al. 2001).

Several forms of gastrointestinal impaction have also been associated with feeding practices. For at least a few decades, it has been suggested that large intestine impaction is associated with ingestion of feed with high fiber content (Meagher 1972, Sellers and Lowe 1986, Collatos and Romano 1993, Hudson et al. 2001). This theory is supported by reports of cases of large colon impaction in horses fed mature grass (Lopes et al. 1999) or poor quality hay (Pugh and Thompson 1992). In a retrospective study of 229 cases of colic it was found that pelleted roughage had been fed to a larger number of horses with large colon impaction (24%) than to all other horses (8.2%), which was suggested to be the result of reduced digestibility of pelleted hay (Morris et al. 1989). Feeding Bermuda grass hay, which is known to have high fiber content, has been repeatedly associated with ileal impaction (Embertson et al. 1985, Parks et al. 1989, Pugh and Thompson 1992, Hanson et al. 1998, Little and Blikslager 2002). Ingestion of cracked corn has been reported to form a duodenal impaction in 4 horses (Bohanon 1988). Reports of a case of gastric impaction caused by sugar beet pulp (Jones et al. 1972) and two cases of gastric impaction after ingestion of straw or coarse grass respectively have been published (Owen et al. 1987). Thus, the common feature of the feeding practices associated with gastrointestinal impaction is the ingestion of large particles of poorly digestible feed, which is likely to affect the flow of ingesta through the gastrointestinal tract.

Primary gas colic is known to occur after ingestion of large amounts of soluble carbohydrates. When rapidly fermentable substrates reach the gastrointestinal lumen, the gastrointestinal flora can produce excessive amounts of gas leading to pronounced distention of the stomach and/or large intestine (Becht 1983, Byars 1983, Baker et al. 1992).

Large colon displacement and volvulus are also thought to be related to ingestion of large amounts of soluble carbohydrates and abrupt changes in type or amount of feed (Fischer and Meagher 1986, Snyder et al. 1988). These feeding practices can cause excessive production of gas in the gastrointestinal tract and abnormal plasma electrolyte concentration leading to abnormal motility patterns and displacement of the large colon (Snyder et al. 1988). Although definitive clinical evidence or experimental data to support this hypothesis have not been

published, in a retrospective study of 229 cases of colic, pelleted grain had been fed to a higher proportion of horses with large colon displacement and torsions (23.1%) than with small intestine strangulated obstruction (4.5%) (Morris et al. 1989). Since grain is rich in soluble carbohydrate and pelleted feed passes faster through the gastrointestinal tract (Hintz and Loy 1966), it was suggested that the large amount of soluble carbohydrate reaching the large intestine when the horses are fed pelleted grain can lead to tympany and large colon displacement (Morris et al. 1989).

Gastric ulcers have also been associated with feeding practices. Experiments in horses have shown that when diets rich in soluble carbohydrates were fed, postprandial gastrin secretion was higher than when hay was fed (Smyth et al. 1989). In another experimental study, 14 out of 31 horses fed grain had lesions in the gastric squamous mucosa, while no horse out of the 25 horses fed just hay had any gastric lesion (Coenen 1990, Coenen 1992). These findings suggest that feeding grain may produce increased postprandial acidity in the stomach, which is known to damage the gastric squamous mucosa (Murray 1999). It has also been reported that lower gastric pH and ulceration of the squamous epithelial mucosa can be produced by feed deprivation in contrast to higher pH and absence or even healing of ulcers produced by feed deprivation when the horses were offered free choice hay. These findings suggest that intermittent availability of feed may cause ulcers in the squamous mucosa of the stomach (Murray and Schusser 1993, Murray and Eichorn 1996). Another experiment has shown that despite the higher concentration of volatile fatty acids in the stomach of horses fed alfalfa hay plus grain, gastric pH was higher and the number and severity of lesions in the squamous mucosa were lower than when grass hay was fed. The authors suggested that the alfalfa-grain diet may have a better buffering effect due to the higher calcium and protein content compared to the grass hay (Nadeau et al. 2000).

Other indications of the association between feeding practices and gastrointestinal disease were found in the retrospective study reported by Morris et al. (1989): sweet feed had been fed to a higher proportion of horses with small intestine strangulating obstruction (86.4%) than with ileal impaction (58.8%); Bermuda grass hay had been fed to a smaller proportion of horses with proximal enteritis (37.5%) and small colon obstruction (28.6%) than to all other horses (66.7% and 65.8% respectively); mixed legume-grass hay had been fed to a higher proportion of horses with proximal enteritis (18.8%) than with other types of colic; alfalfa hay had been fed to a smaller number of horses with small intestine obstruction (4.5%) and to a larger number of horses with small colon obstruction (71.4%) than to horses with other types of colic (26.6% and 23% respectively). Mechanisms by which these feeding practices could cause colic have not been described.

Conclusions

Despite the indications that feeding practices are important risk factors for colic, information about the mechanisms involved is scarce. To establish effective prophylactic measures the effects of feeding practices on equine gastrointestinal physiology need to be better investigated.

3- Grain ingestion, gastrointestinal function and water balance

Grain ingestion has long been recognized as a cause of gastrointestinal dysfunction in horses (Hintz 1984). The fact that horses evolved as grazers explains why large grain meals are not tolerated by the equine gastrointestinal tract. Grains are rich in rapidly fermentable carbohydrate and may cause digestive problems because the equine gastrointestinal tract evolved to digest plant material with high fiber content (Clarke et al. 1990a). Recent epidemiological studies have confirmed that ingestion of grain is a major risk factor for gastrointestinal diseases affecting domestic horses (Reeves 1996, Tinker et al. 1997b, Hudson et al. 2001) but the mechanisms involved in grain-induced gastrointestinal dysfunction have not been completely elucidated.

Digestion of hydrolyzable carbohydrates in the equine gastrointestinal tract

Hydrolyzable carbohydrates are the fraction of plant carbohydrates that can be digested by the enzymes of the equine gastrointestinal tract. This fraction includes hexoses, disaccharides, digestible starch and some oligosaccharides. About one third of the non-structural carbohydrates in forages and from one-half to all the non-structural carbohydrates in concentrates are hydrolyzable carbohydrates (Hoffman et al. 2001).

Saliva does not seem to be important for carbohydrate digestion in horses, since in this species, amylase content in saliva is minimal. Hydrolysis of large carbohydrate molecules is carried out almost exclusively by pancreatic amylase. In horses amylase concentration in the pancreatic secretion is low, but is compensated for by the large volume of secretion (Alexander and Hickson 1970). The brush border enzymes of the small intestine complete carbohydrate hydrolysis to produce monosaccharides, which are absorbed by the epithelium of the small intestine (Roberts 1975).

Hydrolyzable carbohydrates can also be fermented by the gastrointestinal flora to produce short chain fatty acids (Stillions et al. 1970, Hintz et al. 1971b, Hintz et al. 1971a). Although microorganisms can be found throughout the equine gastrointestinal tract, transit through the stomach and small intestine is fast and carbohydrate fermentation occurs mainly in the large intestine (Alexander and Benzie 1951, Argenzio et al. 1974a, Argenzio et al. 1974b, Mackie and Wilkins 1988). Not only fiber and other resistant carbohydrates but also hydrolyzable carbohydrates that escape digestion in the small intestine are fermented in the large intestine (Stillions et al. 1970, Hintz et al. 1971b, Hintz et al. 1971a, Massey et al. 1985).

The amount of hydrolyzable carbohydrates that reaches the large intestine varies depending on diet content, transit time through the small intestine and the availability and concentration of enzymes (Massey et al. 1985, Meyer et al. 1993, Meyer et al. 1995, Cuddeford 2001). Although considerable individual variation exists (Meyer et al. 1995), it has been estimated that the limit for starch digestion in the small intestine is about 0.4% of body weight per meal (Potter et al. 1992). The amount of carbohydrate, as well as carbohydrate composition and processing, and the presence of other components in the diet will determine the extent of carbohydrate digestion in the small intestine (Householder et al. 1977, Meyer et al. 1993, Meyer et al. 1995, Kienzle et al. 1997).

Effects of grain ingestion on the gastrointestinal microbiota and pH

The gastrointestinal tract of normal horses constitutes a unique ecosystem inhabited by a large number and variety of microorganisms (Kern et al. 1974, Garner et al. 1978, Orpin 1981, Bonhomme-Florentin 1988, Gold et al. 1988, Mackie and Wilkins 1988, Lin and Stahl 1995, Julliand et al. 1999). The resident microbiota has an important role in digestion and is essential for the health of the gastrointestinal tract. As in any ecosystem the environmental conditions and the availability of nutrients are main determinants of the composition of the population. Thus diet can profoundly affect the gastrointestinal microbiota and, consequently, concentration of products of microbial fermentation (Buddington and Weiher 1999).

Several studies have been conducted to assess the effects of grain ingestion on the equine gastrointestinal microbiota. In one of the first studies, ponies with cecal fistulas were fed 4 diets (2% of body weight / day): 1) timothy hay, 2) 75% timothy hay + 25% oats, 3) clover hay and 4) 75% clover hay + 25% oats. The ponies were fed 2 meals / day, had free access to water and salt mixture, and had 3 to 4 weeks to adapt to the diet. Samples were collected from the cecum 3 h after the morning meal. Diets containing oats produced a significant increase in the number of bacteria, number of viable bacteria and concentration of bacterial DNA and a decrease in the number of Gram negative rods isolated from cecal fluid. The only change seen in volatile fatty acids was an increase in the molar concentration of acetic, propionic and valeric acids when oats and clover were fed. Feeding oats also produced an increase in the number of a species of protozoa (*Blepharocorys uncinata*), but did not produce any change in pH, number of cellulolytic bacteria or total number of protozoa (Kern et al. 1973).

In another study using ponies, the effects of a high-fiber-low-protein diet containing 3% urea and a hay-grain commercial diet were compared after an adaptation period of 6 weeks. Both diets were fed twice daily and the ponies had been trained to eat their meal within 1 h. The hay-grain diet produced higher concentrations of volatile fatty acids and lactate in the gastrointestinal content, and this difference was greatest in the large colon. The hay-grain diet also produced higher gastric pH 8 h and 12 h post-feeding (Argenzio et al. 1974b).

In a study using cecal-fistulated horses fed twice daily, the effects of a 100% grain diet were compared to the effects of a 100% hay diet after a period of 3 days for adaptation to the diet. The pH of the cecal fluid was decreased 4, 5 and 6 h after ingestion of 6 kg of sweet feed (mean pH ranging from 6.12 to 6.43) compared to horses fed 8 kg of mixed grass-legume hay (mean pH ranging from 6.87 to 6.92). Sweet feed also produced a decrease in the molar percentage of acetate and an increase in the molar percentage of propionate and lactate in the cecal liquid. Furthermore intracecal administration of sodium bicarbonate prevented at least in part the changes in pH and volatile fatty acid concentration seen when sweet feed was offered. Changes produced by the diet were interpreted as the result of changes on number and activity of gastrointestinal microorganisms (Willard et al. 1977).

In another study using horses with a cecal fistula, 17.6 g/kg of body weight of a meal rich in hydrolyzable carbohydrates (85% corn starch and 15% wood cellulose flour) administered via nasogastric tube produced significant changes in cecal fluid microbial flora and pH. There was a significant increase in the number of *Lactobacillus* sp. and a significant decrease in *Enterobacteriaceae* sp., aerobic and anaerobic *Streptococcus* sp. and in pH 8 h after carbohydrate administration. Twenty four hours after carbohydrate administration, the number of *Lactobacillus* sp. was still high and the number of *Enterobacteriaceae* sp. was still low, while the number of *Streptococcus* sp. were similar to pre-treatment values. Further decrease in pH was

also observed 24 h after carbohydrate overload. These findings were interpreted as the result of increased production of lactic acid by the microorganisms that predominated following carbohydrate overload leading to rapid decline in pH and death of large numbers of Gram negative bacteria (e.g., *Enterobacteriaceae* sp.) (Garner et al. 1978). In another experiment using the same model (horses with cecal fistulas receiving 17.6 g/kg of body weight of a meal composed of 85% corn starch and 15% wood cellulose flour via nasogastric tube) an increase in the concentration of endotoxin in the cecal fluid was detected (Moore et al. 1979).

Subsequently a study was conducted to assess the effects of abrupt change to a concentrate diet (8.4 g/kg of body weight of a meal composed of 86.7% ground corn and 13.3% soybean) in a pony with a cecal fistula and adapted for 8 weeks to chopped alfalfa hay. When concentrate was fed, the total number of anaerobic bacteria and the number of organisms growing in selective starch medium increased, while the number of organisms growing in selective soluble fiber medium and the total number and the number of species of protozoan decreased. Seven days after the pony was started on the concentrate diet, the number of bacteria growing on lactate medium increased. Minimum pH readings of the cecal fluid were 6.4 and 5.8 recorded when alfalfa and grain respectively were fed, and the nadir was observed 6 h post-feeding. Dry-matter content of cecal ingesta had a diurnal pattern of variation, which was basically the inverse of the pH curve. When forage was fed the volume of the cecal liquid ranged from 1.6 to 3.4 liters (mean, 2.2 liters) and when grain was fed the volume of the cecal liquid ranged from 0.6 to 8.6 (mean, 3.9 liters). The interpretation of these findings was similar to what has been described for ruminants after sudden change to a grain diet: An immediate growth of lactate producers resulted in an immediate decrease in pH, which was followed by a slower increase in the number of lactate utilizers (Goodson et al. 1988).

In another study in ponies with fistulas in the cecum and colon fed once daily and treated with DSS to remove hindgut protozoa, a high concentrate diet (60% alfalfa hay and 40% concentrate) produced an increase in total bacterial concentrations in the colon (Moore and Dehority 1993).

In a study with Standardbred horses where several biochemical properties of feces were analyzed, it was found that horses in training had higher urobilinogen concentration in feces than horses that were not in training. Since horses in training were fed 5 kg of oats daily while the others were fed only 0.5 kg of oats daily, an association between diet and the microbial synthesis of urobilinogen was suggested. The authors also suggested that the difference in fecal urobilinogen should be attributed at least in part due to the different level of activity (Collinder et al. 2000).

Recently, studies have been conducted with ponies with fistulas in the cecum and right ventral colon to compare a 100% hay diet to a hay+barley diet fed twice daily. Abrupt incorporation of barley produced an increase in the number of total anaerobic bacteria, *Lactobacillus* sp., and *Streptococcus* sp. 29 hours after feeding. An increase in lactate concentration and a decrease in the (acetate+butyrate)/propionate ratio were also observed, which were not followed by a significant change in pH. Some of these changes were already present 5 h after the incorporation of barley to the diet. The authors acknowledged that the amount of soluble carbohydrates was not as high as reported in previous studies which may explain why grain produced less pronounced effects than previously reported: the amount of starch fed per meal did not exceed 2.3 g/kg of body weight per meal, which is under the limit of 4 g/kg of body weight recommended to prevent digestive dysfunction (Fombelle et al. 2001). Changes in the microorganisms of the large intestine produced by grain ingestion were also seen

after a period of 14 days for adaptation to the diets. In the cecum and colon the total number of bacteria, lactate-utilizing bacteria, *Lactobacillus* sp. and *Streptococcus* sp. increased while the number of cellulolytic bacteria decreased. Although total volatile fatty acid concentration did not change, the (acetate+butyrate)/propionate ratio and pH decreased when grain was fed (Julliand et al. 2001).

The effects of a 100% hay diet and a hay+barley diet twice daily on the digestive efficiency were studied in normal ponies. After an adaptation period of 21 days, it was observed that feeding barley increased the digestibility of organic matter and decreased the digestibility of fiber. It was concluded that the changes in the microbial ecosystem contributed to the lower digestibility of fiber when grain was fed (Drogoul et al. 2001).

Effects of grain ingestion on the mucosal barrier

It has been demonstrated that ingestion of large amounts of grain can disrupt the mucosal barrier. In a study of four horses with cecal fistulas, administration of 17.6 g/kg of body weight of a meal rich in soluble carbohydrates (85% corn starch and 15% wood cellulose flour) via nasogastric tube caused damage to cecal mucosa identified under electron microscopy. Mucosal damage was thought to be caused by the increase in intraluminal acidity produced by carbohydrate overload (Krueger et al. 1986). The same model of carbohydrate overload produced clinical signs of endotoxemia (Garner et al. 1975, Garner et al. 1978, Moore et al. 1979) and an increase in plasma endotoxin concentration (Sprouse et al. 1987) indicating that the mucosal barrier had been damaged and endotoxin had been absorbed. Thus when extremely large amounts of starch is consumed, the end products of starch digestion can produce significant intraluminal acidosis, increase the concentration of endotoxins and disrupt the gastrointestinal mucosa leading to endotoxemia.

Effects of grain ingestion on gas production and accumulation in the gastrointestinal tract

Gas accumulation within the gastrointestinal tract after ingestion of large amounts of soluble carbohydrates can produce gastric dilation (Becht 1983, Baker et al. 1992) and tympany of the large intestine in horses (Byars 1983, Baker et al. 1992). It has also been suggested that gas within the large intestine can reduce the weight of an intestinal segment and lead to displacements or volvulus in horses (Snyder et al. 1988). However there is no report of a study investigating the mechanisms of grain induced bloat in horses.

In ruminants several aspects of the etiopathogenesis of bloat secondary to grain ingestion have been unraveled (Cheng et al. 1998, Nagaraja et al. 1998). When large amounts of soluble carbohydrates reach the rumen, rapid fermentation produces large amounts of gas (Lippke et al. 1972). Due to microbial synthesis of mucopolysaccharides and other substances the viscosity of the ruminal fluid is increased (Cheng and Hironaka 1973, Cheng et al. 1976). The result is the formation of large amounts of stable froth, which cause an increase in intraruminal pressure and rumen distention (Lippke et al. 1972). In ruminants, froth formation compromises eructation, which is the main mechanism to release gas produced by fermentation in the rumen (Cheng et al. 1998). Inhibition of ruminal motility due to ruminal acidosis has also been mentioned as a factor on the pathogenesis of bloat (Nagaraja et al. 1998). There are indications that individual

variability also plays a role and breed predisposition to bloat has been found. Factors such as anatomical differences in the rumen, salivary production and appetite may explain why some individuals are predisposed (Cheng et al. 1998).

It is possible that mechanisms similar to these described for ruminants with bloat may play a role in gastrointestinal dysfunction in horses after grain overload, although there are several physiologic differences (e.g., horses are hindgut fermenters, horses do not eliminate gas by eructation). The equine cecum is effective at handling gas, but excessive amounts of gas can overwhelm these physiological mechanisms (Cottrell et al. 1998). Furthermore horses eating grain can have intraluminal acidosis (Willard et al. 1977, Garner et al. 1978) which may compromise gastrointestinal motility as seen in ruminants. Considering that in ruminants and horses the gastrointestinal microbiota and its response to grain overload are similar, froth is also likely to form in horses after excessive grain ingestion. However froth formation in the gastrointestinal tract of horses after ingestion of large amounts of grain has not been documented.

Effects of grain ingestion on gastrointestinal transit

There is evidence that grain ingestion delays gastrointestinal transit. In a study with ponies comparing high-fiber and low-fiber diets after a 10 day adaptation period and using chromium-mordanted fiber, higher fiber content resulted in higher DM intake and faster rate of passage of chromium (Yoder et al. 1997). In another study, gastrointestinal transit was evaluated in horses alternately fed an all-forage diet (alfalfa cubes and alfalfa-grass hay) or a mixture of forage and grain (alfalfa cubes, alfalfa-grass hay and grain) for periods of 4 weeks. Faster rate of passage of ytterbium (after oral administration of ytterbium chloride) was observed when the horses were fed the all-forage diet (Pagan et al. 1998). In a study with ponies, the effects of a 100% hay diet and a hay+barley diet twice daily on the rate of ingesta passage after an adaptation period of 21 days were compared. Ponies were fed hay labeled with europium and barley labeled with ytterbium. A dose of CrEDTA was administered via nasogastric tube. It was observed that the transit through the gastrointestinal tract was slower when grain was fed as evidenced by the increased mean retention time of all markers (chromium, ytterbium and europium) (Drogoul et al. 2001). In all articles, the authors concluded that the faster rate of passage with diets with higher fiber content was a consequence of the increased dry matter intake (Yoder et al. 1997, Pagan et al. 1998, Drogoul et al. 2001). In one article, it was also suggested that higher saliva production and water intake seen when only forage is fed may have contributed to the speedier gastrointestinal transit (Pagan et al. 1998). However diet composition affects gastric emptying (Sojka and Cantwell 1989, Read 1994), which may contribute to slow gastrointestinal transit when grain is fed.

Effects of grain ingestion on water consumption

Several studies have shown that horses eating grain tend to consume less water. In a experiment with Standardbred horses, the average water consumption when the horses were fed all-roughage diets was greater (mean, 31.4 kg/day) than when the horses were fed a hay-grain diet (mean, 17.5 kg/day). The author also observed that water consumption was highly correlated

to the dry matter intake and that the water consumed / kg of dry matter was higher for hay (mean, 3.6 kg/kg DM) than for grain (mean, 2.6 kg/kg DM). Horses had free access to metered water bowls and the environmental temperature ranged from 3 to 15 C (Fonnesbeck 1968).

In a study conducted with ponies fed grain and water ad lib, it was found that adding sawdust to the grain resulted in an increase in water intake from about 8.7 to more than 11 kg/day. However there was no correlation between the proportion of sawdust added and the amount of water consumed. Environmental temperature and the method to measure water consumption were not mentioned (Sufit et al. 1985).

Similar results were obtained in another study where water consumption in ponies and horses eating hay or grain pellets were compared. Environmental temperature ranged from 15 to 20 C. Water was offered in buckets 2 to 4 times daily, but the method to measure consumption was not mentioned. Horses eating hay drank more water (mean, 5.0 liters/100 kg BW/day) than horses eating pelleted grain (mean, 2.7 liters/100 kg BW/day). In addition, the volume of water consumed per kg of dry matter intake was larger when hay was fed (mean, 3.2 liters/kg DM) than when pelleted grain was fed (mean, 2.0 liters/kg DM) and water consumption was positively correlated to fiber intake (Cymbaluk 1989).

In two other studies, while horses were being prepared for an exercise test, water consumption was higher when hay was fed (means, 17.8 and 16.35 kg) than when hay and grain was fed (means, 10.1 and 11.03 kg) (Danielsen et al. 1995).

In a study with ponies comparing high-fiber and low-fiber diets there was no difference in water consumption. In this study, weighing the water buckets was the method used to measure water consumption. The authors did not comment on the discrepancy in relation to other studies (Yoder et al. 1997).

The observation that low fiber diets result in less water consumption was corroborated in another study where horses consumed more water when fed a high-fiber diet (mean, 3.3 l/kg dry matter) than when fed a low-fiber diet (mean, 2.6 l/kg dry matter). The horses had free access to water buckets, but the method to measure water consumption and the environmental temperature were not mentioned (Warren et al. 1999).

Effects of grain ingestion on ingesta osmolality and ionic composition

Ingesta osmolality and ionic composition may be affected by grain ingestion. In a study using ponies, the effects of a high-fiber-low-protein diet containing 3% urea and a hay-grain commercial diet were compared after an adaptation period of 6 weeks. Both diets were fed twice daily and the ponies had been trained to eat their meal within 1 h. The hay-grain diet produced higher osmolality and more variation in osmolality than the high-fiber diet (Argenzio et al. 1974a). The hay-grain diet also produced post-prandial changes in ionic composition of ingesta which were not seen with the high-fiber diet (Argenzio and Stevens 1975).

Effects of grain ingestion on ingesta and fecal water content

Ingesta and fecal water content may be affected by grain ingestion. In one study the hydration of ingesta was compared in 3 groups of 4 mature ponies fed one of the following isocaloric diets: alfalfa, alfalfa-grain (3:2) and alfalfa-grain (1:4). Water content in the small

colon was significantly lower when grain was fed. However, feeding grain did not affect water content of ingesta in any other gastrointestinal segment (stomach, small intestine, cecum or large colon) (Hintz et al. 1971a). In another study, fecal hydration was lower when the horses were fed the low-fiber diet (mean, 73.6%) than when the horses were fed the high-fiber diet (mean, 81.7%). The horses had free access to water buckets, but the environmental temperature and the method to measure water consumption were not mentioned (Warren et al. 1999).

Effects of grain ingestion on systemic hydration

The gastrointestinal tract may function as a reservoir of water and electrolytes in exercising horses and that the fiber content of the diet can affect the capacity of this water reservoir. In a study with Thoroughbred horses submitted to endurance exercise on a treadmill, feeding only hay the night before the exercise resulted in lower plasma protein and in higher plasma potassium than feeding hay and grain (Danielsen et al. 1995). However in a study comparing changes in plasma volume in horses 4 h after furosemide injection, no difference could be seen when the effect of diets with low and high fiber content were compared (Warren et al. 1999).

Other effects of grain ingestion

Soft feces (from pasty to liquid), increased abdominal sounds and abdominal distention were observed when ponies and horses were treated with 17.6 g/kg of body weight of a meal composed of 85% corn starch and 15% wood cellulose flour via nasogastric tube (Robinson et al. 1976). Soft and acidic feces, an increase in plasma lactate and signs of laminitis were observed in Standardbred horses receiving high-starch pelleted feed (85% ground maize and 13% soybean meal) ad lib. These changes were not seen when the antibiotic virginiamycin was added to the meal, although the horses ate less grain during the first 3 days the antibiotic was added. These findings were interpreted as the result of the reduced feed intake and the prevention of growth of lactate-producing bacteria by the antibiotic (Rowe et al. 1994).

Conclusion

Although information available in horses is limited when compared with what has been studied in ruminants, it is evident that grain ingestion can produce significant changes in gastrointestinal function. Feeds rich in fiber keep the gastrointestinal microbiota healthy with a high cellulolytic activity, whereas feeds rich in rapidly fermentable carbohydrates lead to fast growth of the population of starch utilizing microorganisms and decline of the cellulolytic flora. These changes can be tolerated up to a certain limit, although extreme changes can lead to severe gastrointestinal dysfunction.

Although the association between grain ingestion and colic has long been identified, the mechanisms involved in grain induced gastrointestinal dysfunction have not been completely unraveled. Keeping the horses on pasture under more natural conditions is likely effective for the prophylaxis of many gastrointestinal diseases, but this approach is usually limited by economic

and logistic factors. Thus in order to prevent colic in domestic horses better understanding of the changes in gastrointestinal function produced by the current feeding practices is necessary.

4- Episodic feeding, gastrointestinal function and water balance

There is evidence that not only the type and amount of feed ingested, but also the frequency and size of the meals can affect gastrointestinal function in horses. Feeding a few large meals per day differs remarkably from the condition in which the horse evolved: having continuous access to grass. Thus episodic feeding is thought to predispose to gastrointestinal dysfunction (Clarke et al. 1990a).

Effects on water balance

In the first large experiment investigating the effects of episodic feeding on water balance, two different diets were fed twice daily to two groups of ponies trained to eat their meal within 1 h. Both a high-fiber-low-protein diet containing 3% urea and a hay-grain commercial diet produced post-prandial changes in the ionic composition and osmolality of ingesta. These changes were thought to be associated with bouts of gastrointestinal secretion and absorption after ingestion of the meal (Argenzio and Stevens 1975).

Recent studies have shown that passage of fluids into the gastrointestinal tract after large meals produces systemic dehydration. The effects of a large meal on systemic hydration were first demonstrated in an experiment where 3 meals / day were fed to horses: 1.8 kg of a complete cube diet was fed at 9:15 and 12:15 and 2.7 kg of the same diet plus 5.5 kg of hay was fed at 16:30. Marked post-prandial dehydration and electrolyte imbalance (15.8% increase in hematocrit, a 12.2% increase in plasma protein and a 16.6% decrease in plasma potassium) were seen after the large meal (cube diet plus hay), but not after the other meals. For plasma protein and potassium the maximal deviation occurred 1 h after the large meal was offered and a return to pre-feeding values was seen 2 h later while the horses were still eating. These observations were interpreted as the result of salivation while the horses were eating the large meal (Kerr and Snow 1982).

The first experiment comparing a large meal with multiple small meals feeding regimen was conducted in ponies fed a complete pelleted diet. The same amount of feed was offered as a single 1 h meal or divided into 6 equal meals per day. There was an increase of 10.9% in plasma protein and 2.1% in osmolality after the single meal, while only an increase of 3.8% was seen in plasma protein after one of the six daily meals. The peak of the plasma protein was seen 20 minutes after the start of the single meal and the peak of the osmolality was seen 40 minutes later. Plasma protein returned to pre-feeding values 30 minutes after the end of the meal, while plasma osmolality was still high when the horses were fed the large meal. These findings were thought to be the result of post-prandial dehydration due to saliva production (Youket et al. 1985).

Evidence of post-prandial dehydration after a large meal was also seen in another experiment comparing a hay-grain pelleted diet fed either 6 times daily (every 4 h) or once daily. Horses were allowed to eat for only 2 h when in the single meal regimen, which limited consumption to about 4 kg (63% of the allotted mean of 6.3 kg). A single large meal produced an increase in plasma renin 0.5, 1 and 3 h after the onset of feeding, plasma aldosterone 3, 5, and 7 h after the onset of feeding, plasma protein 0.5 and 1 h after the onset of feeding and hematocrit at 5 and 7 h after the onset of feeding. No change in hematocrit or plasma renin or protein was observed with multiple feedings. Only a trend towards increased aldosterone concentration in the

early morning was seen with this feeding regimen. These findings suggest that feeding a single large meal caused post-prandial dehydration and activation of the renin-angiotensin-aldosterone system. Based on the changes in plasma protein it was estimated that a single meal resulted in a plasma volume loss of about 10 to 13% (Clarke et al. 1988).

Similar results were obtained in another study investigating the effect of meal size on hydration status in ponies fed complete pelleted feed. Although feeding frequency did not affect water intake, the single daily 1 h meal produced a significant increase in plasma protein, hematocrit and osmolality, but only osmolality did not return to pre-feeding levels 1 h after the end of the meal. A smaller meal (one sixth of the daily ration fed every 4 h) produced less pronounced increase in plasma protein, hematocrit and osmolality. After the large meal plasma protein increased by about 10.2% and after the small meal the increase was about 3.6%. The authors concluded that post-prandial dehydration due to secretion by the upper alimentary tract was the explanation for the results (Houpt et al. 1988).

The hypothesis of post-prandial dehydration after a large meal was confirmed by another experiment where ponies were submitted to 2 feeding regimens: multiple small meals regimen (maintenance pelleted hay-grain diet fed in 12 equal meals every 2 h) or 2 large meals regimen (maintenance pelleted hay-grain diet fed in 2 equal meals every 12 h over a 1 h feeding period). Using the indocyanine green dilution technique, post-prandial reduction of plasma volume of about 15% was observed with the large meal. This change was observed as early as at the end of the 1 h feeding period, and plasma volume returned to the pre-feeding value 2 h later. Plasma volume reduction was seen again 6 h after the onset of feeding, but the change was less pronounced. Increase in plasma protein (by 12%) and aldosterone (by 104%) was also seen 1 h and 3 h after the onset of feeding respectively. Post-prandial decrease in urinary sodium clearance and increase in urinary potassium clearance were also observed and were interpreted as the response to the increase in plasma aldosterone concentration. According to the authors, the plasma volume reductions seen at end of the feeding of the large meal and 6 h after the onset of feeding were likely to be caused by secretion by the upper alimentary tract and large intestine respectively (Clarke et al. 1990b).

Recently, signs of post-prandial dehydration were reported in athletic horses fed about 7.4 kg of grass hay and 4.1 kg of grain mix divided in 2 equal meals every 12 h. There was an immediate increase in plasma protein of 3 to 6% and an increase in osmolality of 2%. These changes were not observed when the same amount of feed was divided into 6 equal meals every 4 h. Both plasma protein and osmolality returned to pre-feeding levels within 1 h after the onset of feeding. However there was no effect of feeding frequency on hematocrit, plasma aldosterone concentration, water intake, fecal output and fecal hydration. Furthermore feeding frequency did not affect any variable during or after exercise. The less pronounced post-prandial dehydration observed in this experiment when compared with other studies was attributed to the slower consumption of feed in this study, since the horses received more feed and did not have a time limit to eat and feed was not pelleted (Jansson and Dahlborn 1999).

Effects on digestibility and rate of passage

In an experiment with ponies allowed to consume their single daily meal in 1 h or fed small meals every 6 h, there was no effect of feeding frequency on rate of passage of chromic oxide or digestibility of fiber and protein (Houpt et al. 1988). These findings were corroborated

by another study in athletic horses fed about 7.4 kg of grass hay and 4.1 kg of grain mix divided in 2 equal meals every 12 h. This feeding regimen did not affect fecal output or fecal hydration when compared to what was seen when the horses were fed the same amount of feed divided into 6 equal meals every 4 h (Jansson and Dahlborn 1999).

Conclusions

Although post-prandial dehydration has been consistently produced in several studies with horses, the clinical significance of this phenomenon has not been documented. It has to be emphasized that even with extremely artificial feeding protocols (e.g., a single 1 h meal per day) only short term dehydration (2 or 3 h) could be produced. Furthermore large meal regimes have not been shown to affect gastrointestinal transit. Thus it is not clear if post-prandial dehydration can cause significant ingesta dehydration and gastrointestinal impaction.

5- Epidemiological aspects and etiopathogenesis of gastrointestinal impaction in horses

Impaction is defined as “lodgment or an instance of lodgment of something (such as a tooth or feces) in a body passage or cavity” (Merriam-Webster Medical Dictionary 1997). Based on this definition gastrointestinal impaction could result from any ingested material including foreign bodies or even concretions formed in the gastrointestinal lumen (e.g., enteroliths) or helminthes. Although the mechanisms that result in obstruction may be similar despite the cause of the impaction, only impactions formed by ingested material will be discussed in this text.

Incidence and fatality of gastrointestinal impaction

Impactions are among the most common gastrointestinal diseases in horses and the most common causes of equine colic. The fatality rate depends on a series of factors such as the anatomical location and etiology of the impaction (Table 2.1).

Etiopathogenesis

Gastrointestinal impaction is a disturbance of ingesta flow. The major factors that determine ingesta flow and may be involved in gastrointestinal obstruction were illustrated in the study conducted by Morel et al. (1990). In this study, the correlation between intestinal flow, intraluminal pressure, intestinal diameter and ingesta viscosity was investigated using segments of the small intestine of rabbits and humans. Gastrointestinal flow was maintained by a mean pressure of 3.4 cm H₂O, which is produced in vivo by contractions of the gastrointestinal muscles. When segments of small intestine were connected to a pump, reduction of the intestinal lumen down to 60% of the normal diameter did not affect flow or intraluminal pressure. However further luminal reduction led to an exponential decrease in flow or increase in the pressure required to maintain the original flow. When the diameter was reduced to about 30-35% of the original diameter, flow was stopped despite an increase in pressure. In an attempt to maintain flow, pressure was raised exponentially up to a point where rupture of the proximal segment of the intestine occurred. Another important observation was the increase in the critical diameters of the intestine produced by an increase in the viscosity of the fluid pumped through the intestinal segments.

Although no similar study using segments of the equine gastrointestinal tract has been reported, it is reasonable to expect that the same principles can be applied to explain gastrointestinal flow in horses. In this case the etiopathogenesis of gastrointestinal impaction could be explained as the result of one the following mechanisms or their combination: 1- Decrease in gastrointestinal motility; 2- Decrease in gastrointestinal diameter; 3- Increase in ingesta viscosity.

1- Decrease in gastrointestinal motility and impaction - Since gastrointestinal contractions are responsible for producing the pressures required to maintain flow (Morel et al. 1990, Gregersen and Kassab 1996), reduced gastrointestinal motility can compromise flow. In the horse, perhaps the best explored factor related to the etiopathogenesis of gastrointestinal impaction is reduced

gastrointestinal motility for which amitraz toxicosis has been the prototype. Cases of large colon impaction in horses bathed with amitraz have been reported (Auer et al. 1984, Lopes et al. 1999).

Table 2.1 – Incidence and case fatality of gastrointestinal impactions in horses.

Reference (case series)	Impaction location	Cases (Incidence)	Case Fatality*
(Tennant et al. 1972)	cecum	5 (1.1%)	NI
(453 colic cases)	large colon	65 (14.3%)	NI
	small colon	42 (9.3%)	NI
(Hekmati and Shahrasbi 1974)	cecum	9	33.3%
(9 cases of surgical cecal impaction)			
(Sembrat 1975)	cecum	7 (1.7%)	NI
(423 cases of gastrointestinal disorders)	large colon	56 (13.2%)	NI
(Boles and Kohn 1977)	large and small colon	10	50%
(10 cases of foreign body impaction)			
(Gay et al. 1979)	small colon	6	16.7%
(6 cases of foreign body impaction)			
(Edwards 1981)	ileum	2 (2.0%)	0.0%
(98 horses undergoing colic surgery)			
(Barclay et al. 1982)	stomach	4	0.0%
(4 cases of gastric impaction)			
(Campbell et al. 1984)	cecum	21	66.7%
(21 cases of cecal impaction)			
(Auer et al. 1984)	large colon	3	0.0%
(3 colic cases after amitraz bath)			
(Embertson et al. 1985)	ileum	12 (6.9%)	33.3%
(174 colic cases)			
(Wolfers and Bohm 1985)	esophagus	79	2.5%
(79 cases of esophageal obstruction)			

Fatality was based on the number of cases discharged from the hospital.

NI – not informed.

Table 2.1 – Incidence and fatality of gastrointestinal impactions in horses (continued).

Reference (case series)	Impaction location	Cases (Incidence)	Case Fatality*
(Foreman and White II 1986) (118 colic cases in the field)	cecum	2 (1.7%)	NI
	large colon	16 (13.6%)	NI
	small colon	5 (4.2%)	NI
	large intestine (total)	27 (22.9%)	NI
(Owen et al. 1987) (216 horses treated surgically for colic)	stomach	2 (0.9%)	0.0%
(Adams et al. 1988) (20 foals treated surgically for colic)	large intestine	1 (5.0%)	0.0%
(Specht and Colahan 1988) (48 cases of surgical sand colic)	large intestine	46 (95.8%)	NI
(Craig et al. 1989) (61 cases of esophageal disorders)	esophagus	27 (44.3%)	20.0%
(Parks et al. 1989) (75 cases of ileal impaction)	ileum	75	46.7%
(Morris et al. 1989) (229 colic cases at a hospital)	ileum	17 (7.4%)	NI
	large colon	34 (14.8%)	NI
(Ragle et al. 1989) (40 cases of surgical sand colic)	large intestine	40	22.5%
(Reeves et al. 1989) (158 colic cases treated surgically)	small intestine	5 (3.2%)	80.0%
	cecum	3 (1.9%)	0.0%
	large colon	16 (10.1%)	50.0%
	small colon	14 (8.9%)	22.0%
(Milne et al. 1990) (3 cases of gastric impaction)	stomach	3	100.0%

* Fatality was based on the number of cases discharged from the hospital.

NI - not informed.

Table 2.1 – Incidence and fatality of gastrointestinal impactions in horses (continued).

Reference (case series)	Impaction location	Cases (Incidence)	Case Fatality*
(White 1990) (2385 colic cases at hospitals)	stomach	(0.3%)	12.5%
	ileum	(1.7%)	50.0%
	cecum	(1.7%)	42.9%
	large colon	(7.4%)	16.9%
(Proudman 1991) (200 colic cases in the field)	pelvic flexure	10 (5%)	NI
	other	19 (9.5%)	NI
(Rook et al. 1991) (8 cases of impaction)	large colon	8	12.5%
(Ruggles and Ross 1991) (1488 colic cases at a hospital)	colon	273 (18.3%)	NI
	small colon	28 (1.9%)	14.3%
(Dart et al. 1992b) (102 cases of small colon disease)	small colon	42 (41.2%)	NI
(Edwards 1992) (900 horses undergoing colic surgery)	small colon	14 (1.6%)	21.4%
(McClure et al. 1992) (4 cases of fecalith impaction)	small colon	4	25%
(Pugh and Thompson 1992) (7 cases of impaction)	ileum	2 (28.6%)	0.0%
	large colon	5 (71.4%)	0.0%
(Ragle et al. 1992) (15 horses undergoing colic surgery)	large colon	5 (33.3%)	0.0%
	small colon	8 (53.3%)	0.0%
(Boening and Leendertse 1993) (115 cases of colic in pregnant mares)	cecum	1 (0.9%)	0.0%
	large colon	3 (2.6%)	0.0%
(Collatos and Romano 1993) (48 cases of cecal impaction)	cecum	48	41.7%

* Fatality was based on the number of cases discharged from the hospital.

NI - not informed.

Table 2.1 – Incidence and fatality of gastrointestinal impactions in horses (continued).

Reference (case series)	Impaction location	Cases (Incidence)	Case Fatality*
(Phillips and Walmsley 1993)	cecum	2 (1.3%)	0.0%
(151 colic surgeries)	large colon	6 (4.0%)	17.7%
	small colon	3 (2.0%)	33.3%
(Cohen et al. 1995)	ileum	14 (1.7%)	NI
(821 colic cases in the field)	cecum	13 (1.6%)	NI
	colon	196 (23.9%)	NI
(Dabareiner and White 1995)	large colon	147	5.4%
(147 cases of large colon impaction)			
(Hanson et al. 1996)	ileum	10	0.0%
(10 cases of ileal impaction)			
(Reeves 1996)	NI	96 (24.4%)	NI
(406 colic cases at hospitals)			
(Vatistas et al. 1996)	ileum	1 (1.5%)	0.0%
(67 foals undergoing colic surgery)	large colon	10 (14.9%)	0.0%
	small colon	25 (37.3%)	9.0%
(Dart et al. 1997)	cecum	38 (39.6%)	34.2%
(96 cases of cecal disease)			
(Hillyer and Mair 1997)	colon	4 (6.9%)	NI
(58 cases of recurrent colic)			
(Mair and Hillyer 1997)	colon	32 (30.2%)	0.0%
(106 cases of chronic colic)	cecum	3 (2.8%)	66.7%
(Tinker et al. 1997a)	NI	9 (8.7%)	11.1%
(104 colic cases in the field)			
(Hanson et al. 1998)	ileum	28	14.3%
(28 cases of ileal impaction)			

* Fatality was based on the number of cases discharged from the hospital.

NI - not informed.

Table 2.1 – Incidence and fatality of gastrointestinal impactions in horses (continued).

Reference (case series)	Impaction location	Cases (Incidence)	Case Fatality*
(Cohen and Woods 1999) (1030 colic cases in the field)	large colon	202 (19.6%)	NI
(Lopes et al. 1999) (14 cases of large colon impaction)	large colon	14	0.0%
(Rhoads et al. 1999) (3349 colic cases at a hospital)	small colon	84 (2.5%)	13.1%
(Feige et al. 2000) (34 cases of esophageal impaction)	esophagus	34	11.8%
(Southwood et al. 2000) 6 cases of focal eosinophilic enteritis	jejunum	6	16.7%
(Hudson et al. 2001) (182 colic cases in the field)	large colon	30 (16.5%)	NI
(Little and Blikslager 2002) 78 cases of ileal impaction	ileum	78	3.8%

* Fatality was based on the number of cases discharged from the hospital.

NI - not informed.

Experimentally, it has been shown that this acaricide can depress gastrointestinal motility in horses and cause large colon impaction after topical and intravenous administration (Roberts and Seawright 1983). Experimental studies also demonstrated that the effects produced by amitraz on large colon motility can be prevented by the α 2-adrenergic antagonist yohimbine, which suggests that amitraz acts as an α 2-adrenergic agonist (Sellers et al. 1985).

The association between abnormal motility and gastrointestinal impaction was also evident in a mare with inflammation and fibrosis of the myenteric ganglia that had recurrent impaction of the small colon (Burns et al. 1990). Further evidence was found in a retrospective study of 101 horses with large colon impaction where follow up information revealed that 33 (32.7%) had at least one episode of colic after discharge from the hospital. This observation suggested that the initial colic episode may have caused permanent damage to the colon (Dabareiner and White 1995). This hypothesis was supported by the observation that the number of neurons in the myenteric plexus of horses with chronic large colon disease was decreased (Schusser and White 1997).

Cecal and large colon impactions have been frequently reported in horses submitted to general anesthesia and surgery and/or confined in a stall (Haynes et al. 1980, Campbell et al. 1984, Ross et al. 1985, Edwards and Ruoff 1991, Collatos and Romano 1993, Dabareiner and White 1995, Mair and Hillyer 1997). In those cases, it is likely that impaction can be explained

by reduced gastrointestinal motility because it has been demonstrated that anesthetics (Lester et al. 1992, Freeman and England 2001) and reduced physical activity (Orton et al. 1985) may produce gastrointestinal hypomotility and slower gastrointestinal transit. However other factors such as dietary changes may also play a role in the etiology of impaction in horses submitted to anesthesia, surgery and reduced physical activity. Another factor that can be related to stall rest is the administration of non-steroidal anti-inflammatory drugs (Haynes et al. 1980, Campbell et al. 1984, Ross et al. 1985, Edwards and Ruoff 1991, Collatos and Romano 1993, Dabareiner and White 1995), which led to the speculation that these drugs could affect gastrointestinal motility and contribute to impaction formation. However this hypothesis is not supported by a few experiments analyzing gastrointestinal myoelectric activity, contractility and passage of radiolabeled markers in horses treated with flunixin meglumine, indomethacin and dipyron, since only minimal effects on gastrointestinal motility could be seen (Lowe et al. 1980, Adams et al. 1984, Roger and Ruckebusch 1987, Lester et al. 1998). In contrast with these observations, recent in vitro studies have demonstrated that phenylbutazone, flunixin meglumine, ketoprofen, carprofen and indomethacin can inhibit contractility of strips of taenia, circular muscle and longitudinal muscle of the large colon (Van Hoogmoed et al. 1999, Van Hoogmoed et al. 2000).

Esophageal impaction in horses with megaesophagus (Whitehair et al. 1989, Feige et al. 2000) can also be explained by abnormal motility patterns. Furthermore recurrence of esophageal impaction was more likely in horses with megaesophagus than in horses with apparently normal esophagus (Feige et al. 2000). Three cases of gastric impaction diagnosed at necropsy in horses with history, clinical signs, and pathologic changes suggestive of liver disease due to ragwort (*Senecio jacobaea*) toxicosis have been reported. These findings led to the speculation that abnormal neural function by mechanisms similar to what has been proposed for hepatic encephalopathy (e.g., disturbance of the plasma amino acid pattern, generation of false neurotransmitters) could have affected gastric motility and contributed to impaction formation (Milne et al. 1990). The association between mesenteric ischemia and impaction has also been suggested. The presence of lesions in the mesenteric vessels and intestinal ischemia during surgery or necropsy in horses with impaction in the small intestine and large intestine was the base for this suggestion (White 1981, Embertson et al. 1985). Since mesenteric ischemia can alter intestinal motility (Davies and Gerring 1985), it is possible that ischemia could cause impaction by compromising intestinal motility. However, an unsuccessful attempt to produce large intestine impaction by experimentally producing mesenteric arteritis with *Strongylus vulgaris* larvae has been reported (Sellers et al. 1982).

2- Decrease in gastrointestinal lumen and impaction - Resistance to ingesta flow is inversely correlated to the diameter of the gastrointestinal lumen (Morel et al. 1990). This fact explains why, in horses with no congenital or acquired distortion of the gastrointestinal lumen, impactions commonly form at sites of transition from a larger lumen to a smaller lumen, such as the cecum, pelvic flexure and transverse colon (Allen and Tyler 1990, Dart et al. 1992b). Furthermore there is evidence that congenital or acquired strictures of the gastrointestinal lumen are preferred sites for impaction and may lead to acute or chronic recurrent obstruction. Large colon impaction has been experimentally produced in horses with partial obstruction of the pelvic flexure by passing a filled rubber bag through a colostomy (Lowe et al. 1980). In experimental studies evaluating different surgical techniques for intestinal anastomosis in horses, impaction at the site of the anastomosis has been a common complication attributed to reduced intestinal lumen diameter (Reinertson 1976, Hanson et al. 1988). In one study, luminal diameter at the site of the

anastomosis was significantly smaller in horses that did not respond to medical treatment for impaction than in horses that did not have impaction or that responded to medical treatment (Hanson et al. 1988).

Further indications that luminal narrowing may predispose to impaction have been found in clinical reports. Several cases of impaction secondary to esophageal strictures have been reported (Hoffer et al. 1977, Suann 1982, Nixon et al. 1983, Gideon 1984, Craig and Todhunter 1987, Craig et al. 1989, Feige et al. 2000). It has also been observed that in cases of esophageal stenosis, recurrence of impaction was more likely than in cases where no morphological changes in the esophagus were identified (Craig et al. 1989, Feige et al. 2000). A case of stomach impaction in a horse with pyloric stenosis has also been reported (Munroe 1984).

Several cases of impaction at the site with a narrower lumen (usually ileum) in horses with muscular hypertrophy of the small intestine have been reported (Parks et al. 1989, Chaffin et al. 1992, Little and Blikslager 2002). An uncommon form of small intestinal impaction secondary to luminal stenosis was also found in a horse with enteric pythiosis (Allison and Gillis 1990). Impaction at the site of anastomosis in the small intestine or small colon has been reported as a common complication in horses submitted to surgery (Parks et al. 1989, Haven et al. 1991, Gerard et al. 1996, Fugaro and Cote 2001). Impactions at sites of stenosis of the small intestine in horses with inflammatory bowel disease (Scott et al. 1999) and eosinophilic enteritis (Southwood et al. 2000) have also been described. Impaction secondary to fibrous strictures of the large colon has been reported in three horses, but in no case was the cause of the stricture determined (Rose et al. 1991). Similarly a circumferential fibrous stricture in the small colon was found to cause impaction in a mare (Dart et al. 1992a). Large colon impaction secondary to partial luminal obstruction due to mural lymphoma has also been observed in two horses (Dabareiner et al. 1996). A series of 22 cases of secondary large colon impaction in horses with partial luminal obstruction due to segmental eosinophilic colitis has been published (Edwards et al. 2000). An impaction at a site where the small colon had become entwined around the left ovary producing a kink and reduction of luminal size has been reported in four mares (Edwards 1992, Mair 2002).

3- Increase in ingesta viscosity and impaction - Resistance to ingesta flow is directly correlated to ingesta viscosity (Morel et al. 1990). Although no study assessing the viscoelastic properties of ingesta in horses with impaction has been published, an increase in viscosity of gastrointestinal content has been demonstrated to be a major feature in diseases such as meconium impaction in human infants (Griffiths and Watkeys 1976). Ingesta viscosity is inversely correlated to water content and laxatives can decrease ingesta viscosity by promoting ingesta hydration (Griffiths and Watkeys 1976, McRorie et al. 1998). The viscoelastic properties of ingesta also depend on the nature of its solid phase (Srivastava and Srivastava 1989). Thus any condition leading to ingesta dehydration (e.g., systemic dehydration) (Clarke et al. 1992) or to larger and/or harder particles within the gastrointestinal tract (e.g., poor quality roughage, poor dentition) (Ralston et al. 2001) can increase ingesta viscosity.

Although the rheological properties of ingesta have not been studied in horses, experimental data indicate that similar assumptions about the effects of ingesta viscosity on ingesta flow are valid for this species. Large colon impaction was experimentally produced in horses by offering alfalfa pellets (a diet with laxative effects) for a few weeks followed by an abrupt change to coarse hay (Lowe et al. 1980). In another study, water content of gastric ingesta

was higher and the rate of passage through the stomach was faster in horses fed ground roughage than in horses fed long hay (Meyer et al. 1980).

Several clinical reports provide further evidence that an increase in ingesta viscosity may be associated with gastrointestinal impaction in horses. Several cases of esophageal obstruction due to coarse ingested material (e.g., coarse hay, pieces of carrots, apples or sugar beet, bedding, hair) or dry pellets or cubed feed have been reported (Harris 1981, Wolfers and Bohm 1985, MacDonald et al. 1987, Craig et al. 1989, Orsini et al. 1991, Feige et al. 2000). Reports of a case of gastric impaction caused by sugar beet pulp (Jones et al. 1972) and two cases of gastric impaction after ingestion of straw and coarse grass respectively have been published (Owen et al. 1987). A case of gastric impaction caused by ingestion of persimmon fruit (*Diospyros kaki*) and mesquite beans (*Prosopis juliflora*) has been reported (Honnas and Schumacher 1985). A case of gastric impaction (Cummings et al. 1997) and a case of gastric and small intestine impaction (Kellam et al. 2000) caused by ingestion of persimmon fruit (*Diospyros virginiana*) have also been reported. Persimmon fruit (especially when not ripe) is rich in tannins which form an adhesive coagulum when exposed to gastric acid leading to formation of a hard mass containing the large and hard persimmon seeds and other feed material (Cummings et al. 1997), (Kellam et al. 2000). Ileal impaction has been repeatedly associated with the ingestion of Bermuda grass hay (Emberson et al. 1985, Parks et al. 1989, Pugh and Thompson 1992, Hanson et al. 1998, Little and Blikslager 2002), which is known to have a relatively low digestibility (Lieb et al. 1993). Ingestion of other coarse material such as hair (Turner 1986), cracked corn (Bohanon 1988) and hemp bedding (Green 1996) has also been reported to cause small intestine impaction in horses. In a retrospective study of 229 cases of colic, it was found that feeding pelleted hay was more common in the group of horses with large colon impaction than in the other horses with colic. It was suggested that the reduced digestibility of pelleted hay could explain this finding. Furthermore, the fact that more horses with large colon impaction had raced or trained the day prior to onset of colic, led to the speculation that reduced digestibility of feed and dehydration due to exercise may have contributed to impaction formation (Morris et al. 1989). A case of several impactions by fibrous foreign bodies in the small intestine and large colon has also been reported (Getty et al. 1976). In a series of eight cases of large colon impaction caused by solid masses of hard seeds after ingestion of cockspur hawthorn fruit (*Crataegus crusgalli*) the authors suggested that reduced water consumption due to cold weather was likely a contributing factor (Rook et al. 1991). Feeding coarse Bermuda grass hay combined with intense physical activity and possibly reduced water consumption due to cold weather were suggested as the cause of large colon impaction in a series of 5 cases (Pugh and Thompson 1992). The report of a case of large colon impaction in an aged horse with poor dentition, chronic weight loss and difficult mastication (Steinebach and Cole 1995) is likely another example of impaction secondary to an increase in ingesta viscosity, because poor mastication results in swallowing of larger pieces of grass and/or hay. An association between large colon impaction and coarse roughage was also suggested in a series of 12 horses and 2 mules with impaction (Lopes et al. 1999). Recently it was reported that the dry matter content of ingesta from the pelvic flexure of 7 horses with feed impaction was more than double the dry matter at the same site in normal horses (Freeman 2002). Obstructions of the transverse or small colon by ingestion of other coarse materials such as fibrous synthetic material used for fencing (Boles and Kohn 1977), rope, twine, rag (Gay et al. 1979) and hair have also been reported (Yvorchuk-St Jean et al. 1993). Reports of obstruction of the large intestine by sand and gravel can also be found. The physical properties of sand and

gravel lead to sedimentation with formation of impactions at different sites of the large intestine (Specht and Colahan 1988, Ragle et al. 1989, Gilroy and Bellamy 1998).

Other factors that may be associated with the etiopathogenesis of impaction

There is evidence that factors such as season, weather condition, age, breed, gender and intestinal parasitism may be associated with an increased risk for gastrointestinal impaction. Some of the associations between these factors and gastrointestinal impaction are not clear, although the mechanisms previously discussed seem valid in most cases.

Season / weather conditions – In a series of 42 cases of small colon impaction, most horses were admitted during winter and early spring (Tennant et al. 1972). In a series of 12 cases of ileal impaction, 10 cases occurred between June and November (Embertson et al. 1985). Large colon impaction in 8 horses caused by ingestion of cockspur hawthorn fruit (*Crataegus crusgalli*) was likely associated with unseasonably cold weather which might have contributed to an unusual drop of large fruits and reduced water consumption (Rook et al. 1991). In a series of 28 cases of small colon impaction, 20 (71.4%) were admitted between October and April (Ruggles and Ross 1991). In a series of 84 cases of small colon impaction it was found that these horses were more likely to be admitted in fall and winter, when compared with horses with other forms of colic (Rhoads et al. 1999). The association between season / weather and impaction may be explained by the mechanisms described by Morel et al. (1990): It is possible that cold and dry weather conditions contribute to gastrointestinal impaction by reducing ingestion of water, causing systemic dehydration and leading to ingestion of coarse roughage or some unusual feed.

Age – In a series of 7 cases of cecal impaction, the proportion of horses 8 years or older (5/7) was significantly higher than in the hospital population (23%) (Sembrat 1975). In a series of 17 cases of ileal impaction the proportion of horses between 1 and 4 years was significantly higher than in the rest of the 212 colic cases (Morris et al. 1989). In a series of 42 cases of small colon impaction, it was found that horses older than 15 years were overrepresented when compared with the hospital population. It was suggested that aged horses are prone to impaction due to deterioration in teeth wear, mastication, and gastrointestinal function (Dart et al. 1992b). In a series of 57 cases of cecal impaction, horses were significantly older (mean, 11.4 years) than the hospital population (Collatos and Romano 1993). In another series of 38 cases of cecal impaction, it was found that horses older than 15 years were over represented relative to the hospital population (Dart et al. 1997). Thus, most reports suggest that impactions are more common in older horses. Although the mechanisms are not known, it may be possible to explain the relation between old age and impaction using the mechanisms demonstrated by Morel et al. (1990). Aging may lead to poor dentition and changes in the gastrointestinal tract (e.g., damage to the myenteric plexus) that would affect gastrointestinal flow by increasing the viscosity of ingesta and compromising intestinal motility respectively.

Breed – In several series of cases of small colon impaction, it has been found that miniature horses and ponies were overrepresented when compared with the hospital population (Tennant et al. 1972, Gay et al. 1979, Dart et al. 1992b, McClure et al. 1992, Ragle et al. 1992). It has been speculated that hay may be relatively too coarse for small sized horses (McClure et al. 1992,

Ragle et al. 1992). Other breeds have also been suggested to be at an increased risk of gastrointestinal impactions. In a series of 17 cases of ileal impaction, Arabians were overrepresented relative to the other 212 colic cases (Morris et al. 1989). In a series of 38 cases of cecal impaction, it was found that Arabian, Morgan and Appaloosa horses were overrepresented relative to the hospital population (Dart et al. 1997). In a series of 30 cases of chronic colic in mature horses (age 1 year or older) due to large colon impaction, the proportion of Thoroughbreds (50%) was higher than in the hospital population (Mair and Hillyer 1997). Thus breed predisposition other than the increased incidence in small breeds has not been consistently reported. It is possible that in certain breeds morphologic and physiologic features of the gastrointestinal tract could affect ingesta flow and predispose to impaction. In the case of small breeds, the smaller diameter of the intestine would lead to an increase in resistance to flow (Morel et al. 1990).

Gender – Gender predisposition for large colon impaction was suggested in a series of 147 cases where 92 horses (62.6%) were females (Dabareiner and White 1995). However these findings contradict other studies. In a series of 34 cases of large colon impaction out of 229 colic cases, geldings were overrepresented (55.9%) when compared to the other colic cases (Morris et al. 1989). In a series of 30 cases of chronic colic in horses older than 1 year due to large colon impaction, the proportion of males was significantly higher (72%) (Mair and Hillyer 1997). Thus there is no definitive evidence of gender predisposition to gastrointestinal impaction in horses. However at least theoretically, differential expression of morphologic and physiologic features affecting ingesta flow could cause gender predisposition to gastrointestinal impaction.

Tapeworm infection – A strong association between ileal impaction and infection by *Anoplocephala perfoliata* diagnosed by both a coprological and an antibody assay has been reported (Proudman et al. 1998). This association was further suggested by the finding of a high prevalence of infection by *Anoplocephala perfoliata* in a group of horses having a high incidence of colic where 4 cases of ileal impaction were diagnosed (Proudman and Holdstock 2000). It has also been reported that in horses not treated with pyrantel salt (an effective anthelmintic against *Anoplocephala perfoliata*) for 3 months the risk for ileal impaction is increased (Little and Blikslager 2002). This parasite is frequently found attached to the mucosa of the terminal ileum and base of the cecum (Pearson et al. 1993, Fogarty et al. 1994, Nilsson et al. 1995, Williamson et al. 1997). Pathological changes in the ileocecal valve attributed to parasitism by *Anoplocephala perfoliata* have been reported and the intensity of the lesions (from mild inflammation to severe inflammation and necrosis) is proportional to the intensity of the infection (Nilsson et al. 1995, Williamson et al. 1997, Rodriguez-Bertos et al. 1999). It is likely that these worms increase the resistance to ingesta flow through the ileocecal valve because clusters of worms within the terminal ileum (Pearson et al. 1993, Fogarty et al. 1994, Nilsson et al. 1995, Williamson et al. 1997), inflammation and thickening of the valve (Lyons et al. 1983, Nilsson et al. 1995, Williamson et al. 1997, Rodriguez-Bertos et al. 1999), fibrosis and reduced distensibility of the valve (Beroza et al. 1986), and prolapse of the terminal ileum (Williamson et al. 1997, Rodriguez-Bertos et al. 1999) have been observed in parasitized horses. However the possibility that *Anoplocephala perfoliata* can cause impaction by other mechanisms such as interfering with gastrointestinal motility cannot be ruled out (Proudman et al. 1998).

Conclusions

Gastrointestinal impactions are common diseases in horses. Most forms of gastrointestinal impaction have a relatively low fatality rate. However, for some forms of impaction (e.g., cecal impaction), fatality can be quite high even when appropriate treatment is provided. Although important information is still lacking, the data compiled in this text indicate that one or more of the following mechanisms may lead to the occurrence of gastrointestinal impaction in horses:

- 1- Decreased force (generated by the gastrointestinal muscles) to promote ingesta flow
- 2- Increased resistance to ingesta flow
 - 2.1- Decreased gastrointestinal lumen diameter
 - 2.2- Increased ingesta viscosity

In this extensive search through scientific journals (using Medline) no clinical report or experimental study could be found that would contradict the validity of these mechanisms in explaining gastrointestinal impaction. Thus, the mechanisms presented above appear to be valid for the vast majority if not all cases of equine gastrointestinal impaction ever reported.

6- Current treatments for large colon impaction not caused by sand, gravel or other foreign materials

Medical treatment is the first choice for large colon impaction, at least in the majority of cases. The aims of medical treatment are to control pain and hydrate colonic ingesta (White and Dabareiner 1997). According to current textbooks, medical treatment for large colon impaction should include intravenous fluid therapy, laxatives and analgesics (White and Dabareiner 1997, Dabareiner 1998, Jones et al. 1998, Sullins 1999, Blikslager 2002). It is thought that the state of overhydration produced by IV fluid therapy plus the increase in ingesta osmolality produced by saline cathartics promote ingesta hydration (White and Dabareiner 1997). However no controlled study documenting these effects has been published. Most authors also emphasize that horses with impaction should be fasted until no impaction could be detected by rectal palpation to avoid further enlargement of the impaction (White and Dabareiner 1997, Dabareiner 1998, Blikslager 2002). Some authors acknowledge that enteral fluid therapy may be beneficial, but clearly emphasize that IV fluid therapy is the best option (White and Dabareiner 1997, Dabareiner 1998).

When medical treatment fails (pain cannot be controlled, peritoneal fluid suggests bowel deterioration, or signs of endotoxemia develop) surgical treatment is indicated (White and Dabareiner 1997, Dabareiner 1998, Jones et al. 1998, Sullins 1999, Blikslager 2002). The large colon is exteriorized through a median laparotomy and a pelvic flexure enterotomy is performed to allow emptying of colonic content (Dabareiner 1998, Sullins 1999). Manipulation of a heavily impacted colon may result in colon rupture, which would lead to severe contamination of the peritoneal cavity. Thus, a large abdominal incision is recommended to minimize stretching the colon and to avoid rupture (Dabareiner 1998, Sullins 1999).

In the only published report of a large number of cases of horses with large colon impaction, all 147 horses were treated with analgesics and intravenous fluid therapy, while 49 horses also received laxatives (magnesium sulfate or mineral oil). The volume of intravenous fluids administered ranged from 54 to 350 liters/day (mean, 118.9 liters/day). Feed was withheld until the impaction was no longer palpable on rectal examination and fecal transit was re-established. Medical treatment was effective in 120 of the 147 cases (81.6%) and duration of treatment ranged from 1 to 6 days (mean, 2 days). Surgery was performed in 24 horses (16.3%), while 3 horses (2.0%) were euthanized due to financial constraints after unsuccessful medical treatment for 48 h. Five horses (3.4%) were euthanized because colon rupture occurred during surgery (Dabareiner and White 1995).

An alternative approach has been reported in a series of 12 horses and 2 mules with large colon impaction: Fluids were administered exclusively via nasogastric tube and laxatives were not administered. Contrary to current recommendations, the horses were fed low fiber meals (1 kg of grain every 12 h) as soon as analgesics were not required to control pain even when an impaction could still be identified by rectal palpation. The volume of fluids administered ranged from 20 to 72 liters/day (mean 47.8 liters/day). All horses responded to medical treatment within 1 to 5 days (mean, 2.6 days). Treatment cost was low since IV fluids were not administered and surgery was not necessary in any case (Lopes et al. 1999).

It is not a surprise that enteral fluid therapy can be effective in treating large colon impaction. In horses, fluids are emptied from the stomach and moved through the small intestine very rapidly (Alexander and Benzie 1951, Argenzio et al. 1974a, Sosa Leon et al. 1997). Thus fluids given by nasogastric tube will rapidly reach the large intestine. Furthermore enteral fluid

therapy may stimulate gastrointestinal motility by mechanisms such as the gastrocolic response (Freeman et al. 1992). Although the recommendation in the textbooks is to fast horses with impaction, no controlled study has been published to support this recommendation. Indeed several studies have demonstrated that fasting reduces, while feeding increases gastrointestinal motility (Alexander 1952, Sellers et al. 1979, Roger and Ruckebusch 1987, Ross et al. 1990, Merritt et al. 1995). These findings suggest that feeding a low bulk diet may be beneficial for horses with large colon impaction. In order to determine the best treatment for large colon impaction controlled studies are necessary to compare the traditional IV fluid therapy + fasting approach with what appears to be more physiologic: enteral fluid therapy + low bulk meals.

7- Enteral fluid therapy for horses

Fluid therapy is commonly used in equine medicine. Indications for fluid therapy range from maintenance of hydration in horses with adipsia to correction of severe dehydration seen in conditions such as intestinal obstruction or colitis (Holbrook and Eades 1995). The availability of commercial administration sets designed for the administration of large volumes of fluids to horses unrestrained in the stall, has made intravenous fluid therapy easy, and the almost exclusive method of fluid therapy for horses (Schott 1998). Although it seems practical to bypass the gastrointestinal tract, this is not the most physiologic or economic way to administer fluids in many clinical situations. If fluids are administered by the enteral route, the GI mucosa acts as a natural selective barrier for absorption, and iatrogenic imbalances are less likely. The other great advantage of enteral fluid therapy is its low cost, since sterile fluids with finely adjusted compositions are not required.

Transit of fluids through the gastrointestinal tract

In horses, movement of fluids through the proximal gastrointestinal tract is rapid, reaching the large intestine in less than 1 h. In weaned foals, a considerable amount of barium was shown to move into the cecum 30 minutes after oral administration (Alexander and Benzie 1951). In adult ponies, the majority of chromium given by nasogastric tube as a marker of the liquid phase of ingesta could be found in the cecum 2 h after administration (Argenzio et al. 1974a). In a study using phenol red concentration as an indicator of gastric volume in 1 to 8 week old pony foals, about 85% of fluids (water or 0.9% saline) given by nasogastric tube at a dose of 15 ml/kg were emptied in 10 minutes (Baker and Gerring 1994). In adult horses, the same technique demonstrated that 90% of 8 liters of a rehydration solution administered by nasogastric tube was emptied from the stomach in 15 minutes. No difference was observed when isotonic fluid at 20°C or 5°C, isotonic fluid with glucose at 20°C, and a hypertonic fluid at 20°C were compared. Exercise at 70% VO₂ max until exhaustion did not affect gastric emptying of isotonic fluids (Sosa Leon et al. 1997).

The gastrocolic response

In horses and in other species, gastric distension produces an immediate increase in colonic motility, which is called the gastrocolic response. This mechanism has been demonstrated by electromyography and changes in intraluminal pressure in the equine colon after feeding (Sellers et al. 1979, Roger and Ruckebusch 1987, Merritt et al. 1995). Increased colonic motility due to the gastrocolic response may be beneficial in some clinical conditions such as large intestine impactions. However the gastrocolic response may produce pain or even contribute to rupture of an extremely distended bowel (Lopes 1999, Lopes et al. 1999).

Absorption of fluids and electrolytes through the gastrointestinal tract

Water lost through sweat, urine, expired air, and feces needs to be replaced by water ingestion. The volume of water ingested daily varies widely depending on many factors such as physical activity, environmental temperature and diet. Water intake (voluntary drinking plus water content of feed) of horses kept in stalls at temperatures ranging from 3 to 25° C and fed free choice hay ranges from 55.1 to 73.2 ml/kg/day. Horses eating grain drink less than horses eating just hay (Tasker 1967, Fonnesbeck 1968, Groenendyk et al. 1988). Water intake in horses submitted to exercise or horses that are dehydrated may be dramatically increased. Ponies submitted to 19 h of water deprivation can drink more than 50 ml/kg in 30 minutes (Sufit et al. 1985).

Water in the GI tract does not come exclusively from the diet. Large volumes of watery products from the GI glands are secreted, and water passively moves into the gut due to the increased osmolality produced by digestion of feed. In a 100 kg pony, the daily volumes secreted by the parotid glands, pancreas, biliary system and large intestine have been estimated to be 10 to 12, 10 to 12, 4 to 5 and 7.4 liters respectively. Discounting the volume secreted by the stomach and small intestine, which have not been quantified yet, the volume of secretion would reach 31.4 to 36.4 liters per day. The fact that only about 0.9 liters of water are excreted in feces per day, illustrates the extraordinary ability of the gastrointestinal tract to absorb water (Argenzio et al. 1974a, Argenzio 1990).

Absorption of water is a passive process that occurs via both transcellular and paracellular pathways following the absorption of electrolytes, carbohydrates, amino acids, peptides, volatile fatty acids, water-soluble vitamins, and bile salts. Sodium, the main extracellular cation, is actively absorbed by the GI mucosa and is the main driver for water absorption. The main mechanisms of sodium absorption are the Na⁺-glucose and Na⁺-amino acid co-transporters, the electroneutral Na⁺ and Cl⁻ transport (through Na⁺/H⁺ and Cl⁻/HCO₃⁻ exchange), the electroneutral Na⁺ and volatile fatty acid transport (through the absorption of undissociated volatile fatty acids and Na⁺/H⁺ exchange), and the electrogenic Na⁺ absorption (through Na⁺ channels). For the first four mechanisms glucose, amino acids, chloride and volatile fatty acids respectively are needed. Thus the presence of these nutrients in the GI lumen enhances sodium and consequently water absorption (Argenzio 1990, Barrett and Dharmasathaphorn 1994, Schedl et al. 1994, Crowe and Powell 1995, Acra and Ghishan 1996, Sandle 1998). Conversely, the presence of a poorly absorbed solute (e.g., magnesium sulfate) impairs water absorption because water is absorbed passively in the presence of an osmotic gradient (Izzo et al. 1996, Heizer et al. 1997).

Besides the composition of ingesta, several physiologic mechanisms determine the extent by which electrolytes and indirectly water are absorbed and secreted through the gastrointestinal mucosa. According to Starling's laws, the hydrostatic pressure in intestinal vasculature is a major factor. Increased hydrostatic pressure occurring with hypervolemia favors water secretion whereas low hydrostatic pressure occurring with hypovolemia favors water absorption. Another major factor, which is also explained by Starling's laws, is plasma oncotic pressure. A decrease in water absorption is seen when plasma protein and oncotic pressure is reduced such as in hypervolemia (e.g., caused by fluid therapy) or hypoproteinemia. The opposite occurs when plasma protein and oncotic pressure is increased such as in hypovolemia (Murray 1988).

The renin-angiotensin-aldosterone system has a major role in the regulation of the extracellular fluid. This system is activated by hypovolemia, and the resultant increase in the

concentration of angiotensin II causes arteriolar vasoconstriction further reducing hydrostatic pressure in intestinal vasculature, contributing to increase water absorption. An increase in aldosterone levels soon follows, which directly increases sodium and water absorption through the GI mucosa. On the other hand, hypervolemia results in the rapid decline of renin-angiotensin-aldosterone activity and decreased absorption of sodium and water. The plasma concentration of ions and osmolality also influence aldosterone secretion. Hyponatremia, hyperkalemia and low osmolality result in an increase in aldosterone release (Clarke et al. 1988, Clarke et al. 1992, Kenefick et al. 2000).

Another consequence of hypervolemia is an increase in preload leading to atrial stretching, which results in the release of atrial natriuretic peptide (ANP). Although the effects of ANP on the GI tract of horses have not been investigated, in other mammals there is evidence that ANP acts directly on epithelial cells, decreasing sodium and water absorption (Pettersson and Jonsson 1989, Moriarty et al. 1990, Argenzio and Armstrong 1993). Additionally ANP may reduce sodium and water absorption by 2 mechanisms: ANP inhibits the release of renin, which limits the activity of the renin-angiotensin-aldosterone system; and ANP acts directly on the adrenal glands reducing aldosterone secretion (Levin et al. 1998).

Thus, the extraordinary ability of the GI mucosa to absorb electrolytes and water can be used for fluid therapy. Absorption and secretion through the GI tract is influenced by the composition of the intestinal content, which can be manipulated by changing the composition of the fluid administered. Furthermore, absorption through the GI mucosa is strictly regulated by several physiologic mechanisms: In a horse with hypovolemia needing rapid replacement of fluids, sodium and water absorption is enhanced; in a horse with hypervolemia, absorption is decreased reducing the risks of severe overhydration.

Hemodynamic effects of enteral fluid therapy

The magnitude of the hemodynamic changes produced by enteral fluid therapy, as well as the time required for these changes to occur, has not been fully evaluated. However there are indications that these changes occur quickly and that they can be pronounced. In dehydrated humans, a significant increase in plasma volume was observed within 30 minutes after ingestion of oral hydration solutions (Greenleaf et al. 1998). Another study using ultrasonography showed that marked dilation of renal calyces and hydronephrosis can be detected in humans 60 minutes after ingestion of large volumes of water (20 ml/kg) (Morse et al. 1999). In humans with severe diarrhea, enteral fluid therapy can markedly reduce the amount of IV fluids required (Endsley and Galbraith 1998). In horses previously dehydrated by furosemide and water deprivation, the administration of 10 liters of fluids by nasogastric tube was able to restore PCV in 30 minutes (Rose et al. 1986). In another study, enteral administration of 4 liters of water or an electrolyte solution with 50 g of deuterium to horses was followed by the appearance of deuterium in blood 10 minutes later, with the peak deuterium concentration occurring after around 40 minutes (Marlin et al. 1998b). In horses with large colon impaction, administration of water by nasogastric tube rapidly produced polyuria and urine dilution (Lopes et al. 1999). In normal horses treated with an electrolyte solution by nasogastric tube, lower PCV and plasma protein, as well as polyuria and hypostenuria were seen after 2 h, but plasma expansion was limited when compared to IV fluid therapy (Lopes et al. 2002b).

Pathologic conditions affecting enteral fluid therapy

Intestine obstruction and ileus - In horses with intestinal obstruction or ileus, the stomach and small intestine may become distended by ingesta and secretions produced by the digestive tract (White 1999). In these cases enteral fluid therapy is contraindicated.

Mucosal inflammation - Mucosal inflammation can compromise mucosal integrity and function, and absorption of electrolytes and water may be severely affected. However in humans and calves the GI mucosa retains significant ability to absorb fluids in most forms of enteritis and colitis (including diseases such as cholera, *E. coli* and rotavirus). In humans and other animals with diarrhea, enteral fluid therapy can improve clinical condition and prevent death, despite the increase in the volume of diarrhea produced by enteral fluid therapy. If enteral fluid therapy cannot completely restore and maintain hydration, it might at least reduce the requirement of fluids by the IV route (Brooks et al. 1996, Hogan 1996, Endsley and Galbraith 1998, Nappert et al. 2000). In horses there is practically no information about the effect of mucosal inflammation on the ability of the GI tract to absorb fluids. In the only published study, enteral fluid therapy failed to produce a significant increase in plasma volume in adult horses with diarrhea induced by castor oil. According to the authors these results can be explained by the extremely fast transit of fluids through the small intestine produced by rapid administration of fluids (3 boluses of 8 to 10 liters administered every 30 minutes) which may have compromised fluid absorption (Ecke et al. 1998).

Indications of enteral fluid therapy

To restore and maintain hydration, electrolyte balance, and acid-base balance - Electrolyte and acid-base imbalances, and dehydration are commonly seen in horses, and enteral fluid therapy can be used to treat these conditions. Enteral fluid therapy can also be helpful in preventing dehydration in horses with ongoing fluid losses or that are not drinking properly. Conditions such as post-exercise dehydration, diarrhea and some types of colic can be successfully managed with enteral fluid therapy (Ecke et al. 1998, Marlin et al. 1998a, Lopes et al. 1999). In human medicine, enteral fluid therapy is known to be effective in the vast majority of patients with diarrhea, and its use is known to reduce morbidity and mortality (Snyder 1995). In humans and horses, the administration of enteral fluids before and during exercise is known to prevent dehydration and electrolyte imbalances, contributing to better performance. The use of enteral fluid therapy after exercise helps to restore water and electrolyte deficits, contributing to a speedier recovery (Jansson et al. 1995, Convertino et al. 1996, Hyypa et al. 1996, Marlin et al. 1998b, Casa et al. 2000).

To increase hydration of gastrointestinal content – In horses enteral fluid therapy promotes hydration of gastrointestinal content (Lopes et al. 2002b). In other species an increase in water content of ingesta reduces ingesta viscosity (McRorie et al. 1998) which facilitates ingesta flow (Morel et al. 1990). These effects are not exclusive to the proximal segments of the gastrointestinal tract. Since fluids administered by nasogastric tube can rapidly reach the large intestine, significant changes in the distal segments of the gastrointestinal tract occur shortly after fluid administration. An increase in hydration of gastrointestinal content is thought to be

beneficial to horses with impaction (White and Dabareiner 1997) and horses submitted to intestinal or perineal surgical procedures (Freeman 1999). To hydrate the contents of the rectum and small colon, an enema can also be used (Hjortkjaer 1979). Enteral fluid therapy may result in an increase in fecal volume and a further decrease in fecal consistency in patients with diarrhea, which should not be seen as undesirable (Brooks et al. 1996, Ecke et al. 1997). In fact the increased water content of feces produced by enteral fluid therapy may be beneficial in patients with diarrhea, contributing to the removal of toxins and pathogenic microorganisms (Alverdy and Piano 1997).

To stimulate intestinal motility - Reduced intestinal motility can be an important feature in the pathogenesis of large colon impaction. Even if it is not an initiating factor, intestinal hypomotility may occur later (White and Dabareiner 1997). Enteral fluid therapy may initiate the gastrocolic response and increase intestinal motility, helping to resolve large intestine impaction (Freeman et al. 1992, Lopes et al. 1999).

To promote diuresis - Overhydration is indicated to increase glomerular filtration and to improve excretion of toxic pigments (hemoglobin, myoglobin) and drugs (e.g., aminoglycosides, non-steroidal anti-inflammatories), helping to prevent kidney damage (Divers 1999). This may be another use for enteral fluid therapy since this treatment can rapidly increase plasma volume and urine production (Morse et al. 1999, Lopes et al. 2002a, Lopes et al. 2002b).

To increase hydration of pulmonary secretions - Horses with respiratory disease can have a thick exudate occluding the airways, especially if they are dehydrated. Enteral fluid therapy may contribute to increase hydration of the exudate and improve expectoration (Rapp 1988).

Contraindications of enteral fluid therapy

Lack of access to the gastrointestinal tract - If neither a nasogastric nor an esophagostomy tube can be passed (e.g., an obstruction in the thoracic esophagus), enteral fluid therapy cannot be used. An enema could still be used, but the effects would be limited primarily to hydration of the contents of the rectum and small colon (Hjortkjaer 1979).

Ileus and gastric reflux - If gastrointestinal motility is severely compromised and the horse has small intestinal distention and/or net reflux through the nasogastric tube, nothing should be given orally or by nasogastric tube. It is obvious that in this case fluids would not move further aborally and would instead contribute to gastric and intestinal distention. Under these conditions IV fluid therapy is absolutely indicated.

Horses unable to stand - In horses that cannot stand, the administration of large volumes of fluids into the stomach may result in reflux and aspiration. To use the enteral route, it seems advisable to keep the horse in sternal recumbency. If this is not possible, enteral fluid therapy is contraindicated.

Severe hypovolemia and shock - When immediate plasma expansion is needed, enteral fluid therapy should not be used alone and intravenous fluid therapy is indicated to immediately

restore volemia. It should also be considered that hypovolemia may result in poor gastrointestinal perfusion (Edouard et al. 1994) which may affect gastrointestinal motility (Davies and Gerring 1985) and absorption and lead to intolerance to enteral fluid therapy. The critical degree of dehydration where intravenous fluid therapy becomes mandatory is not known. However, in human medicine, dehydration as severe as 9% can be treated solely with enteral fluids (Snyder 1995). In many situations, enteral fluid therapy can be used simultaneously with IV fluid therapy, and, as soon as signs of shock remit, IV fluid therapy can be interrupted.

Administration of enteral fluid therapy

Route - Oral fluid therapy is the most physiologic and the least invasive way to restore hydration, but most sick horses will not voluntarily drink sufficient fluid volumes. Forced oral administration of large volumes of fluids is not recommended due to the risk of aspiration. However small volumes of hypertonic electrolyte solutions or salt paste administered with a syringe have been successfully used in endurance horses to replace electrolytes and stimulate thirst (Nyman et al. 1996, Sosa Leon et al. 1998, Dusterdieck et al. 1999).

Fluids can also be given by a nasogastric or nasoesophageal tube. A tube long enough to reach at least the caudal cervical esophagus is indicated (e.g., a nasogastric tube for colicky horses), although placing the tube in the stomach is advantageous because the presence of net gastric reflux can be ruled out before enteral fluid therapy is started, and the stomach can be emptied in case discomfort occurs due to gastric distention produced by fluid therapy. Tube manipulation must be gentle to avoid damaging the nasal passage, pharynx, larynx and esophagus. Alternatively, a tube with a small caliber (external diameter, 6 mm) designed for enteral nutrition (Veterinary Enteral Feeding Tube, Mila International, Inc.) can be used. This type of tube has the advantage of producing less pressure on the mucosa and less discomfort for the horse (Lopes et al. 2002a), which is particularly important for prolonged treatments. However the small bore tube cannot be used to check for reflux or to drain fluids given in excess. Before administering fluids, the position of the nasogastric tube has to be checked to avoid inadvertent administration of fluids in the trachea. When a large bore tube is used, it is easy to feel the resistance produced by the collapsed esophagus while inserting the tube. It is also possible to palpate the tube in the neck dorsolaterally to the trachea. While passing the small bore tube, it is not so easy to feel the difference between the resistance of the esophagus and the lack of resistance when the tube is in the trachea. The horse usually coughs when the tube is positioned in the trachea. If the horse does not react, a small volume (100 ml) of water can be administered, which would likely produce coughing in case the tube had been passed into the trachea. Radiography (Figure 2.1) or ultrasonography (Figure 2.2) of the neck or endoscopy of the pharynx (Figure 2.3) can be used for definitive confirmation of appropriate tube position.

The best way to administer enteral fluid therapy is by gravity rather than with a pump (Ecke et al. 1997). A large funnel made with a plastic gallon jug (e.g., empty milk jug) can be used (Figure 2.4). Enteral fluid therapy can also be administered continuously to horses kept unrestrained in the stall using a coiled line connected to the nasogastric tube (Figure 2.5). When the small bore tube is used (Figure 2.6), it is safe to allow the horse to have free access to feed (Murray and Schusser 1993), while it is more likely that the large-bore nasogastric tube would interfere with deglutition.

When the nasoesophageal or nasogastric tube cannot be passed (e.g., due to nasopharyngeal or esophageal obstruction), it may be possible to give fluids through an esophagostomy. A stoma is surgically created in the cervical esophagus and a large-bore tube is passed into the caudal esophagus (Freeman and Naylor 1978, Lopes 2001). Besides fluid therapy, the esophagostomy permits the administration of liquid diets in the form of a slurry made of regular horse feed. As soon as the horse regains the ability to eat and drink, the tube can be removed. Complete healing of the fistula occurs in 2 or 3 weeks. Because severe complications such as laryngeal hemiplegia, periesophageal infection and mediastinitis can occur, (Stick et al. 1981, Lopes 2001) an esophagostomy should only be performed as the last resort.

Intracecal administration of fluids through a percutaneous catheter to maintain hydration has been reported in ponies, but 3 of the 6 ponies developed septic peritonitis (Mealey et al. 1995). The high risk of complications precludes using this route of administration.

Rectal administration of large volumes of fluids (44 liters) has been reported to increase the water content in the small colon and large dorsal colon of dehydrated horses, but did not change central venous pressure, PCV or total plasmatic protein (Hjortkjaer 1979). Current use of enemas is limited to cases of impactions of the small colon and rectum such as meconium impaction (Hanson 1999), with the objective of softening the impacted mass. Enemas can cause tenesmus and rupture of the rectum and should be administered cautiously.

Volume and rate of infusion - Since stomach capacity is about 18 liters in the average horse (Pfeiffer and MacPherson 1990), and fluids rapidly leave the stomach after nasogastric administration (Sosa Leon et al. 1997), relatively fast rates of infusion can be achieved with enteral fluid therapy. Normal horses and horses with large colon impaction may tolerate as much as 10 liters every 30 minutes (40 ml/kg/h for a 500 kg horse) (Rose et al. 1986, Sosa Leon et al. 1997, Lopes et al. 1999). Slower rates of infusion have been proposed by other authors, although no data has been presented to support these recommendations (Morris 1987, Schlipf and Baxter 1992, Holbrook and Eades 1995). Fluids can be administered either as continuous infusion or as large boluses. Continuous infusion may reduce the risk of excessive gastric distention but may minimize the gastrocolic response. However these hypotheses were not proven when 80 liters were administered to normal horses during 8 h as 10 liters boluses or continuous infusion (167 ml/min) (Lopes et al. 2002a). To prevent excessive gastric distention and abdominal discomfort it is advisable to start with smaller doses or slower rates of infusion and gradually increase the dose over a few hours.

Temperature - Because of concerns about adverse effects of fluid temperature, warm fluids have been used (Lopes et al. 1999). However in normal horses, fluids at 5° C did not affect gastric emptying or fluid absorption (Sosa Leon et al. 1995). Since cold fluids may increase energy consumption (Carlson 1971), it is preferable to use warm fluids (38° C) whenever large volumes are going to be administered, particularly when the environment is cold and in horses with malnutrition or hypothermia. On the other hand, the administration of cold fluids may help to combat hyperthermia in horses with post-exercise dehydration.

Composition of fluids for enteral use

Horses that are being fed and have normal kidney function may tolerate large volumes of fluids that do not have the ideal composition. In this instance, the diet will be a source of electrolytes and the kidneys will help to prevent severe electrolyte imbalances. For horses with anorexia or kidney disease and for prolonged treatments (more than 24 h), the composition of the fluid is more critical.

Water - Water is a hypotonic solution (10-20 mOsm/liter) with very low concentrations of electrolytes (Gisolfi and Duchman 1992). Since water is absorbed passively following the absorption of sodium, maximum absorption should not be expected with the administration of plain water, although a major part will be absorbed rapidly (Sosa Leon et al. 1995, Marlin et al. 1998b, Monreal et al. 1999). The administration of small volumes of water (e.g., 30 liters per day for a 500 kg horse) may be safe in horses that are eating and are going to be supplied with some electrolytes from their diet. However hyponatremia (Sosa Leon et al. 1995, Marlin et al. 1998b, Lopes 1999, Monreal et al. 1999), synchronous diaphragmatic flutter (Monreal et al. 1999), and seizures (Lopes 1999) have been reported after the administration of water by nasogastric tube. Therefore large volumes of water should never be administered without closely monitoring plasma electrolytes.

Electrolyte solutions - A 0.9% NaCl solution (9 g of table salt per liter of water) is isotonic and contains sodium, which is important for water absorption. However a 0.9% NaCl solution can produce hypernatremia, hyperchloremia and acidosis, because its sodium and chloride concentration is higher (154 mEq/l of each electrolyte) than in the plasma (Lopes et al. 2002a). Since it does not have potassium, 0.9% NaCl administration can contribute to hypokalemia, which may be already present in a horse that has not been eating. When large volumes are going to be administered other electrolyte solutions, containing different concentrations of sodium and chloride and including a source of potassium such as KCl, should be used. To adjust the Na:Cl ratio closer to what is normal in the plasma, a chloride free salt such as sodium bicarbonate (NaHCO₃) can be added. As an example, a solution made of 5.9 g of NaCl, 0.3 g of KCl and 3.4 g of NaHCO₃ per liter of water has sodium, potassium and chloride concentrations close to what is normally found in plasma (141, 4 and 105 mEq/l respectively). The administration of 60 liters of this solution at the rate of 10 liters/h to normal horses, did not produce any abnormality in plasma sodium, potassium or chloride (Lopes et al. 2002b).

Glucose is known to improve absorption of rehydration solutions in humans and other animals, because of the co-transport mechanism of sodium and glucose. Amino acids have the same effect, but due to problems of stability and toxicity they are not commonly included in rehydration solutions (Schedl et al. 1994). Despite the good results obtained with glucose solutions, it is thought that fluids containing carbohydrate polymers (e.g., maltodextrin) have advantages: 1- More solute (including electrolytes) can be added while keeping osmolality low, which may contribute to increase water absorption since the small intestine is highly permeable to water, and acts as an equilibration chamber; 2- Hydrolysis of polymers by brush border enzymes results in a higher concentration of glucose on the mucosal surface, increasing the efficiency of glucose absorption by membrane transporters (Thillainayagam et al. 1998).

Despite the known benefits for humans (Gisolfi and Duchman 1992, Convertino et al. 1996), glucose failed to improve absorption of oral rehydration solutions in horses

experimentally dehydrated by furosemide administration (Sosa Leon et al. 1995, Monreal et al. 1999). The extremely fast transit of fluids through the small intestine in the horse compared to humans may limit the efficiency of glucose-electrolyte solutions. Additionally horse sweat is hypertonic and horse athletes may need to replace more electrolytes than human athletes, but there is a limit imposed by the osmolality of the rehydration solution. Although it has not been tested in horses, replacing glucose by a carbohydrate polymer would make it possible to increase the electrolyte concentration in the rehydration solution, which may improve water absorption and electrolyte replacement.

When an increase in hydration of ingesta is a major goal of fluid therapy (e.g., to treat impactions), it is tempting to add a poorly absorbed solute to hold water in the gastrointestinal lumen. Magnesium sulfate has been traditionally used as an osmotic laxative because its absorption is thought to be limited and slow. Oral administration of magnesium sulfate (1 g/kg of body weight) is considered to be safe and effective (Freeman et al. 1992). However this dose is more than 6 times the dose needed by IV route to cause neuromuscular blockade and recumbency in horses (Bowen et al. 1970) and magnesium toxicosis has been reported in dehydrated horses treated with magnesium sulfate (Henninger and Horst 1997). Sodium sulfate (0.8 g/kg) has also been shown to produce cathartic effects in horses (Meyer et al. 1986) and may be associated with a lower risk of toxicosis. Traditionally, saline cathartics have been administered with a small volume of fluids followed or not followed by IV fluid therapy, but the effectiveness and safety of the administration of saline cathartics with large volumes of fluids by nasogastric tube have not been evaluated.

Solutions containing glycerol - Solutions containing glycerol are thought to increase blood osmolality favoring an increase in plasma volume, but their use is controversial. Theoretically, glycerol ingestion before exercise would benefit athletes trying to avoid the deleterious effects of dehydration caused by prolonged physical activity. However many studies in humans failed to demonstrate improvement in hydration status or performance. The effects of glycerol ingestion on gastric emptying and the potential for toxicity have not been fully investigated; however bloating, nausea and vomiting have been reported in humans (Wagner 1999). Oral administration of 2.4 ml/kg of body weight of glycerol in conjunction with electrolytes immediately before and during endurance exercise has been evaluated in horses, but the effects were similar to electrolytes alone (Dusterdieck et al. 1999, Schott et al. 1999).

Planning and monitoring enteral fluid therapy

The same criteria recommended for IV fluid therapy can be followed for enteral fluid therapy. Clinical and laboratory findings are the basis for selection of the appropriate fluid composition, volume and rate of administration (Rose 1981, Barton and Moore 1999, Corley 2001). However the margins for error may be significantly wider than for IV fluid therapy and close attention to fluid volume and composition is not so critical.

Based on studies evaluating water intake (Tasker 1967, Fonnesebeck 1968), the maintenance rate of fluid administration for normal horses has been estimated to be about 2.5 ml/kg/h (Corley 2001). However the ongoing fluid losses may be significantly higher as in cases of horses with diarrhea (Rose 1981). To calculate the initial rate of infusion, the water and

electrolyte deficits also have to be considered. The response to fluid therapy should be monitored frequently, and adjustments in fluid composition and infusion rate should be made as needed.

If hydration status cannot be restored or maintained with enteral fluid therapy such as in cases of severe colitis, IV fluid therapy must be administered. Intolerance to enteral fluid therapy may be manifested by gastric dilation and abdominal discomfort, which can be managed by slowing the rate of infusion. Although, in some cases, enteral fluid therapy may have to be discontinued.

Complications of enteral fluid therapy

Inadvertent administration of fluids into the respiratory tract - Horses with dysphagia (e.g., caused by tetanus or other neurologic diseases) or esophageal obstruction should not be allowed to drink because of the risk of fluid aspiration. Forced administration of oral fluids is also a risky procedure even in normal horses and should not be used. The administration of fluids through a nasogastric tube can also result in fluid administration into the respiratory tract if the tube is accidentally passed into the trachea. Usually the horse reacts vigorously when the tube is in the trachea, but depression due to disease or sedative administration can prevent the horse from reacting (Scarratt et al. 1998). Besides the absence of this characteristic reaction, other signs of correct intubation have to be verified to prevent inadvertent administration of fluids into the trachea.

Other accidents due to nasogastric intubation - Nasal bleeding can occur even with the gentlest maneuvers to pass the nasogastric tube. In almost all cases, bleeding is not severe enough to cause any problem and bleeding will stop without treatment. A tube with an appropriate gauge and a smooth tip lubricated with water-soluble jelly should be used in order to prevent bleeding. Proper restraint of the horse and careful handling of the tube, which should be advanced slowly through the ventral meatus, is also recommended (Adams 1970). Trauma to the pharynx, larynx, and esophagus may also occur, which can lead to nasal discharge of feed and saliva, dysphagia, and anorexia. Severe lesions can occur when a tube with sharp edges is used or when the tube is passed with rough maneuvers. The risk is increased when large diameter tubes are used, when the tube is kept in place for several days and when the horse resists intubation (Hardy et al. 1992).

Abdominal discomfort - If pain is observed immediately after fluid administration, excessive gastric distention should be suspected and the nasogastric tube should be used to empty the stomach. Large doses of fluids (more than 5 liters for a 200 kg horse, or more than 10 liters for a 500 kg horse) should be avoided to prevent excessive gastric distention. When even small volumes of fluids (e.g., 5 liters for a 500 kg horse) cause pain, pathologic conditions of the stomach such as gastric ulcers or impaction should be considered. In horses with intestinal obstruction or displacement, the increase in motility produced by the gastrocolic response may cause pain, which can be explained by an intestinal contraction moving over an impaction, or stretching the intestine and the mesentery (Steinebach and Cole 1995, Lopes et al. 1999). Pain produced by increased motility can be managed with drugs to relieve spasm (e.g. scopolamine, xylazine) or analgesics (e.g. flunixin).

Rupture of stomach or intestine - The administration of an excessive amount of fluids into the stomach may cause gastric rupture. Two cases of rupture after tube feeding have been reported (Todhunter et al. 1986) and one case of gastric rupture in a horse with gastric impaction has been attributed to enteral fluid therapy (Milne et al. 1990). If the horse is not heavily sedated or severely depressed, excessive gastric distention is likely to produce signs of pain and rupture may be prevented by prompt drainage of fluids through the nasogastric tube. In horses with severe impaction, the increase in intestinal motility produced by enteral fluid therapy may result in rupture as reported in a horse with cecal impaction (Lopes 1999). However the risk of cecal rupture in horses with cecal impaction is high even when enteral fluids are not used (Collatos and Romano 1993). Large colon or small colon rupture due to enteral fluid therapy has not been reported.

Electrolyte imbalances - Electrolytes and water are readily absorbed through the gastrointestinal mucosa and enteral fluid therapy can change electrolyte concentration in plasma (Lopes et al. 2002a). Close monitoring of plasma electrolytes should guide enteral fluid therapy. This is particularly important in horses that are not eating or when kidney function is abnormal.

Pulmonary edema - Horses seem quite tolerant to high volumes of fluids given by the IV route, and pulmonary edema is rare (Barton and Moore 1999). Although fluid therapy has not been extensively studied in horses, it is reasonable to believe that the risk of pulmonary edema may be even lower when fluids are given enterally. In an “overhydrated” horse, in addition to the activation of diuresis, water absorption by the gastrointestinal mucosa is likely to be reduced by several mechanisms. Thus the rate of delivery of fluids into the circulation is slowed down when enteral fluid therapy is used, while these natural mechanisms are bypassed when intravenous fluid therapy is used.

Conclusions

Enteral fluid therapy is an effective, practical and inexpensive treatment for horses with many clinical conditions. The gastrointestinal tract has an extraordinary capacity to move and absorb large volumes of water and enteral fluid therapy can produce both gastrointestinal and systemic hydration. Horses with normal gastrointestinal transit may tolerate large volumes administered in a short period of time, while some horses may require slower rates of administration and in some situations (e.g., ileus and gastric reflux), enteral fluid therapy is contraindicated. Rational judgment of clinical condition based on clinical and laboratory findings should be used to adjust fluid composition and rate of administration according to the horse's needs. More studies are needed to better define the guidelines for enteral fluid therapy in horses.

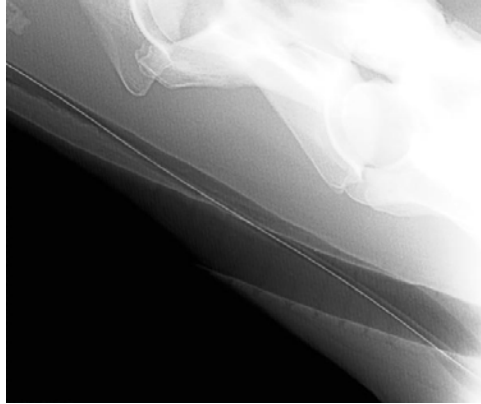


Figure 2.1 – Radiographic image of the neck (lateral view) of a horse with an enteral feeding tube in the esophagus. The radiopaque line in the tube can be seen. There is no superimposition with the trachea throughout the neck indicating that the tube had been correctly placed in the esophagus.

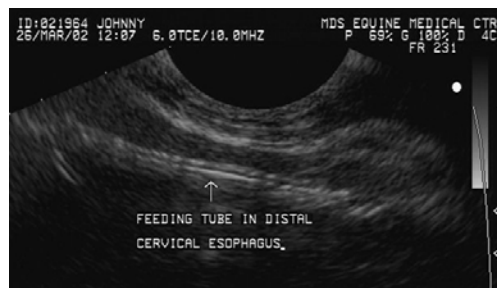


Figure 2.2 – Ultrasonographic image (longitudinal view) of the neck of a horse with an enteral feeding tube in the esophagus. The tube is indicated by the arrow.



Figure 2.3 – Endoscopic image of the pharynx of a horse with an enteral feeding tube in the esophagus.



Figure 2.4 – Administration of fluids via a large-bore nasogastric tube using a funnel.



Figure 2.5 – Administration of fluids via a small-bore nasogastric tube using a coiled line.



Figure 6 – Horse with a small-bore nasogastric tube eating hay.

8- Effectiveness and safety of laxatives for horses

Laxatives have long been administered to horses to treat impactions and many other clinical conditions (Teigland 1968). However only a few controlled studies investigating the safety and effectiveness of laxatives in horses have been published and in most cases toxicity or lack of any laxative effect were observed. Toxicity of high doses (0.65 or 1.00 g/kg) of sodium docusate (sodium dioctyl sulfosuccinate) by nasogastric tube was investigated in healthy horses that had been fasted overnight. All horses died after manifesting abdominal discomfort, diarrhea and dehydration. Postmortem examination revealed pronounced distention of the gastrointestinal tract with fluid and mucosal damage (Moffatt et al. 1975).

The effects of magnesium sulfate and sodium sulfate (1 g/kg as a 10% solution 2 h after the meal) were investigated in horses with cecal fistulas. Magnesium sulfate produced an increase in the flow of water through the ileocecal valve, while sodium sulfate reduced large intestine absorption of water and produced an increase in fecal hydration (Meyer et al. 1986).

In another study the effects of mineral oil on gastrointestinal transit and fecal hydration were investigated. Healthy horses were alternately treated with mineral oil (2 liters via nasogastric tube), flunixin meglumine (1.1 mg/kg IM), dipyron-scopolamine (20 mg/kg IV) or 0.9% sodium chloride (20 ml IM). At the same time 10 g of chromium oxide were administered via nasogastric tube. The horses received hay and grain twice daily and had free access to water and salt. With mineral oil there was an increase in fecal dry matter 24 and 36 h after administration, which was not seen with any other treatment. It was also observed that mineral oil produced a delay in the appearance of chromium in feces. These findings were interpreted as indications of a delayed gastrointestinal transit produced by mineral oil (Macoris 1989).

In the most complete study evaluating laxatives, healthy horses were alternately treated via nasogastric tube with 4 treatments: 6 liters of warm water, 2 different doses of magnesium sulfate (0.5 or 1.0 g/kg) in 6 liters of warm water and sodium docusate (50 mg/kg) in 6 liters of warm water. Horses were fed hay and grain divided in 3 daily meals and had free access to water and salt. It was observed that water consumption and mean retention time of transit markers (polyethylene glycol and plastic markers) were not affected by any treatment. The highest dose of magnesium sulfate produced significant increase in total weight of feces and fecal water excretion, while sodium docusate and the lower dose of magnesium sulfate did not have any laxative effect. It was also reported that water alone had some laxative effect (increased fecal output, fecal water, number of defecations and fecal hydration) and that sodium docusate toxicosis (colic and diarrhea 3 h after administration) was seen in one horse (Freeman et al. 1992).

The laxative effects of raw linseed oil and mineral oil were compared in healthy horses. Two doses of linseed oil (2.5 ml/kg of body weight) or mineral oil (10 ml/kg of body weight) were administered through a nasogastric tube 12 h apart. In all horses linseed oil produced watery diarrhea 12 to 36 h after the second dose and diarrhea lasted 36 to 48 h. Mineral oil produced only softening of feces, which was seen 6 to 12 h after the second dose and lasted less than 24 to 30 h. It was also observed that after linseed administration all horses had depression and anorexia, 4 horses had neutropenia, 3 horses had mild colic and one horse had increased liver enzymes. It was mentioned that one of the authors had been using linseed oil to treat small colon impactions successfully and that depression and anorexia was commonly seen. The authors suggested that the side effects seen with linseed oil were likely to be caused by mucosal irritation leading to colic and endotoxemia (Schumacher et al. 1997).

The laxative effect of psyllium has also been investigated in normal ponies submitted to laparotomy to place 10 g/kg of body weight of sand into the cecum. All ponies were fed hay and grain, while ponies in the treatment group received daily doses of psyllium (1 g/kg of body weight) with the grain or by nasogastric tube. Treatment with psyllium was started 3 days before surgery and was continued until the end of the experiment. Treatment with psyllium did not affect the amount of sand recovered from the gastrointestinal tract at necropsy 11 days later (Hammock et al. 1998).

Another study was conducted with sodium docusate to investigate if this drug would produce hematological changes in normal horses treated with a single administration of 20 mg/kg in 4 liters of warm water via nasogastric tube. Although no horse had any significant hematological change or sign of toxicosis, no sign of a laxative effect was observed either (Southwood et al. 1999).

Despite the lack of any beneficial effect and even toxicity demonstrated in most studies, recommendations in textbooks do not seem to be changing. For horses with impaction, current textbooks still recommend the use of sodium docusate, magnesium sulfate, sodium sulfate and mineral oil, while for sand impaction the choice is psyllium (Edens and Cargile 1997, Robinson 1997, Dabareiner 1998, Jones et al. 1998, Hanson 1999, Sullins 1999, Blikslager 2002). Thus current recommendations for the use of laxatives in horses are based mostly on tradition and anecdotal evidence.

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Chapter 3

Large experimental fistula of the right dorsal colon in horses

Abstract

Objectives – To devise a surgical technique and design of a cannula to create a large fistula in the right dorsal colon in horses.

Animals – 8 horses with no signs of gastrointestinal disease.

Methods – Surgery was performed in two steps: Step 1 - With the horses standing restrained in stocks and treated with sedatives and local anesthetics, a segment of the 16th rib and costal cartilage was removed, a circular defect was created in the muscular wall and the right dorsal colon was sutured to the muscular wall. The subcutaneous tissue and skin were closed over the attached colon. Step 2 - Two to 6 weeks after the initial surgery, the surgery site was injected with local anesthetics and a full thickness circular incision was made with a scalpel blade to create a fistula. A specially designed, natural rubber cannula (internal diameter, 5 cm) was then inserted in the fistula.

Results – A right dorsal colon fistula was successfully created in 7 horses. The cannula prevented leakage of ingesta and allowed collection of large samples of ingesta. One horse had colon torsion around the colopexy site before the colostomy could be performed. One of the 7 successfully fistulated horses had right colon displacement 43 days after cannula insertion.

Conclusions – A large fistula of the right dorsal colon can be created in horses to allow collection of large ingesta samples. A surgically created attachment of the right dorsal colon to the abdominal wall may predispose to large colon displacement.

Introduction

Experimental fistulas have been used in horses to study gastrointestinal morphology (Roberts and Hill 1974), physiology (Baker et al. 1969, Alexander 1970, Sellers et al. 1982), nutrition (Teeter et al. 1968, Lowe et al. 1970, Kienzle et al. 1997), pathophysiology of diseases (Lowe et al. 1980, Sellers et al. 1982, Krueger et al. 1986) and pharmacology (Lowe et al. 1980, Sellers et al. 1982, Engelking and Mariner 1985, Davison et al. 1986, Van Duijkeren et al. 1996). Fistulas of the gastrointestinal tract permit the collection of ingesta samples from a live animal. Also, fistulas make other procedures possible such as to insert probes to measure variables in the gastrointestinal lumen (e.g., pH, pressure) (Sellers et al. 1982), to administer substances (e.g., markers of gastrointestinal transit, nutrients) (Ralston and Baile 1982), or to insert a device to produce experimental obstruction (Lowe et al. 1980, Sellers et al. 1982). Since data can be collected from the same animal at different times, changes produced by diverse experimental conditions (e.g., diets, feeding protocols, medical treatments) can be compared while controlling for individual variation. Thus, gastrointestinal fistulas allow completion of studies with a small number of animals. Cecal fistulas have been created in the majority of equine studies. There are reports of the use of experimental fistulas of the dorsal colon in which techniques for small diameter fistulas (internal diameter, <1.5 cm) were described (Alexander 1970, Wilkins and Lowe 1993). In preliminary studies with a larger fistula (internal diameter, 2.5 cm diameter),

only small samples could be collected and the water content of ingesta was altered during sampling (White & Temple, 1993 unpublished data). This suggests that a fistula larger than 2.5 cm would be necessary to collect representative samples of colonic ingesta. The objective of this study was to devise a surgical technique and to design a cannula to create a large fistula (internal diameter, 5 cm) in the right dorsal colon of horses.

Material and methods

Eight horses with no signs of gastrointestinal disease and no history of gastrointestinal dysfunction for the last 6 months were selected: age, 6 to 17 years old (mean, 10.75); gender, 6 castrated males and 2 females; breed, 6 Thoroughbred and 2 Quarter Horse; body weight ranging from 452 to 580 kg (mean, 512.49). A cannula made of natural rubber was created for this study (Figure 3.1).

Surgery I (colopexy) - The procedures for creating right dorsal colon fistulas were approved by the Animal Care Committee at Virginia Tech. Hay and grain were withheld for 48 h and 12 h, respectively. Each horse was restrained in stocks and the skin on the right side of the thorax and abdomen clipped and aseptically prepared for surgery. In 3 horses a urinary catheter was inserted to drain urine and prevent change in posture for urination during surgery. Sedation was produced with repeated IV boluses of detomidine and xylazine (3 horses) or with acepromazine (20 mg IV) followed by continuous IV infusion of detomidine (15 mg diluted in 500 ml of 0.9% NaCl IV) (5 horses). Twenty ml of bupivacaine 2% was injected 5 cm abaxial to the dorsal midline at each intercostals space to anesthetize the 14th, 15th, 16th and 17th intercostal nerves. The depth and location of the rib was estimated with the aid of ultrasonography and an 8 inch spinal needle was advanced into the intercostal space. Five milliliters of the local anesthetic were injected at a level 1 cm ventral to the dorsal surface of the rib. The needle was gradually withdrawn while 5 ml were injected into 3 additional sites 1, 2 and 3 cm dorsal to the first site. In addition to the intercostal block, approximately 60 ml of bupivacaine were injected in the subcutaneous tissue and muscular wall in a 20 cm long transverse line caudal to the last costochondral joint. When the intercostal block was ineffective, infiltration with bupivacaine close to the site of the incision was used as needed. Butorphanol (5 mg IV) was administered approximately 5 minutes before incising the skin.

A 18-20 cm curved skin incision was made over the ventral quarter of the right 16th rib and dorsal segment of costal cartilage. The cutaneous trunci muscle, subcutaneous tissue and external oblique muscle were incised with the scalpel to expose the 16th rib (Figure 3.2A). At the 15th and 16th intercostal spaces, the intercostal muscles were incised with scissors at their attachment to the 16th rib for a length of 10 to 14 cm to free the ventral section of the rib and the dorsal section of the costal cartilage. The dissected rib and costal cartilage segment was cut with bone-cutting forceps (Figure 3.2B) and removed. Blunt dissection using scissors was used to separate the retroperitoneal fat and expose the peritoneum, which was then penetrated by digital pressure.

The peritoneal cavity was explored and the right dorsal colon identified. A small fold of the lateral wall of the right dorsal colon was brought to the surgical wound and the amount of tension that would be produced by suturing the right dorsal colon to the abdominal wall was evaluated. Two semi-circle sections of the external oblique muscle were removed with scissors

to create a circular defect 6-7 cm in diameter (Figure 3.2C). This defect was created in order to maximize the area of the adhesion of the right dorsal colon to the abdominal wall. When necessary a segment of the 17th rib and/or its costal cartilage was also resected to increase the size of the defect in the abdominal wall. A fold of the lateral wall of the right dorsal colon was exposed and held in place with Allis tissue forceps. The colon was sutured to the borders of the semi-circular defects in the external oblique muscle using #2 polyglactin (Vicryl, Ethicon, Inc.) in an interrupted simple pattern. The remaining linear defects in the external oblique muscle dorsally and ventrally to the circular defect were closed with #2 polyglactin suture in a simple interrupted pattern (Figure 3.2D). The subcutaneous tissue and cutaneous trunci muscle were apposed with #2-0 polyglactin using a simple continuous pattern. The skin was closed using #2-0 nylon in a simple interrupted pattern.

Trimethoprim-sulfamethoxazole (TMS) (30 mg/kg PO) was administered perioperatively (1 h before surgery and 12h after the first dose). Four doses of phenylbutazone were administered after surgery (4 mg/kg IV 12 h). Rectal temperature was monitored twice daily and horses with fever were medicated with TMS (30 mg/kg PO 12 h) until 2 normal temperatures had been recorded. The surgical wound was inspected daily and cleaned with chlorhexidine when necessary. The skin sutures were removed 14 days after surgery.

Surgery II (stoma creation and cannula implantation) - Two to 6 weeks after the first surgery, the colostomy was created. The site of the first surgery was evaluated ultrasonographically to assess the extent and location of the adhesion of the right dorsal colon to the abdominal wall (Figure 3.3). TMS (30 mg/kg PO) was administered 1 h before surgery. The horse was restrained in stocks and the skin at the first surgery site was clipped and aseptically prepared for surgery. The horse was sedated with detomidine (5 mg IV) and the subcutaneous tissue and muscular wall circling the site of the adhesion was injected with 2% bupivacaine. A scalpel was used to create a 5 cm diameter circular defect in the skin. Subcutaneous tissue and fibrous scar tissue were excised with a scalpel blade and scissors and were removed to create a circular cone-shaped defect to expose a soft layer with the approximate thickness of the colon wall. A circular full thickness incision approximately 4 cm in diameter was then created in the colon wall with the scalpel. The margins of the defect (from skin to colonic mucosa) were stretched with Army-Navy retractors and the tubular part of the cannula was folded, held tightly with a sponge forceps and inserted. After removing the sponge forceps and retractors, the cannula was unfolded, the external shaft was inserted and the cannula was closed with the plug and cap. The colostomy site was cleaned with water daily. Identification of the fistulated segment was made by inspection with a 9 mm diameter flexible endoscope passed through the cannula and at necropsy (Figures 3.4A, 3.4B and 3.4C). These horses were used to study the effects of diets, fluid therapy and laxatives on water content of ingesta. After completing the experiments, all horses were euthanized with IV administration of pentobarbital (39 g) and phenytoin (5 g) (Beuthanasia-D Special, Schering-Plough Animal Health, Corp.).

Results

The surgical procedures were well tolerated and signs of pain were not observed. However all horses shifted weight repeatedly from one hind limb to the other during surgery producing significant distortion of the surgical field. Continuous detomidine infusion and

continuous evacuation of the bladder with a urinary catheter decreased the weight shifting markedly. In one horse, the intercostal space was narrower and a segment of the 17th rib and costal cartilage was also removed.

Swelling and formation of a fluid pocket between the cutaneous trunci muscle and the colon wall was detected after the initial surgery in all horses by palpation and/or ultrasonography. One horse had wound infection (pronounced swelling of the surgical site, depression, reduced appetite and fever) which was managed by surgical drainage. In all horses the surgical wound healed and the surgical site was minimally inflamed 2 to 6 weeks after surgery. At that time, ultrasonographic examination revealed a 5 to 7 cm in diameter adhesion of a segment of the large intestine to the surgical site. Thirty days after surgery one horse had moderate colic, which was non-responsive to analgesics. A tight intestinal band felt by rectal examination was suggestive of a colon displacement or torsion. Euthanasia was performed using the method previously described and at necropsy the large colon was found twisted at the point of the colopexy.

A right dorsal colon fistula was created in 7 horses. In 2 horses the peritoneal cavity was entered at a point dorsal to the adhesion, but gross contamination with ingesta did not occur. In both cases the defect was small (1-2 cm) and was closed with a single cruciate suture with #0 polyglactin. One additional dose of TMS was administered to these 2 horses 12 h after the first dose. The next morning one of these horses was febrile, but neither horse developed signs of peritonitis. Fever was also observed in two horses in which cannula implantation was uncomplicated. During the first 2 weeks after cannula implantation, moderate swelling was seen in 4 horses while the other 3 horses had minimal swelling.

Studies on the effects of diets and medical treatments on colonic hydration were conducted with these horses. Cannula manipulation was well tolerated by all horses. Interference with the cannula by biting or rubbing it against the wall or hay net was commonly seen. Occasionally interference with the cannula resulted in removal of the plug or in some cases the whole cannula. Delay in reinsertion of the cannula for more than 2 hours resulted in marked wound contraction. In these cases administration of xylazine or detomidine was required to produce analgesia and abdominal relaxation and to facilitate cannula insertion. When necessary a scalpel was used to remove granulation tissue and enlarge the stoma. Interference with the cannula was more frequent 6 weeks or more after cannula placement. Repeated episodes were managed by fitting a neck cradle on the horses.

One horse had signs of moderate colic 43 days after fistula creation. A large colon displacement was identified on rectal palpation, and the horse was euthanized using the method previously described. At necropsy the colopexy was intact and there was a right dorsal displacement of the large colon. Sixty nine days after cannula placement one horse had severe swelling at the colostomy site and the cannula was occluded by swollen colonic mucosa. This horse had been interfering with the cannula repeatedly for 3 days before signs of severe inflammation were seen. Removal of the cannula was followed by remission of swelling and quick contraction and healing of the stoma. Seventeen days after cannula removal, an attempt to create a new stoma failed.

The cannula completely sealed the colon when it was closed and allowed large samples of ingesta to be easily collected (Figures 3.5A, 3.5B and 3.5C). The plug had a piston like function, which facilitated sample collection: The vacuum created by removing the plug filled the cannula with ingesta. Depending on factors such as diet and position of the horse, ingesta would flow spontaneously (Figure 3.5C) with the cannula open or a scoop was used to evacuate

the content of the cannula. In the 5 horses with a right dorsal colon fistula that did not have any complications requiring premature cannula removal or euthanasia, the fistula was maintained for 98 to 162 days (mean, 119.8).

Discussion

Although the use of general anesthesia to create right dorsal colon fistulas in horses has been described (Alexander 1970, Wilkins and Lowe 1993), standing surgery was performed based on the assumption that it would avoid distortion of abdominal topography and allow better evaluation of the appropriate site for colopexy. Intolerance to manipulation of viscera and repeated changes in posture are drawbacks of standing surgery. Administration of detomidine and butorphanol effectively prevented visceral pain, but produced significant ataxia. The diuretic effects of detomidine (Gasthuys et al. 1987) resulted in frequent urinations, which further contributed to postural changes. Continuous infusion of detomidine to produce steady blood concentrations (Hainisch 2001) minimized changes in posture and frequency of urinations. Continuous evacuation of urine with a catheter was also helpful in avoiding changes in posture for urination.

Appropriately placed sutures to reduce the dead space might have helped to avoid seroma formation. However, in this group of horses, the deposit of fluids over the colonic serosa helped to guide dissection at the time of stoma creation. An alternative to the technique used in this study would have been to create a full thickness circular defect in the abdominal wall similarly to what has been described for rumen fistulas (Mogha and Bhargava 1979). In this case the colon would have been sutured directly to the skin leaving the serosa exposed and allowing maximal drainage to prevent seroma formation and infection. This technique would also have made the site for stoma creation obvious at the time of the second surgery. The closed approach was chosen because the horses were to be maintained in an outdoor environment. It is likely that the infection of deep structures seen in one horse after colopexy was performed resulted from contamination brought by a deep bite into the colonic lumen. This complication has been previously described in horses submitted to colopexy (Hance and Embertson 1992).

In all cases the adhesion was relatively small and the margin for error while creating the stoma was narrow. Creation of a larger adhesion would be desirable but was limited by the morphologic features at the site of the fistula and would require additional trauma including the resection of a segment of another rib. Formation of a tunnel of granulation tissue bridging the skin to the mucosa (Figure 3.4C) effectively prevented severe infection in all but one horse. It is likely that the repeated interference with the colostomy by the horse contributed to severe infection in that case. Alternatively pruritus could have been simply an early sign of infection, which could have resulted from another factor (e.g., pressure necrosis produced by the cannula).

Pruritus at the colostomy site could be explained by the persistent low grade inflammation and resulted in interference with the cannula. The use of a cannula made of less reactive material such as medical grade silicon would likely have helped to prevent pruritus. However the design and consistency of the cannula were appropriate as the cannula could be easily folded to facilitate insertion, leakage of gas and ingesta was effectively prevented and large samples of ingesta could be easily collected.

The number of large colon displacements in this group of horses (2/8) seems high. Despite the existence of a natural attachment of the colon to the dorsal abdominal wall close to

the site where the fistula was created, the colopexy may have interfered with colonic positioning and movement predisposing to large colon displacement. However, due to the small number of horses, the possibility that the displacements were not associated with the colopexy cannot be discarded.

Since rapid contraction and healing of the colostomy occurs after cannula removal, immediate insertion after accidental removal of the cannula is necessary or a surgery to recreate the fistula will be needed. Complete recovery of horses with an experimental fistula of the right dorsal colon is possible, although the attachment created from the right dorsal colon to the abdominal wall will remain after fistula closure. Incomplete healing with formation of a chronic enterocutaneous fistula is unlikely to be a problem due to the location of the right dorsal colon fistula. Enterocutaneous fistulas have been reported after accidental or surgical lesions of the ventral abdomen (Watson and Harding 1972, Brown and Meagher 1978, Freeman et al. 1988, Bristol 1994), but the ventral position seems to play an important role promoting continuous drainage of ingesta and preventing healing. If a chronic fistula forms it can be successfully managed by curettage or surgical resection of the fistulous tract (Watson and Harding 1972, Brown and Meagher 1978, Freeman et al. 1988, Bristol 1994).

A large right dorsal colon fistula to allow collection of large samples of ingesta can be created in horses. This model was successfully used to study the effects of diets and medical treatments on ingesta hydration, and can be useful for further studies on nutrition, gastrointestinal physiology, and pathophysiology and treatment of gastrointestinal diseases. Concerns with animal welfare as well as the inherent difficulties and risks of these procedures have to be considered, but may be outweighed by the fact that valuable information can be obtained. Standing surgery can be performed, but appropriate restraint in stocks with continuous infusion of intravenous sedatives, effective regional anesthesia, and continuous drainage of urine are indicated in order to avoid discomfort for the horse and minimize distortion of the surgical field due to posture changes. Further improvements on the surgical technique and on cannula design may be possible.

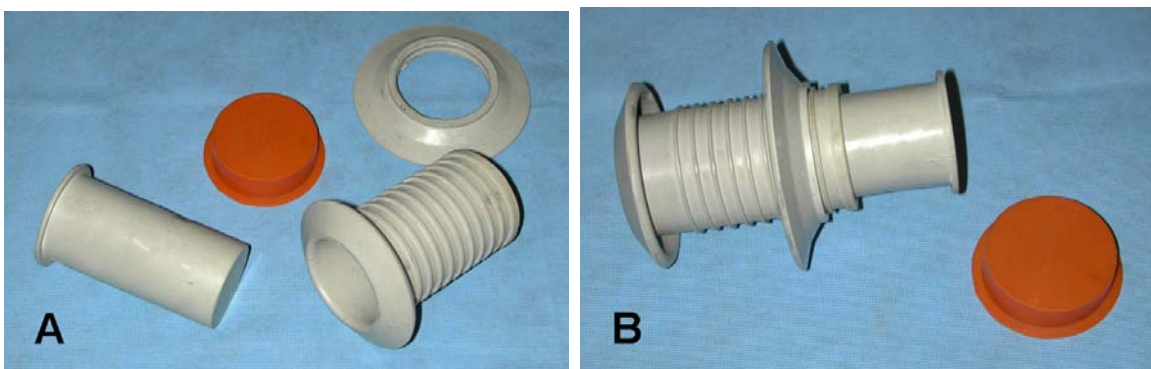


Figure 3.1 – Custom made natural rubber cannula (Kehl Industria e Comercio Ltda, São Carlos, SP, Brazil) composed of 4 parts: A- Tube (length, 10 cm; internal diameter, 5 cm) with a shaft, external shaft (external diameter, 10 cm), plug (external diameter, 4.8 cm), and cap. B- Cannula assembled.

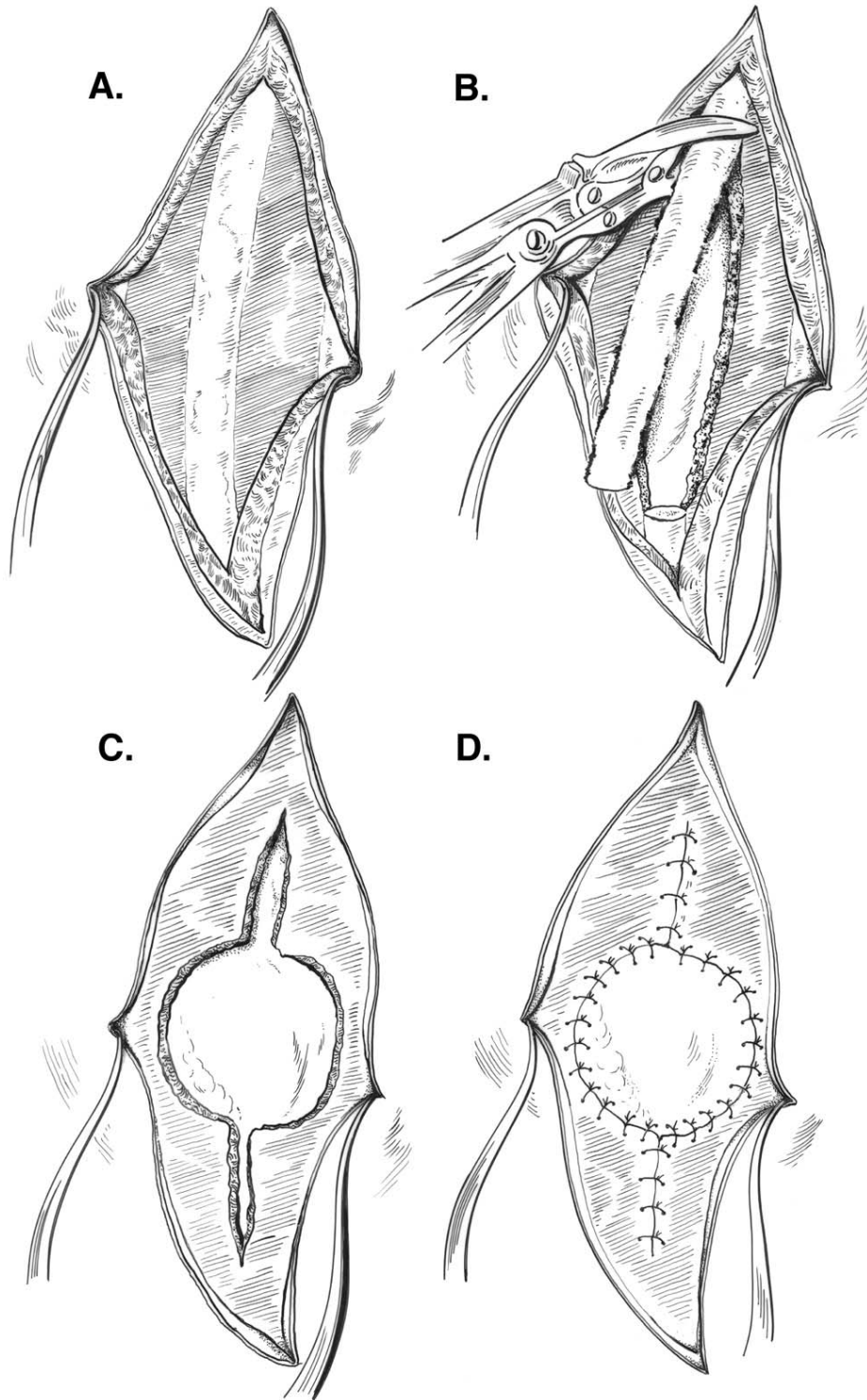


Figure 3.2 – Surgical technique to create a right dorsal colon fistula: A- Incision through skin, cutaneous trunci muscle and external oblique muscle to expose the 16th rib. B- Resection of 16th rib with a bone cutting forceps. C- Incised external oblique muscle. D- Fold of the right dorsal colon sutured to the external oblique muscle. The linear defects in the muscle were also sutured.



Figure 3.3 – Ultrasonographic image showing the adhesion of the right dorsal colon to the abdominal wall 3 weeks after colopexy.

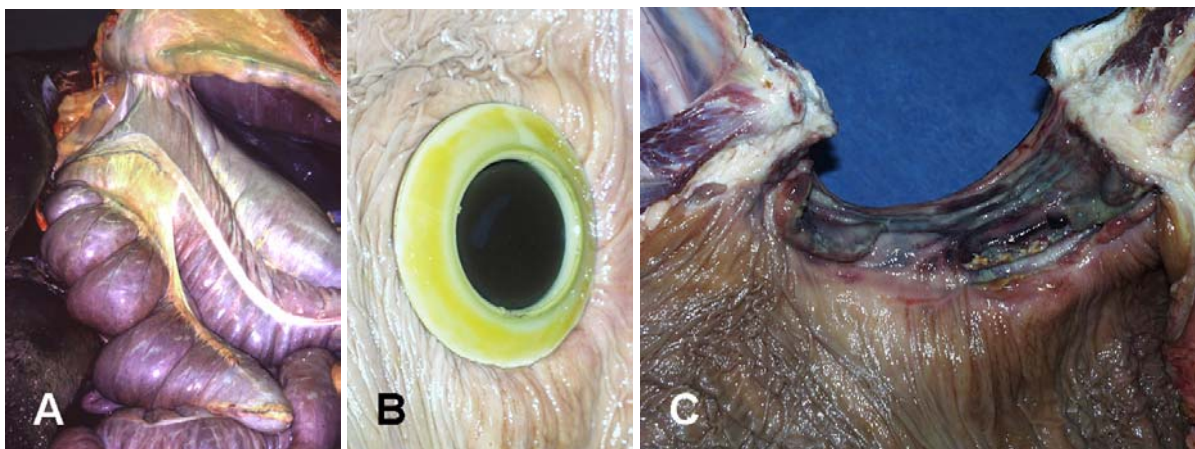


Figure 3.4 – Necropsy of a horse with right dorsal colon fistula 121 days after cannula implantation: A- Right dorsal colon adhered to the abdominal wall. B- View of the colonic lumen showing the internal shaft of the cannula and colonic mucosa. C- Colostomy site showing thick fibrous tissue bridging the colonic mucosa to the skin.

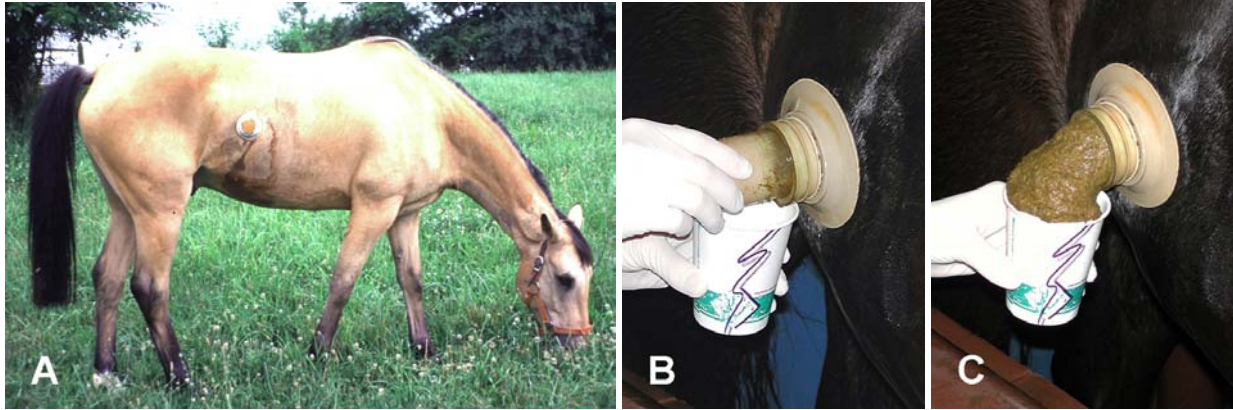


Figure 3.5 – Right dorsal colon fistula: A- Horse with the cannula closed. B- Opening the cannula. C- Collecting samples.

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Chapter 4

Hydration of colonic ingesta and feces in fistulated horses fed large grain meals

Abstract

Objective – To assess changes in systemic hydration, plasma electrolytes, ingesta and fecal hydration and gastrointestinal transit in horses fed large grain meals.

Animals – Six horses with an experimental fistula in the right dorsal colon.

Procedure – The horses were submitted to 3 feeding regimens: 1) Hay diet - fed just hay free choice; 2) Transition hay/hay+grain – after being fed just hay for at least 5 days, fed hay free choice and 4.55 kg meals of sweet feed twice daily; 3) Hay+grain diet – adapted for at least 5 days to hay free choice and 4.55 kg meals of sweet feed twice daily. A clinical examination was performed and blood, ingesta and fecal samples were collected every 6 hours for 48 h.

Results – No horse had any sign of pain or gastrointestinal dysfunction, endotoxemia or laminitis. Grain ingestion had no effect on PCV or plasma protein, but produced changes within the normal limits on plasma electrolytes. Grain ingestion produced marked changes in ingesta and feces: ingesta water content decreased; ingesta became foamy and less dense; spontaneous flow of ingesta was seen when the cannula was open; ingesta volume expanded when heated in the oven; feces became fetid and less formed. Furthermore the amount of large fiber particles in ingesta appeared to be reduced when grain was fed.

Conclusions and clinical relevance – The large amounts of hydrolyzable carbohydrates provided by grain explain the changes in ingesta and feces. Large grain meals may increase the risk of tympany and large intestine displacement.

Introduction

Colic causes more deaths in horses than any other group of diseases (Traub-Dargatz et al. 1991, Traub-Dargatz et al. 2001). In most cases the cause of colic is unknown, but anecdotal observations and a few epidemiological and experimental studies have incriminated current practices of feeding large meals and large amounts of grain as risk factors for colic (Clarke et al. 1990a, Reeves 1996, Tinker et al. 1997). Modifications in the gastrointestinal flora and acidification of the cecal lumen have been repeatedly documented in horses fed large amounts of grain (Kern et al. 1973, Willard et al. 1977, Garner et al. 1978, Moore et al. 1979, Goodson et al. 1988, Moore and Dehority 1993, Collinder et al. 2000, Fombelle et al. 2001, Julliand et al. 2001). Despite the potential mechanisms by which intestinal flora disturbance and acidosis could lead to colic (e.g., mucosal barrier disruption, endotoxemia, abnormal motility patterns) (Krueger et al. 1986), grain ingestion may also affect gastrointestinal function by other mechanisms. Experimental administration of one or two 1 h duration meals per day has been shown to produce a state of transient dehydration with activation of the renin-angiotensin-aldosterone system (Argenzio and Stevens 1975, Clarke et al. 1988, Houpt et al. 1988, Clarke et al. 1990b). Based on those findings it was proposed that feeding a few large meals could cause post-prandial dehydration of colonic ingesta and lead to large colon impaction, which could initiate other

forms of colic such as large colon displacements and volvulus (Clarke et al. 1990a). However this theory was based on artificial feeding regimens not used for domestic horses. Furthermore the changes in water content of colonic ingesta produced by large meals have not been documented. This experiment was conducted to study the effects on water content and other variables of equine colonic ingesta and feces produced by a common feeding regimen: A large grain meal every 12 h while grass hay, water and salt were offered free choice. The effects of abrupt introduction of this feeding regimen were also investigated.

Material and methods

This study was approved by the Virginia-Tech Animal Care Committee. Six horses with a right dorsal colon fistula were used in this study: 5 geldings and 1 mare; 4 Thoroughbred and 2 Quarter Horse; 6 to 17 years old (mean, 10.1 years old); weighing 452 to 580 kg (mean, 517.9 kg). The right dorsal colon fistula had been created 11 to 34 days (mean, 23.3 days) before the start of the experiment. The technique to create the right dorsal colon fistula has been described elsewhere (Lopes et al. 2002).

Feeding regimens - During the entire study the horses were kept in stalls with free access to 2 buckets each containing 12 liters of water, a salt block ^a and orchard grass hay. The study was conducted in a crossover design to compare 3 feeding regimens each for 48 hours: 1) Hay - fed just orchard grass hay free choice for at least 5 days; 2) Transition hay / hay+grain – after being fed just orchard grass hay for at least 5 days, fed hay free choice and twice daily 4.55 kg meals of sweet feed (6.5, 18.5, 30.5, 42.5 h after the start of the observation period); 3) Hay+grain – fed orchard grass hay free choice and 2 daily 4.55 kg meals of sweet feed (on the same schedule as the transition diet), after being adapted to this diet for at least 5 days. The order of the feeding regimens was randomized and the trials were started every Monday for 3 consecutive weeks. A single batch of orchard grass hay and a single brand of grain ^b were used for the whole study. A sample of grain from a pool of samples from 3 different bags and a sample of hay from a pool of samples from 10 different bales were collected for analysis.

Clinical assessment and sample collection - For clinical examination and sample collection the horses were restrained in stocks. Starting immediately before and during the 48 h observation period, a clinical examination was performed every 6 hours. At the same times, blood was collected from a catheter in the jugular vein, ingesta was collected from the right dorsal colon fistula and fecal samples were collected from the rectum or from the floor immediately after defecation. Immediately after performing the first clinical examination and collecting the first set of samples (time 0), cobalt-EDTA (0.04 g / kg of body weight) dissolved in 1 liter of water were administered by nasogastric tube ^c. Cobalt-EDTA was prepared as previously described (Uden et al. 1980). During the 48 h trials environmental temperature and humidity were recorded every 6 h ^d, and water consumption was estimated by measuring the volume needed to refill the buckets. During the trials when grain was fed, the weight of the grain that had not been eaten was recorded every 6 h.

Sample processing and analyses - Blood samples were immediately analyzed for PCV using the microhematocrit technique and plasma protein using a refractometer ^e. Plasma was pipetted

off after 10 minutes of centrifugation at 1124 xg and frozen at -70°C for posterior analysis of electrolytes. Automatic analyzers with ion specific electrodes were used to measure plasmatic concentration of sodium, potassium, chloride, total calcium, total magnesium^f and ionized calcium^g. Subjective assessment of some characteristics of ingesta and feces (odor, color, viscosity, presence of large pieces of fiber, oats and gas bubbles) were performed while samples were being collected and processed. Ingesta and fecal samples were divided in 2 aliquots: from one aliquot, the liquid phase was extracted by filtration in gauze and frozen at -70°C for posterior analysis of cobalt concentration; the other aliquot was weighed immediately after collection, dried in an oven^h at 90°C and repeatedly weighed until no change in the weight was detected. Ingesta and fecal hydration were calculated using the formula: $100 \times (\text{wet weight} - \text{dry weight}) / \text{wet weight}$. Cobalt concentration of the liquid phase of ingesta and feces was measured by atomic absorption spectrophotometryⁱ. The grain and hay samples were sent to a reference laboratory (DHI Forage Testing Laboratory, 730 Warren Rd, Ithaca, NY 14850). Samples of ingesta collected at time 0 from two horses (numbers 5 and 6) while fed hay and hay+grain (feeding regimens 1 and 3) were used to calculate ingesta density using the formula: $\text{density} = \text{weight of the sample} / \text{volume of the sample}$.

Statistical analysis - The MIXED procedure of the SAS System^j was used to perform a mixed model repeated measures analysis of variance to test for effects of diet, time, and diet by time interaction while controlling for horse and period effects. Significant interactions were further investigated using the SLICE option to test for the simple main effect of diet within each time. The mean retention time of cobalt in ingesta and feces was calculated using the noncompartmental approach ($\text{MRT} = \Sigma t_i c_i / \Sigma c_i$ where t_i = time and c_i = cobalt concentration) (Riviere and Williams 1999). For this variable the MIXED procedure of the SAS System^j was used to perform a mixed model analysis of variance to test for effects of diet.

Results

The composition of the hay and grain samples is presented in Table 4.1. The amount of grain fed daily ranged from 1.56 to 2.03% of body weight (mean, 1.76%). The amount of dry matter fed daily in the grain ranged from 1.37 to 1.78% of body weight (mean, 1.55%). The amount of nonstructural carbohydrate provided by grain ranged from 0.60 to 0.77% of body weight per day (mean, 0.68% of body weight/day). The temperature and relative humidity recorded during the experimental periods ranged from 10 to 31°C (mean, 18.6°C) and 23 to 95% (mean, 60.6%) respectively. All horses tolerated the diet and changes in diet. No horse had any sign of colic, diarrhea, dehydration (prolonged skin tent, prolonged capillary refill time, discoloration of mucous membranes), endotoxemia (prolonged capillary refill time, discoloration of mucous membranes or fever), or laminitis (abnormal hoof temperature, abnormal pulse in the digital arteries or lameness). Usually, the horses consumed the whole grain meal within 1 or 2 h, although small amounts (less than 1/2 kg) of grain were occasionally left for more than 6 h. It was evident that the horses consumed less hay when grain was fed.

Water intake was not affected by diet, only by time (Figure 4.1). When the horses were fed just hay, rectal temperature and heart rate were lower than when the horses were adapted to the hay and grain diet (Figures 4.2). Despite the feeding regimen the means of PCV, plasma protein and plasma electrolytes were always within normal limits. There was an effect of time,

but no effect of diet on PCV, plasma protein concentration and plasma potassium concentration (Figure 4.3 and 4). Plasma sodium concentration did not change with time, but there was an effect of diet and diet by time interaction (Figure 4.4). Plasma chloride concentration was significantly higher when the horses were fed the transition diet and there was diet by time interaction, but there was no change with time (Figure 4.5). Plasma magnesium concentration was significantly higher when the horses were adapted to the hay and grain diet than with other 2 feeding regimens (Figure 4.5). The only change on plasma total calcium concentration was seen when the horses were on the transition hay / hay and grain regimen. Total calcium was increased 18 and 30 h after the beginning of the 48 h observation period (Figure 4.6). The only change in plasma ionized calcium concentration was an increase after the first grain meal when the horses were in the transition hay / hay and grain regimen (Figure 4.6).

When the horses were fed only hay, ingesta was fetid, had an olive green color, minimal amount or no visible gas bubbles and 2 distinct phases: a liquid phase, which appeared to have low viscosity (similar to water), and a solid phase composed of pieces of plant fiber (Figure 4.7). Feces had a characteristic sweet smell, a dark olive green color and the fecal balls were well formed. Feeding grain altered the properties of ingesta and feces: ingesta appeared to have less fibrous material and acquired more yellow tones; ingesta had a more homogenous appearance (less distinct separation between the liquid and solid phases) (Figure 4.7) and the liquid phase appeared more viscous, although the overall ingesta viscosity did not appear to be changed; ingesta became foamy and flowed spontaneously when the cannula was open (Figure 4.8); ingesta expanded when heated in the oven (Figure 4.8); the water content of ingesta was significantly decreased (Figure 4.9), although the water content of feces did not change (Figure 4.9); feces acquired more brown tones and became fetid and the fecal balls were less formed or absent; fecal liquid appeared more viscous and had gas bubbles. In the two horses from which ingesta density was measured, grain ingestion produced a decrease in ingesta density from 0.92 and 0.93 kg/L to 0.64 and 0.66 kg/L respectively. When the horses were adapted to the hay and grain diet (feeding regimen 3), the curves of cobalt concentration in ingesta and feces were shifted to the right (Figures 4.10) and the mean retention time of cobalt in feces was increased (Figure 4.11). However diet did not have any effect on the mean retention time of cobalt in colonic ingesta (Figure 4.11). When the horses were submitted to the transition hay / hay+grain regimen, changes produced by the grain were gradual (Figures 4.2 and 4.9). During this feeding regimen, the change in ingesta appearance (including the presence of oats in ingesta) were first noticed 18 to 24 h after the beginning of the 48 h observation period (about 11 ½ h to 17 ½ h after the first grain meal was offered), while the changes in fecal appearance (including the presence of oats in feces) were first noticed 18 to 30 h after the beginning of the 48 h observation period (about 11 ½ h to 23 ½ h after the first grain meal was offered).

Discussion

This study demonstrated that horses eating large grain meals can have marked changes in ingesta and feces. These effects are likely the result of the ingestion of large amounts of hydrolyzable carbohydrate and small amounts of fiber. The reduced intake of hay when grain was fed was expected. In this study the amount of dry matter provided by grain ranged from 68.5 to 118.7% of the predicted dry matter intake for non-working non-pregnant horses (1.5 to 2.0% of body weight) (NRC 1989). Reduced intake of hay in horses with access to grain has been

previously reported (Danielsen et al. 1995). Although several studies have shown that horses eating grain consume less water (Fonnesbeck 1968, Sufit et al. 1985, Cymbaluk 1989, Danielsen et al. 1995, Warren et al. 1999), this trend was not observed in this study as well as in a previous study conducted by Yoder et al. (Yoder et al. 1997). The higher rectal temperature in horses adapted to the grain diet may be explained by large amounts of rapidly fermentable carbohydrates present in the gastrointestinal tract for the whole 48 h observation period. It is likely that a large portion of the hydrolyzable carbohydrates present in grain was not digested in the small intestine and became available for fermentation in the large intestine (Potter et al. 1992). Fermentation is known to result in more heat production than hydrolysis (Kronfeld 1996). The increased heart rate seen when the horses were adapted to the grain diet may be explained by the large amounts of feed available for digestion in the gastrointestinal tract during the 48 h observation period. At least in humans the presence of food within the gastrointestinal tract produces a significant increase in heart rate (Muller et al. 1992). Alternatively, subclinical discomfort due to ingestion of large amounts of grain could have contributed to the increase in heart rate.

In this study, large grain meals had no effect on PCV and plasma protein even when the horses were not adapted to grain intake. However a transient increase in PCV and plasma protein lasting less than 3 hours after ingestion of large meals has been reported by others (Kerr and Snow 1982, Youket et al. 1985, Clarke et al. 1988, Houpt et al. 1988, Clarke et al. 1990b, Jansson and Dahlborn 1999). There are two facts that may explain why postprandial dehydration was not seen in the current study: 1- Blood samples were only collected 5 ½ and 11 ½ h after the large grain meals were offered, while in previous studies a postprandial increase in PCV and plasma protein lasted less than 3 h (Kerr and Snow 1982, Youket et al. 1985, Clarke et al. 1988, Houpt et al. 1988, Clarke et al. 1990b, Jansson and Dahlborn 1999); 2- A more continuous eating pattern was produced in this study since the horses had free access to hay, while in previous studies extreme conditions were created where the animals were allowed to eat one or two daily meals in one or two hours (Kerr and Snow 1982, Youket et al. 1985, Clarke et al. 1988, Houpt et al. 1988, Clarke et al. 1990b, Jansson and Dahlborn 1999). Although the mean concentration of sodium, potassium, chloride, calcium, ionized calcium and magnesium were always within normal limits, some effects of diet were observed. These findings suggest that changes in plasma electrolyte concentration may be important in the pathogenesis of gastrointestinal dysfunction caused by ingestion of large grain meals.

The striking changes in the physical properties of ingesta observed when these horses were fed grain were similar to what has been described for ruminants with frothy bloat. This condition is characterized by formation of frothy ruminal content and occurs in ruminants fed large amounts of hydrolyzable carbohydrates, particularly after an abrupt change to a diet of large amounts of grain. The availability of large amounts of rapidly fermentable carbohydrates in the rumen leads to formation of large amounts of gases and excessive production of substances that increase the viscosity of the ruminal liquid (e.g., microbial mucopolysaccharides) and stabilizes the froth (Cheng et al. 1998). Although the pressure in the colon was not measured in this study, the observation of spontaneous flow of ingesta through the open cannula when the horses were fed grain suggests an increase in intraluminal pressure. Both spontaneous flow of ingesta through a ruminal fistula and increased intraruminal pressure have been documented in ruminants with frothy bloat (Cheng et al. 1998).

The lower density and the frothy appearance of ingesta when grain was fed indicated that there was an increased amount of gas. These findings support previous reports of the association

between ingestion of large amounts of rapidly fermentable carbohydrates and tympany (White 1990). It is also likely that gassy and less dense ingesta may favor abnormal positioning of the large intestine and predispose horses fed large amounts of grain to large intestine displacements and volvulus. This association has been suggested by others (Fischer and Meagher 1986, Snyder et al. 1988) and was supported by the findings of a retrospective study of 229 cases of colic, where pelleted grain had been fed to a higher proportion of horses with large colon displacement and torsions (23.1%) than with small intestine strangulated obstruction (4.5%) (Morris et al. 1989).

It is not clear if reduced ingesta hydration produced by grain intake can be explained by the theory proposed by Clarke et al. (Clarke et al. 1990a) that feeding large meals produces post-prandial dehydration, activation of the renin-angiotensin-aldosterone system and increased water absorption from colonic content. Cyclic post-prandial activation of the renin-angiotensin-aldosterone system as seen in ponies fed a single daily meal of 1 h duration, would probably produce cyclic changes in ingesta hydration (Clarke et al. 1988). In this study, indeed ingesta hydration did not change with time when the horses were adapted to the grain meals. The reduced intake of fiber when grain was fed may have contributed to reduce ingesta hydration since fiber holds water within the gastrointestinal tract (Warren et al. 1999). However it is possible that other mechanisms were also involved. Further studies are necessary to elucidate the effect of diet on gastrointestinal secretion and water transport by the intestinal mucosa.

The assumption that reduced ingesta hydration produced by two large grain meals could contribute to impaction formation may be misleading. At least in rabbits gastrointestinal flow depends on 3 factors: intraluminal pressure, luminal diameter and ingesta viscoelastic properties. Coordinated intestinal contractions generate pressure gradients to overcome the resistance to ingesta flow. Luminal diameter and ingesta viscoelastic properties are major factors determining the resistance to ingesta flow. Decreased intestinal diameter and increased ingesta viscosity increase resistance to ingesta flow up to a point where flow is interrupted (Morel et al. 1990). If these same principals are valid for the horse, it is likely that not ingesta hydration but rather ingesta viscosity is the main variable for impaction formation. Although ingesta dehydration observed when the horses were fed grain should be expected to increase viscosity (McRorie et al. 1998), the increase in the gas content and the decrease in the fiber content of ingesta produced by this feeding regimen would contribute to decrease viscosity (Srivastava and Srivastava 1989). Although viscosity of the liquid phase of ingesta appeared to be increased when the horses were fed grain, the overall ingesta viscosity did not appear to be changed. Unfortunately neither the overall viscosity of ingesta nor viscosity of the liquid phase of ingesta was measured to confirm these observations. The spontaneous flow of ingesta through the 5 cm internal diameter cannula also suggests that ingesta viscosity in horses eating grain does not compromise ingesta flow. Furthermore, in ruminants, a species where significantly more information about the effects of large grain meals is available, formation of frothy ingesta is not associated with intraluminal obstruction. Indeed ruminal distention in animals with frothy bloat is caused by compromised rumen motility due to low ruminal pH and inhibited eructation (Cheng et al. 1998).

The right shift of the cobalt concentration curves and the increase in mean retention time of cobalt indicate a slower transit of the liquid phase when grain was fed because cobalt is a marker of the liquid phase of ingesta (Uden et al. 1980). It is possible that grain ingestion had resulted in an overall delay in gastrointestinal transit, which has been previously suggested (Yoder et al. 1997, Pagan et al. 1998, Drogoul et al. 2001). At least in other species the presence of large amounts of hydrolyzable carbohydrates in the diet is a stimulus for the release of

hormones such as cholecystokinin and amylin, which inhibit gastric emptying (Reidelberger et al. 2001). Furthermore, the sharp separation between the liquid and the solid phases of ingesta seen when the horses were on the hay diet may have contributed to the difference in cobalt transit. The liquid phase could have passed separately and more rapidly than the solid phase when only hay was fed, while the liquid phase passed slower and simultaneously with the solid phase when grain was fed. This hypothesis is in agreement with previous studies where markers of the liquid phase passed more rapidly than markers of the solid phase in horses fed hay (Argenzio et al. 1974, Uden et al. 1982, Dugan et al. 1993).

It can be concluded that two large grain meals can reduce ingesta hydration and lead to formation of frothy ingesta. This study provided further evidence that excessive production of gas may be important in the pathogenesis of colic in horses eating large amounts of grain, although other mechanisms triggered by grain ingestion may also be involved. To better understand the etiology of colic, other aspects of the response of the equine gastrointestinal tract to large meals rich in hydrolyzable carbohydrates need to be investigated.

Manufacturers' addresses

^a Rotomin, Roto Salt, Co., Penn Yan, New York.

^b Reliance 10, Cooperative Milling, Gettysburg, Pennsylvania.

^c 18 French Equine Enteral Feeding Tube, Mila International, Inc., Florence, Kentucky.

^d Thermometer / Humidity Metter, Springfield Precision Instruments, Wood-Ridge, New Jersey.

^e Hand-Held Refractometer, Westover Scientific, Woodenville, Washington.

^f Olympus AU400, Olympus America, Inc., Melville, New York.

^g Rapidlab 348, Bayer, Co., East Walpole, Massachusetts.

^h Precision Mechanical Convection Oven, Precision Scientific, Chicago, Illinois.

ⁱ SpectrAA 220FS, Varian Inc., Walnut Creek, California.

^j SAS System – 8e, SAS Institute, Inc., Cary, North Carolina.

Table 4.1 – Composition of orchard grass hay and grain (Reliance 10 - Cooperative Milling, 1892 York Rd., Gettysburg, Pennsylvania 17325, USA.).

Components	Hay	Grain
% Dry matter	92.10	87.70
% Crude protein	11.20	11.40
Soluble protein (% Crude protein)	30.00	27.00
% Acid detergent fiber	37.10	11.70
% Neutral detergent fiber	61.10	22.30
% Lignin	5.40	1.40
% Non-fiber carbohydrate	13.40	45.60
% Non-structural carbohydrate	8.00	38.40
% Starch	1.20	30.60
% Sugar	6.80	7.80
% Crude fat	3.20	3.70
% Ash	7.56	6.97
% Total digestible nutrients	42.00	66.00
Digestible energy (MJ/kg)	7.75	12.27
Digestible energy (MCal/kg)	1.85	2.93
% Calcium	0.54	1.40
% Phosphorus	0.36	0.63
% Magnesium	0.25	0.38
% Potassium	2.14	0.85
% Sodium	0.004	0.368

* Analysis performed at DHI Forage Testing Laboratory, 730 Warren Rd, Ithaca, NY 14850

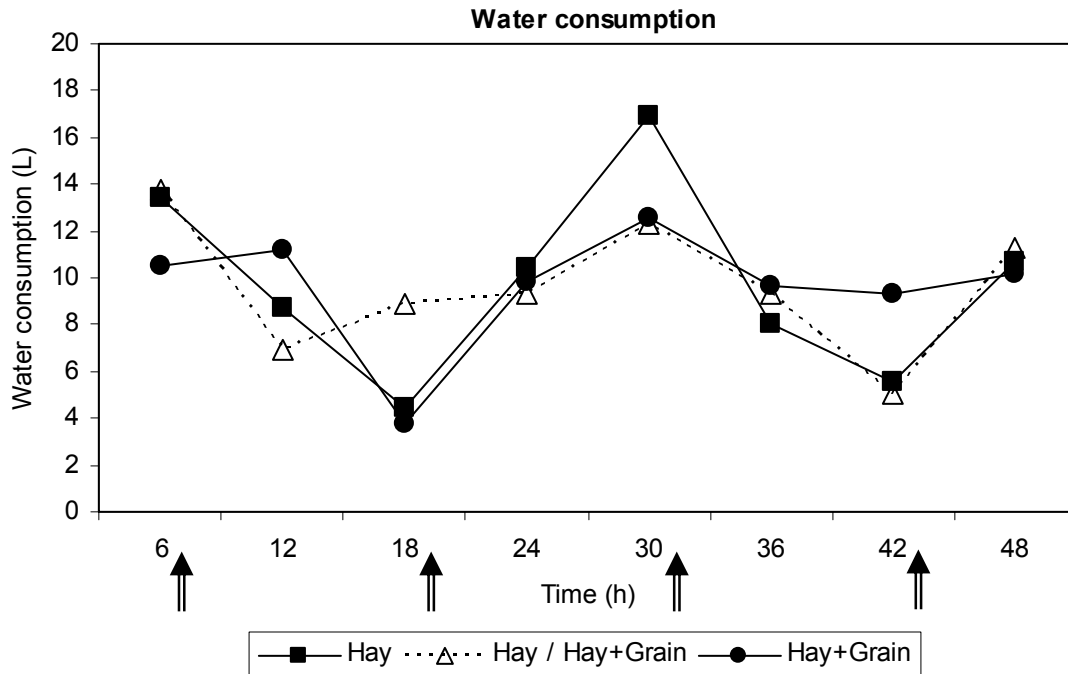


Figure 4.1 – Mean water intake of 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. There was an effect of time ($P < 0.01$), but no effect of diet ($P = 0.97$) or diet by time interaction ($P = 0.25$).

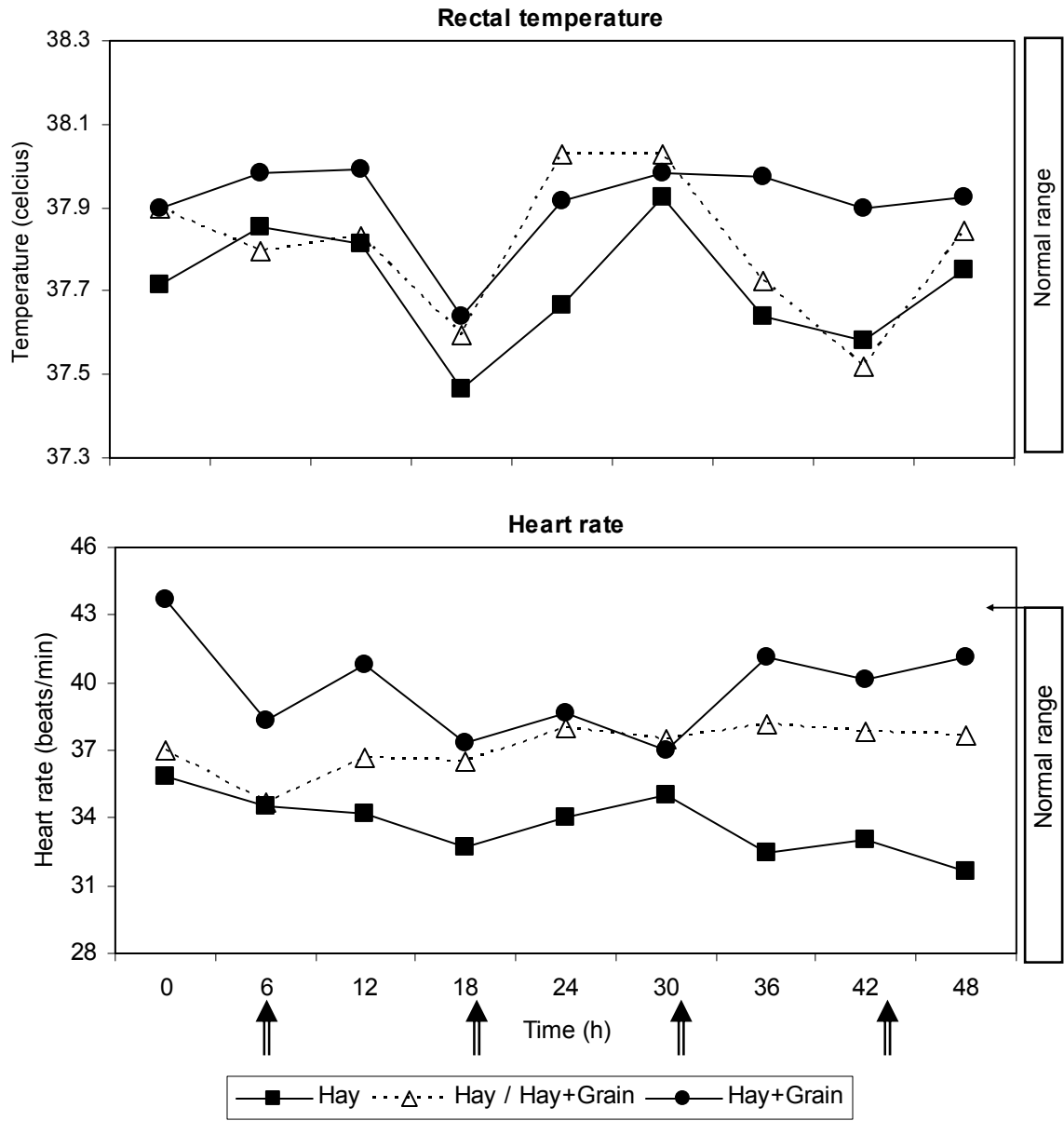


Figure 4.2 – Mean rectal temperature and heart rate in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Rectal temperature was affected by time ($P < 0.01$) and diet ($P = 0.01$), but there was no diet by time interaction ($P = 0.67$). Heart rate was affected by diet ($P < 0.01$), but not by time ($P = 0.11$) and there was no diet by time interaction ($P = 0.09$). For both variables means of horses receiving just hay was significantly lower than when the horses were adapted to the hay and grain diet ($P < 0.01$) according to the Bonferroni corrected multiple comparisons. There was no other difference between diets at $\alpha = 0.05$.

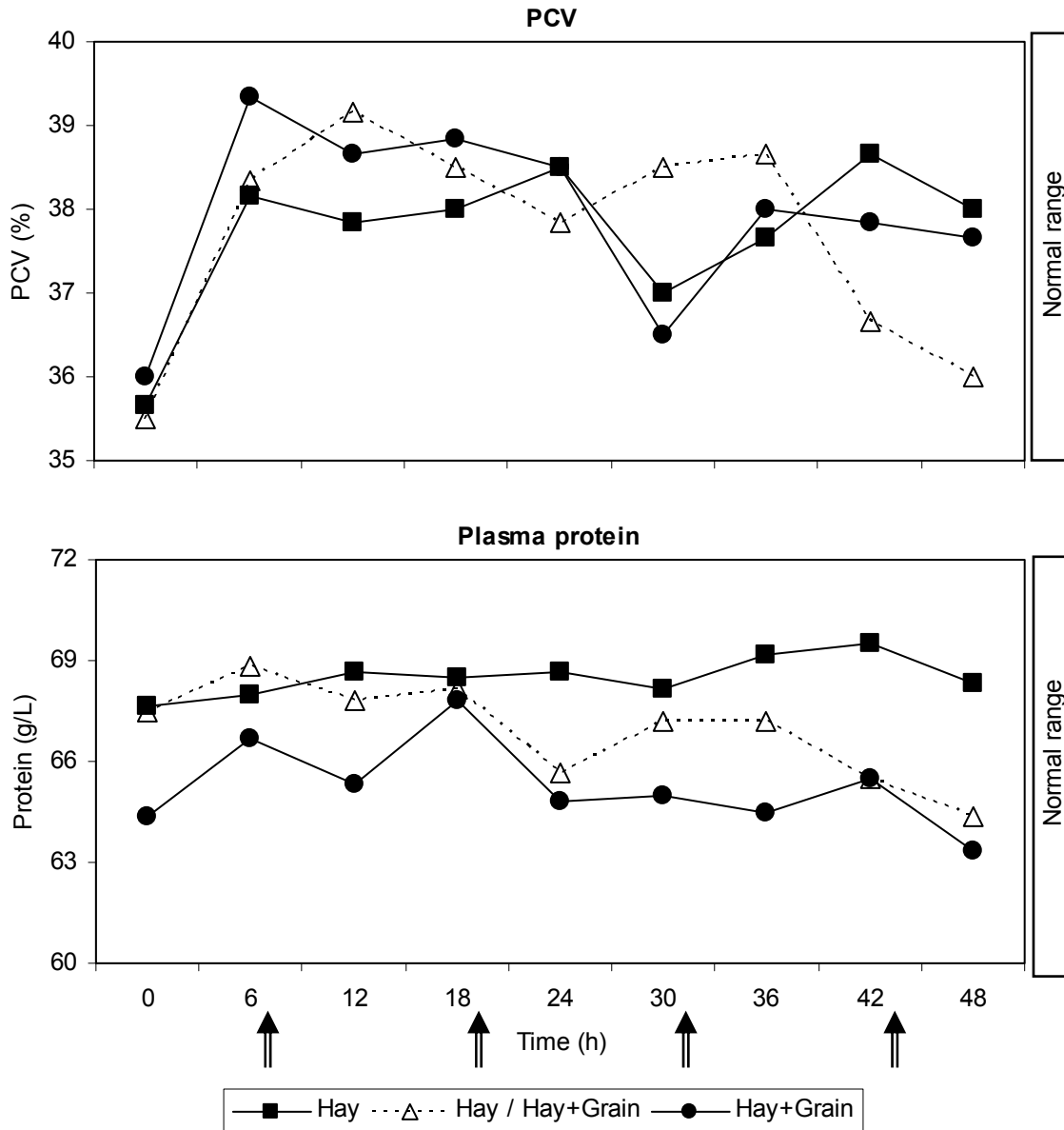


Figure 4.3 – Mean PCV and plasma protein in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. For both variables there was an effect of time ($P < 0.01$), but no effect of diet ($P = 0.89$ and $P = 0.19$) or diet by time interaction ($P = 0.82$ and $P = 0.22$).

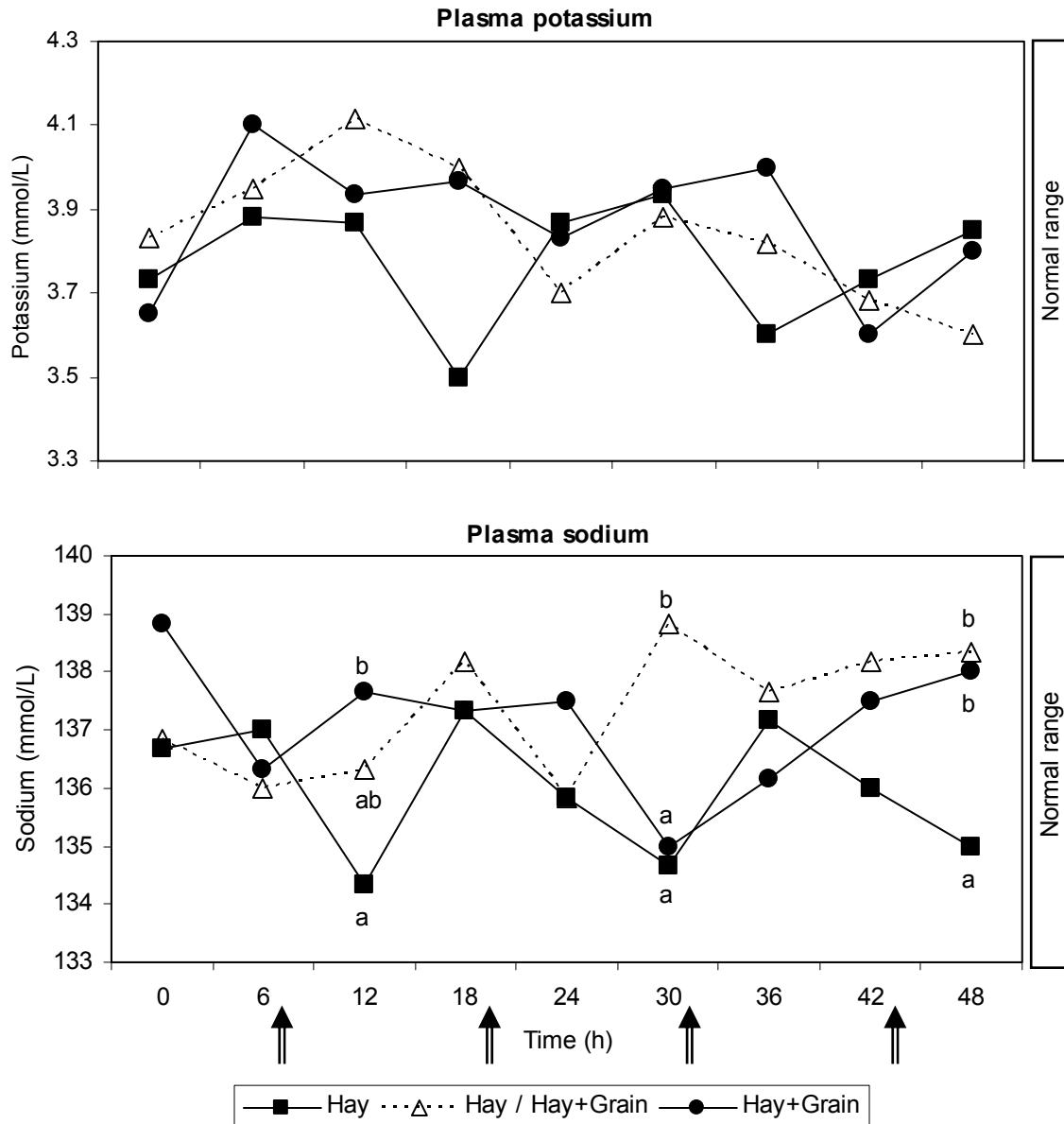


Figure 4.4 – Mean plasma potassium and sodium concentration in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Plasma potassium was affected by time ($P=0.04$), but not by diet ($P=0.67$) and there was no diet by time interaction ($P=0.12$). Plasma sodium was affected by diet ($P=0.04$) and there was diet by time interaction ($P=0.01$), but sodium was not affected by time ($P=0.19$). Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

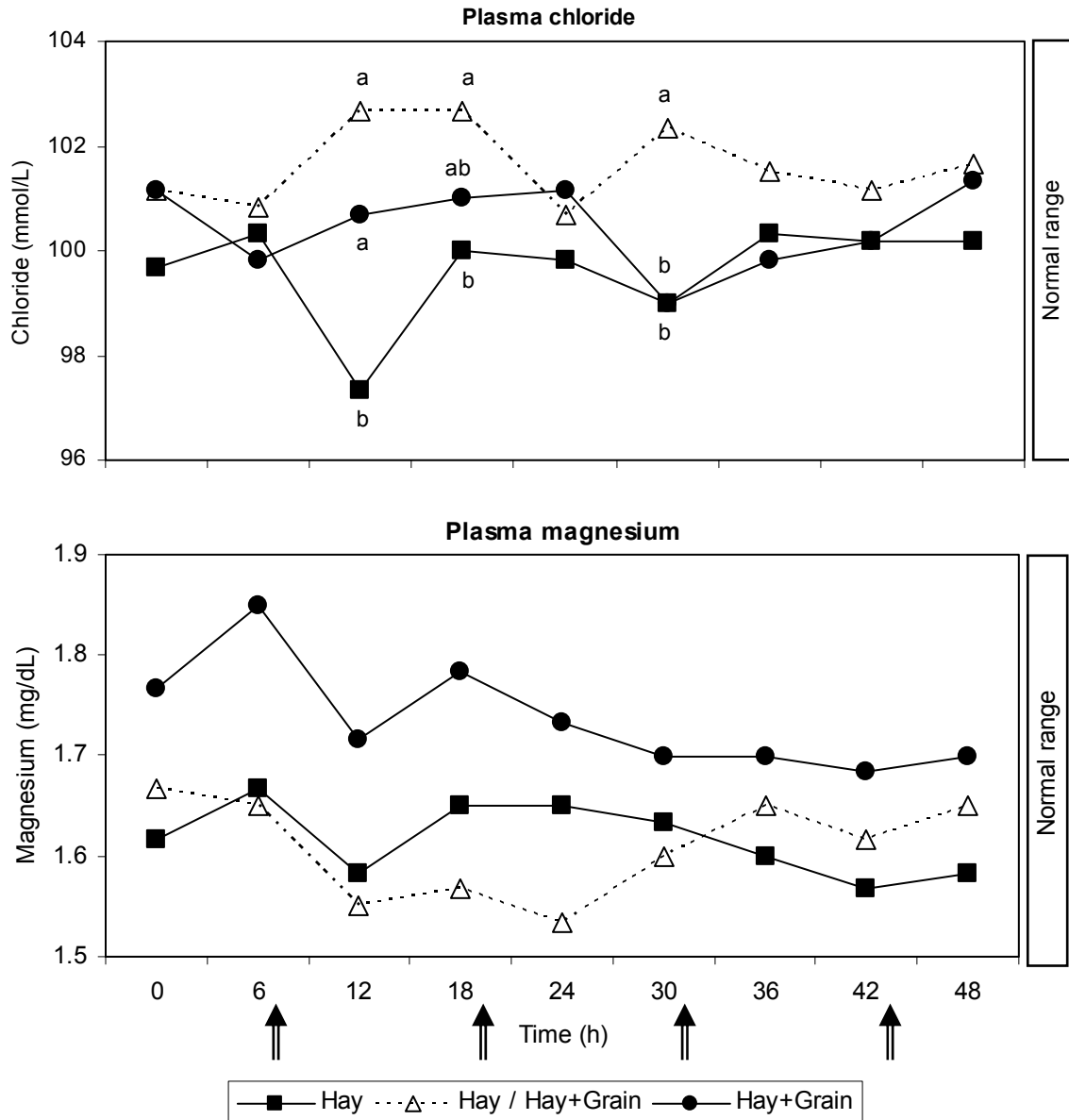


Figure 4.5 – Mean plasma chloride and magnesium concentration in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Plasma chloride was not affected by diet ($P < 0.01$) and there was diet by time interaction ($P = 0.03$), but chloride was not affected by time ($P = 0.55$). Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons. Plasma magnesium was affected by time ($P < 0.01$) and diet ($P < 0.01$), but there was no diet by time interaction ($P = 0.61$). Mean magnesium concentration of horses adapted to the hay + grain diet was significantly higher than when the horses were in the hay regimen ($P = 0.01$) or in the hay / hay + grain regimen ($P < 0.01$) according to the Bonferroni corrected multiple comparisons. There was no other difference between diets at $\alpha = 0.05$.

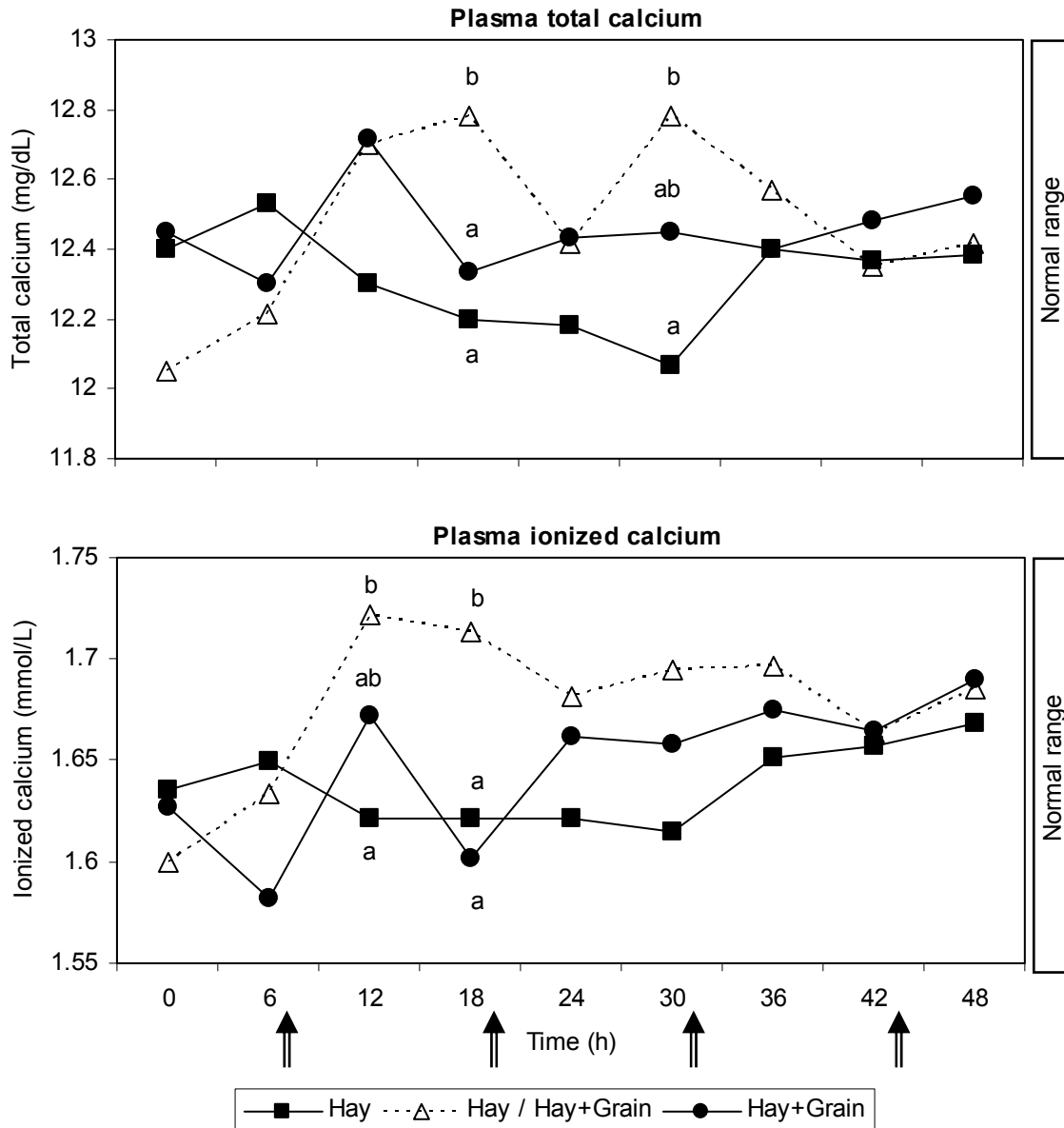


Figure 4.6 – Mean plasma total calcium and ionized calcium concentration in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Total calcium was not affected by time ($P=0.43$) or diet ($P=0.13$), but there was diet by time interaction ($P=0.02$). Ionized calcium was affected by time ($P<0.01$), but not by diet ($P=0.24$), and there was diet by time interaction ($P<0.01$). Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

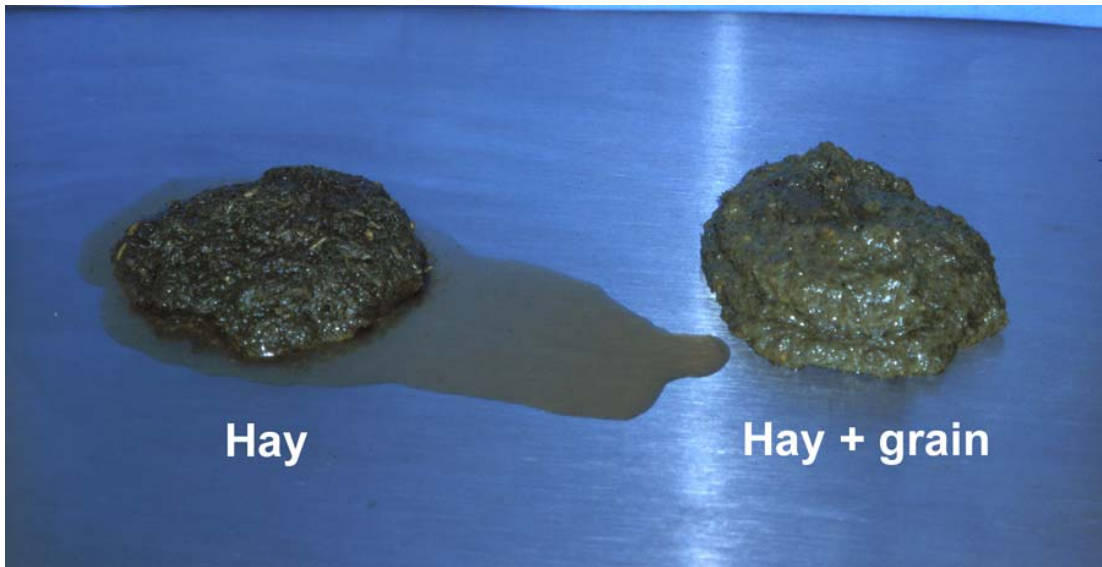


Figure 4.7 – Ingesta from horses in two feeding regimens: when just hay was fed, a clear separation between the solid and liquid phases could be seen, which was not seen when hay+grain was fed.

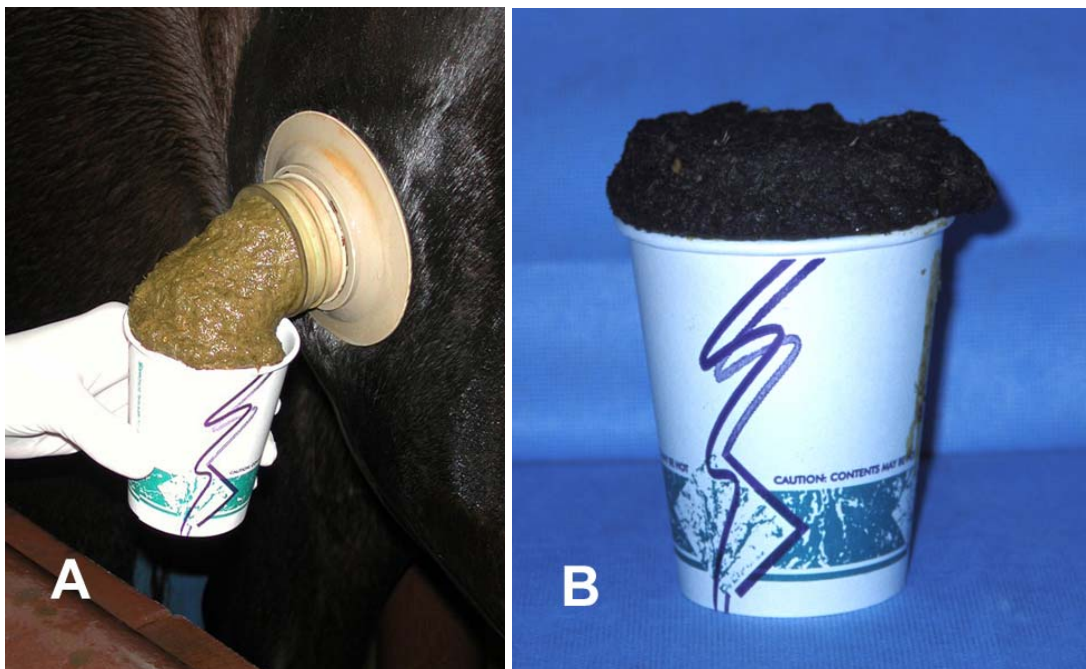


Figure 4.8 – Foamy ingesta from a horse fed hay+grain: A- Spontaneous flow of foamy ingesta through the open cannula; B- Expansion of ingesta produced by heating in the oven.

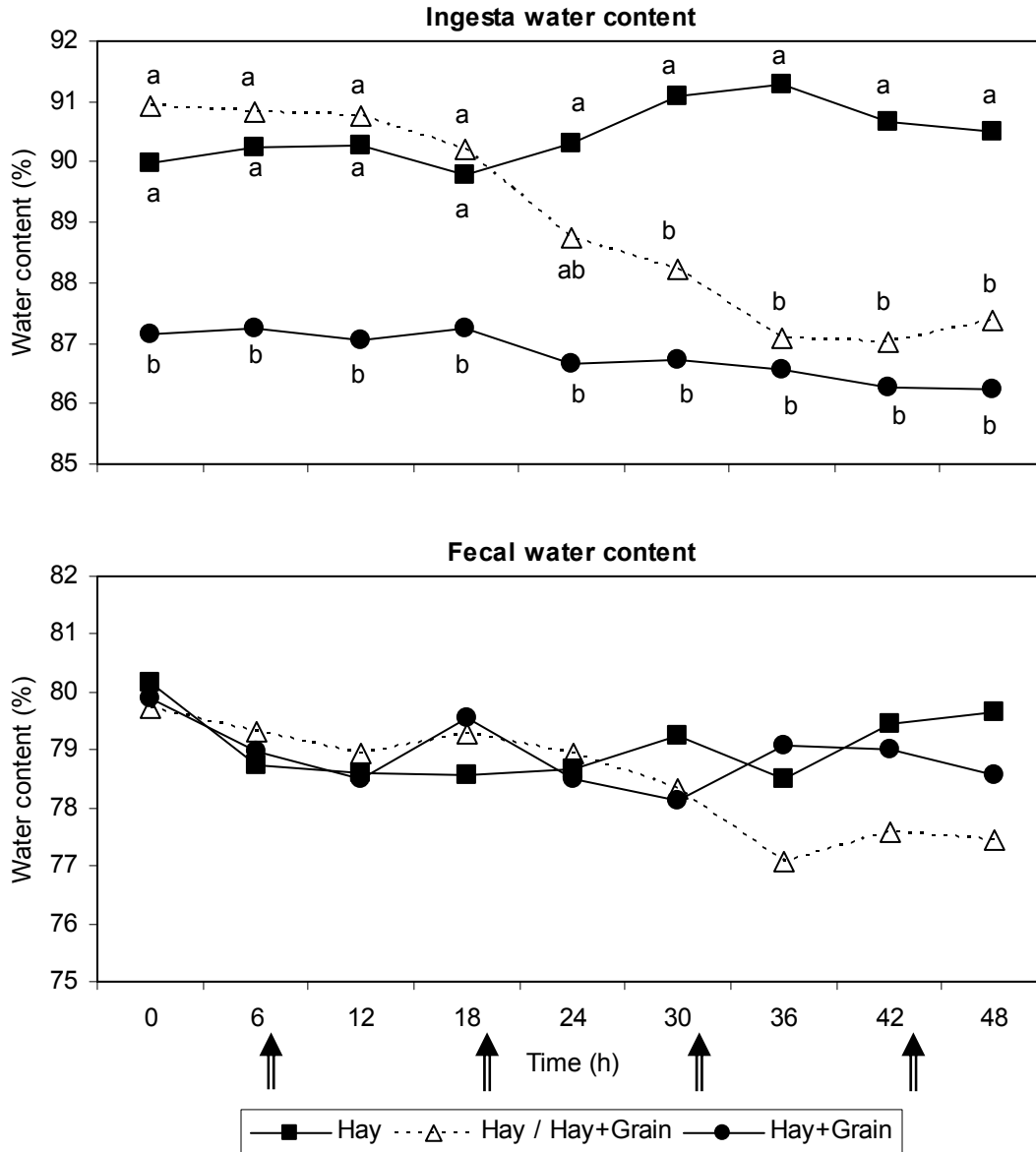


Figure 4.9 – Mean ingesta water content and fecal water content in 6 fistulated horses under 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Ingesta water content was affected by time ($P < 0.01$) and diet ($P < 0.01$) and there was diet by time interaction ($P < 0.01$). Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons. Fecal water content was affected by time ($P < 0.01$), but not by diet ($P = 0.66$) and there was no diet by time interaction ($P = 0.09$).

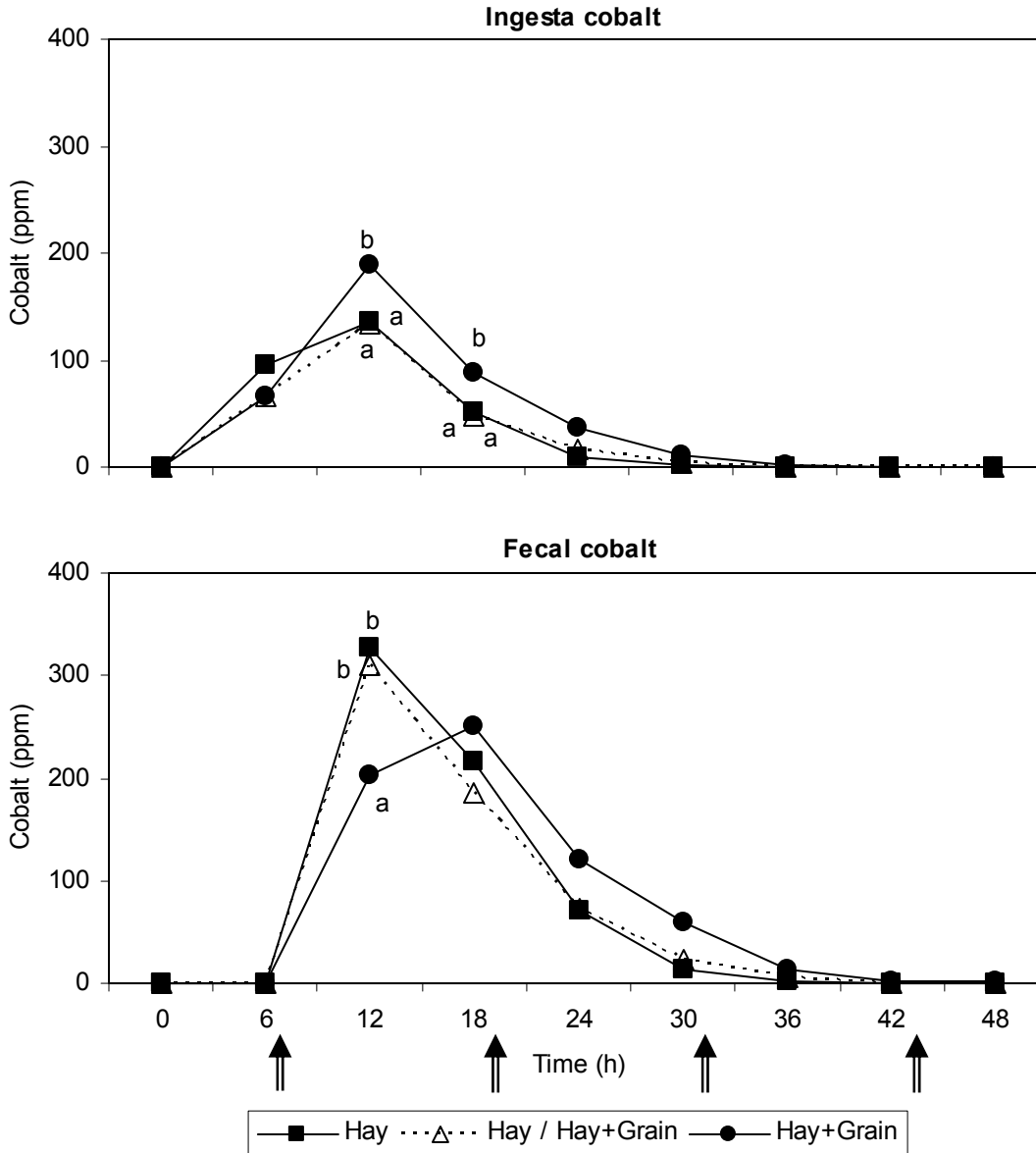


Figure 4.10 – Mean ingesta cobalt concentration and fecal cobalt concentration in 6 fistulated horses treated with 40 mg/kg of CoEDTA via nasogastric tube and submitted to 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. The arrows indicate the times when grain was fed. Both variables were affected by time ($P < 0.01$), but not by diet ($P = 0.09$ and $P = 0.86$), and there was diet by time interaction ($P = 0.02$ and $P = 0.01$). Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

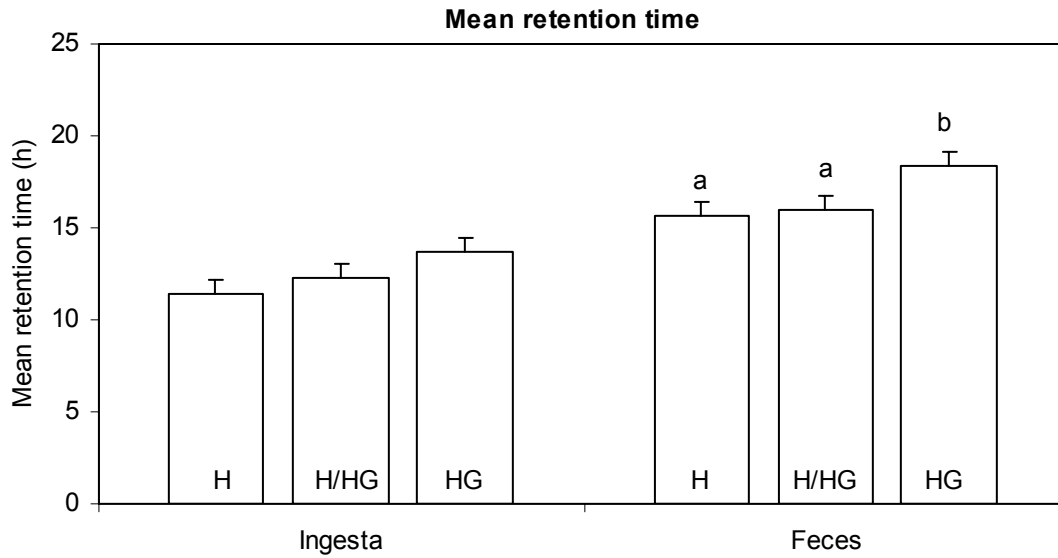


Figure 4.11 – Mean retention time of cobalt in ingesta and feces in 6 fistulated horses treated with 40 mg/kg of CoEDTA via nasogastric tube and submitted to 3 feeding regimens: fed free choice hay; fed hay and 4.55 kg of grain every 12 h, but adapted to free choice hay; fed hay and 4.55 kg of grain every 12 h after at least 5 days on this diet. Vertical bars represent the standard errors of the means. There was an effect of the feeding regimen on the mean retention time of cobalt in feces ($P=0.03$) but not ingesta ($P=0.18$). Different letters on top of the columns indicate significant difference at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

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Chapter 5

Enteral administration of large volumes of 0.9% NaCl to normal horses

Abstract

Objectives – To assess systemic and gastrointestinal effects of large volumes of 0.9% NaCl administered slowly (10 L in 40 to 50 min) through a small bore nasogastric tube or as large boluses (10 L in three to four min) through a large bore nasogastric tube.

Animals – 4 horses with no signs of gastrointestinal disease.

Methods – The total fluid volume was divided in eight equal doses, which were administered every hour for 8 h. Two horses were randomly selected to receive the small-bore tube (slow administration) while the other two horses received the large bore-tube (boluses administration). One week later all procedures were repeated, but the horses that had been treated with the small-bore tube received the large-bore tube and vice versa.

Results – No effect of the rate of fluid administration was observed. Both slow infusion and boluses were well tolerated and produced moderate abdominal distension, increased fecal hydration, decreased PCV, hypernatremia, hyperchloremia, pollakiuria and hyposthenuria. Fifteen hours after starting the last fluid dose all variables had returned to pre-treatment values except plasma chloride.

Conclusions – Enteral fluid therapy administered slowly or as boluses can be used to increase fecal hydration, plasma volume and urine production. However to prevent hypernatremia and hyperchloremia an electrolyte solution other than 0.9% NaCl should be administered.

Introduction

Enteral fluid therapy is a simple and inexpensive option for fluid administration. However enteral fluid therapy is not commonly used in horses and only a few experimental studies have been published on this topic. Enteral fluid therapy has been reported to be an effective treatment for large colon impaction by increasing the hydration content of ingesta (Lopes et al. 1999). In horses, fluids administered into the stomach are rapidly emptied to the intestine (Sosa Leon et al. 1995) and can reach the large intestine in less than one hour (Alexander and Benzie 1951, Argenzio et al. 1974). Additionally, nasogastric administration of fluids may stimulate colonic motility through the gastrocolic reflex, which might contribute to relieve large intestine impaction (Freeman et al. 1992, Lopes et al. 1999).

In the few controlled studies involving the administration of enteral fluid therapy to horses, relatively small fluid volumes (4 to 30 L/day) were used (Rose et al. 1986, Rapp 1988, Freeman et al. 1992, Jansson et al. 1995, Sosa Leon et al. 1995, Hyyppa et al. 1996, Sosa Leon et al. 1997, Ecke et al. 1998, Marlin et al. 1998b, Marlin et al. 1998a, Monreal et al. 1999). However significantly larger volumes may have to be used to treat horses with conditions such as dehydration (Corley 2001) or large intestine impaction (Dabareiner and White 1995, Lopes et al. 1999), and no controlled study investigating if such large volumes of fluids can be tolerated by the equine gastrointestinal tract has been reported. Information on the appropriate fluid composition for enteral fluid therapy is also lacking. In the only report of the administration of

large volumes of fluids by nasogastric tube (>30 L/day), water was used (Lopes et al. 1999). However, water can produce hyponatremia and it therefore is advisable to use an electrolyte solution for enteral fluid therapy (Monreal et al. 1999).

Studies in humans have shown that the rate of gastric emptying critically affects the efficiency of enteral fluid therapy, and factors such as the rate of fluid administration and fluid composition can affect gastric emptying (Gisolfi and Duchman 1992). The rate of fluid emptying from the stomach is proportional to gastric distension. Thus gastric emptying can be maximized by maintaining the maximum gastric distension tolerable (Noakes et al. 1991, Mitchell et al. 1994). Gastric emptying is not affected by the concentration of any specific nutrient, however faster gastric emptying is seen when using fluids of lower osmolality (Noakes et al. 1991, Gisolfi and Duchman 1992). These same factors are likely to play a role in gastric emptying in horses. However no study has been published investigating the maximum fluid volume that can be safely administered through a nasogastric tube in horses or if the rate of fluid infusion affects tolerance to enteral fluid therapy.

In the clinical setting, fluids are usually administered as boluses by a large-bore nasogastric tube designed for gastric lavage and laxative administration for horses with colic (Lopes et al. 1999). This large-bore tube causes discomfort for the horse, and may cause damage to the nasal passages, pharynx, larynx and esophagus (Hardy et al. 1992). Alternatively a tube with a smaller gauge may have the advantage of causing less discomfort for the horse and less pressure to the upper airways and esophagus. Furthermore, with a small-bore tube, fluid delivery will be slower, which may reduce the risk of excessive gastric distension and pain. However a slower rate of administration may reduce the effects of fluids on fecal hydration by limiting gastric emptying and the gastrocolic response, and by allowing more time for fluid absorption.

This experiment was conducted to evaluate the effects of the administration of large volumes of an isotonic electrolyte solution (0.9% sodium chloride) by nasogastric tube to normal horses. The effects of two rate of infusion (as 10 L boluses through a large-bore nasogastric tube or slowly through a small-bore nasogastric tube) on fecal hydration, body weight, PCV, plasma protein and plasma electrolytes were compared. We hypothesized that enteral fluid therapy would be well tolerated and would produce significant increase in fecal hydration. We also hypothesized that the rate of fluid administration would affect the response to treatment: boluses administration would produce more significant effects on fecal hydration, while slow administration would produce more systemic effects.

Material and methods

This study was approved by the Virginia-Tech Animal Care Committee. Four healthy adult horses were used: An eight year old Thoroughbred gelding, a 17 year old Thoroughbred mare, an 18 year old Quarter Horse gelding, and an 11 year old Quarter Horse cross gelding. Body weight immediately before fluid administration ranged from 477 to 600 kg (mean, 533 kg). The horses were maintained on pasture until one day before the experimental periods, when they were moved to a pen with free access to hay, water and a salt block. Feed, but not water, was withheld for 9 h before fluid therapy was started.

The fluids used for enteral fluid therapy were prepared with tap water and 9 g of sodium chloride (Mix-N-Fine Salt, Cargill, Inc.) per liter (0.9% NaCl). The calculated concentrations of ions were 154 mmol/L of Na⁺ and 154 mmol/L of Cl⁻, and the calculated osmolality was 308

mOsmol/L. The calculated electrolyte load provided by enteral fluid therapy was 12320 mmol of both sodium and chloride. The osmolality of two samples (one of each of two batches) of the solution was evaluated by freezing point osmometry (Osmette A, Precision Systems, Inc.).

Experimental Period I - Two horses were randomly assigned to receive a 16 mm external diameter nasogastric tube (internal diameter, 9.5 mm) (Medium PVC Stomach Tube, Jorgensen Laboratories, Inc.), while the other two horses received a 6 mm external diameter enteral feeding tube (internal diameter, 4 mm) (18 French Equine Enteral Feeding Tube, Ross Laboratories). The lengths of the tubes were respectively 274 and 260 cm. Immediately before passing the nasogastric tube, blood samples were collected from the jugular vein with evacuated tubes containing lithium heparin (Vacutainer, Becton Dickinson), fecal samples were collected from the rectum, and a clinical examination was performed. During the period the tubes were kept in place the horses did not have access to water or feed and were kept muzzled to prevent them from displacing the tube by rubbing it on the wall.

Fluid therapy consisted of 80 L of the 0.9% NaCl solution divided into aliquots of 10 L administered hourly for 8 h. To the horses with the large-bore nasogastric tube, fluids were administered through a funnel positioned about 20 cm above the horses' withers. To the horses with the small-bore enteral feeding tube, a fluid line was used to deliver fluid from two plastic bags positioned about 80 cm above the horses' withers. The infusion rate was determined by gravity and tube gauge.

Environmental temperature and humidity measured by a thermometer and humidity meter (Thermometer / Humidity Meter Model 90116, Springfield Precision Instruments) at the beginning, middle and end of fluid therapy were recorded. The time required to deliver each dose of fluid was recorded and the horses were continuously monitored during fluid therapy. Urine samples were occasionally collected during spontaneous urination for specific gravity determination. One hour after starting the last dose of fluids, clinical examination and measurement of body weight were repeated, as well as blood and fecal sampling. The tube was then removed and the horses were returned to a pen with free access to hay and water. The next morning (15 h after starting the last 10 L dose), clinical examination was repeated, the horses were again weighed and blood and fecal samples were collected.

PCV was measured using the microhematocrit technique and plasma protein was measured with a refractometer (Hand-Held Veterinary Refractometer, Westover Scientific). Within 10 min after collection, blood was centrifuged at 1124 xg for 10 min. Plasma was stored at -70° C until analysis for Na⁺, K⁺ and Cl⁻ concentration, which was performed with a flame spectrophotometer (Abbott Spectrum Series II System, Abbott Laboratories). Feces were weighed (Harvard Trip Balance, Ohaus), dried in an incubator (Precision Mechanical Convection Oven, Precision Scientific) at 90-100° C and weighed again until there was no longer a change in dry weight. Fecal hydration was calculated by dividing the difference between wet weight and dry weight by the wet weight. Urine specific gravity was measured with a refractometer (Hand-Held Veterinary Refractometer, Westover Scientific).

Experimental Period II - One week after the first period all procedures were repeated, but the horses that had been treated with the small-bore nasogastric tube received the large-bore nasogastric tube and vice versa. In addition to the other variables evaluated in the first period, the number of defecations and urinations were recorded, abdominal circumference was measured, and fecal consistency was estimated. The following scoring system was used to estimate fecal

consistency: 5 – normal (formed fecal balls); 4 – soft (reduced consistency, but partially formed balls can still be seen); 3 – cow-pie (fecal balls are not seen, but feces form a pile); 2 – cow-pie / watery (intermediate between cow-pie and watery, only a flat pile is formed); 1 – watery (minimal amount of solid material, no pile is formed). Abdominal circumference at the end of expiration was measured at the site with the apparent largest circumference with a graduated tape. This site was identified by clipping the hair in three different places (dorsal midline and both sides of the thorax). Abdominal circumference was measured at four times: immediately before starting fluid therapy, immediately before starting the fifth dose of fluids, one hour after starting the last dose of fluids, and fifteen hours after starting the last dose of fluids.

Statistical analysis - The MIXED procedure of the SAS System – 8e (SAS Institute, Inc.) was used to perform a mixed model repeated measures analysis of variance to test for effects of treatment, time and treatment by time interaction while controlling for horse and period effects. For significant time effects means were separated using Tukey's HSD. No statistical analysis of the effect of treatments on abdominal circumference was performed because data were not collected in the first period.

Results

The measured osmolalities of the 0.9% NaCl solutions were 285 and 286.3 mOsm/l (mean, 285.6). The temperature and relative humidity recorded during the first period ranged from 24 to 38° C and 55 to 70% respectively. The temperature and relative humidity recorded during the second period ranged from 17 to 25° C and 40 to 70% respectively.

Each 10 L dose of fluids could be administered in 3 to 4 min when the large bore tube was used, while administration took 40 to 50 min when the small-bore tube was used. During the period the tube was kept in place all horses presented signs of nasal irritation such as head shaking and sneezing, and rubbed the muzzle on the wall. This behaviour seemed to be more pronounced with the large-bore tube although the frequency and intensity of the signs of nasal irritation were not objectively quantified. Considering the body weights immediately before fluid administration and the fluid volume administered per hour (10 L), the rate of infusion ranged from 16.7 to 21.0 mL/kg/h (mean, 18.9 mL/kg/h). The electrolyte load provided by enteral fluid therapy ranged from 20.5 to 25.8 mmol/kg (mean, 23.1 mmol/kg) of both sodium and chloride.

The response to fluid therapy was not different when the two rates of administration were compared. For all variables analyzed there was no effect of treatment or treatment by time interaction (Table 5.1), but for most variables there were significant changes with time (Table 5.2). All horses had soft feces, pollakiuria (Figure 5.1), hyposthenuria (urine specific gravity ranging from 1.004 to 1.010 g/mL) and moderate abdominal distension during fluid therapy (Table 5.2), but in no case was any sign of abdominal discomfort observed. At the end of fluid therapy body weight, abdominal circumference, fecal hydration and plasma sodium and chloride concentrations were significantly increased, and PCV was significantly decreased (Table 5.2). No significant changes in plasma protein and plasma potassium were seen (Table 5.2). Fifteen hours after starting the last dose of fluids, all values were not different from what had been observed before fluid therapy except for plasma chloride, which was still high, and plasma potassium, which was significantly higher than before fluid therapy (Table 5.2). However, in all horses plasma potassium concentration was within the normal range.

Discussion

This study showed that large volumes of fluids can be safely administered to horses by nasogastric tube, as previously reported (Lopes et al. 1999). Although large volumes of fluids were administered in a short period of time, no sign of abdominal discomfort was seen, which implies that gastric emptying occurred fast enough to prevent severe stomach distension. A previous study had shown that, in horses, a single dose of fluids administered by nasogastric tube is rapidly emptied from the stomach (Sosa Leon et al. 1995). However tolerance to repeated boluses or slow infusion of large fluid volumes had not been documented. The increased body weight and abdominal circumference at the end of fluid therapy indicated that a significant portion of the fluids had not been eliminated as urine or feces at that time, and that most likely a large portion of the fluids was in the gastrointestinal tract. In accordance with previous observations (Lopes et al. 1999), abdominal distension produced by enteral fluid therapy did not cause any problem. However, in horses presenting marked abdominal distension due to conditions such as an extremely large impaction or advanced pregnancy, further distension due to fluid therapy may not be tolerated.

The changes in fecal consistency and hydration produced by enteral fluid therapy are in agreement with previous observations that this treatment can be used for large intestine impaction (Lopes et al. 1999). It has been suggested that the gastrocolic response may play a role in the changes in fecal hydration produced by enteral fluid therapy (Freeman et al. 1992). If this hypothesis is true, both repeated administration of boluses and slow infusion must have similarly affected the gastrocolic response, since no difference could be seen in fecal hydration. Since similar effects could be achieved when small and large-bore tubes were used, the small-bore tube seems more appropriate for fluid therapy because it produces less discomfort for the horse. The fact that a horse with a small-bore nasogastric tube can be allowed to eat (Murray and Schusser 1993) may be another advantage of using this type of tube, while the large-bore tube is more likely to interfere with deglutition.

The fall in PCV, pollakiuria and hyposthenuria were signs that an overhydration status was produced. These findings agree with previous observations in horses that the gastrointestinal tract is able to absorb a major part of the fluids administered by nasogastric tube (Rose et al. 1986, Sosa Leon et al. 1995, Marlin et al. 1998b, Marlin et al. 1998a). However, a significant drop in plasma protein, which would be expected with overhydration, was not seen at the end of fluid therapy. This finding can be explained by the poor specificity of refractometry to measure plasma protein concentration. It is known that a high concentration of other solutes in plasma can produce false high protein readings (Duncan et al. 1994). Thus the high concentration of sodium and chloride at the end of fluid therapy explains the high values of plasma protein when other findings (pollakiuria, diluted urine and low PCV) clearly indicated plasma expansion.

The occurrence of hypernatremia and hyperchloremia can be seen as the result of the administration of a high cumulative load of sodium and chloride. A more dramatic increase in chloride than sodium may be explained by the equimolar concentration of these ions in the fluid administered and the lower values of chloride normally present in plasma. Hyperchloremia had been previously observed when normal and dehydrated horses were treated with only 20 L of 0.9% NaCl intravenously (Carlson and Rumbaugh 1983). The more pronounced rise in the concentration of the strong anion chloride in plasma suggests that acidosis would have been found if blood gas analyses had been performed (Whitehair et al. 1995). A solution containing less sodium and less chloride obtained by replacing some of the sodium chloride with substances

such as potassium chloride, sodium bicarbonate and sodium citrate is indicated. Even the administration of a potassium free solution, did not produce hypokalemia, but higher potassium concentrations were seen at the end of fluid therapy. Potassium shift from the intracellular space caused by acidosis may explain these findings (George 1994). The short period of food deprivation, and the absorption of potassium from the remaining intestinal content further explain the absence of hypokalemia. For a longer fasting period or for conditions leading to high potassium loss, the use of a solution containing potassium may be necessary to prevent hypokalemia. The difference between measured and calculated osmolality of the 0.9% NaCl solution was expected, since the calculation ignores interionic or interparticulate attractions (Murty et al. 1976).

This study demonstrated that horses can tolerate large volumes of fluids administered by the enteral route either as slow infusion through a small-bore tube or as 10 L boluses through a large-bore tube. This study also demonstrated that enteral fluid therapy can be used in horses to increase fecal hydration, plasma volume and urine production. The small bore tube may be advantageous since it produces less nasal irritation and the horse can be allowed to eat while receiving enteral fluid therapy. Since little emphasis has been given to enteral fluid therapy in equine medicine, important basic information is still lacking. At least for horses with normal plasma electrolytes, it was clearly demonstrated that large volumes of 0.9% sodium chloride should not be used for enteral fluid therapy. Rational judgment of clinical condition based on clinical and laboratory findings is mandatory in order to guide enteral fluid therapy and to adjust fluid composition and rate of administration according to the horse's needs.

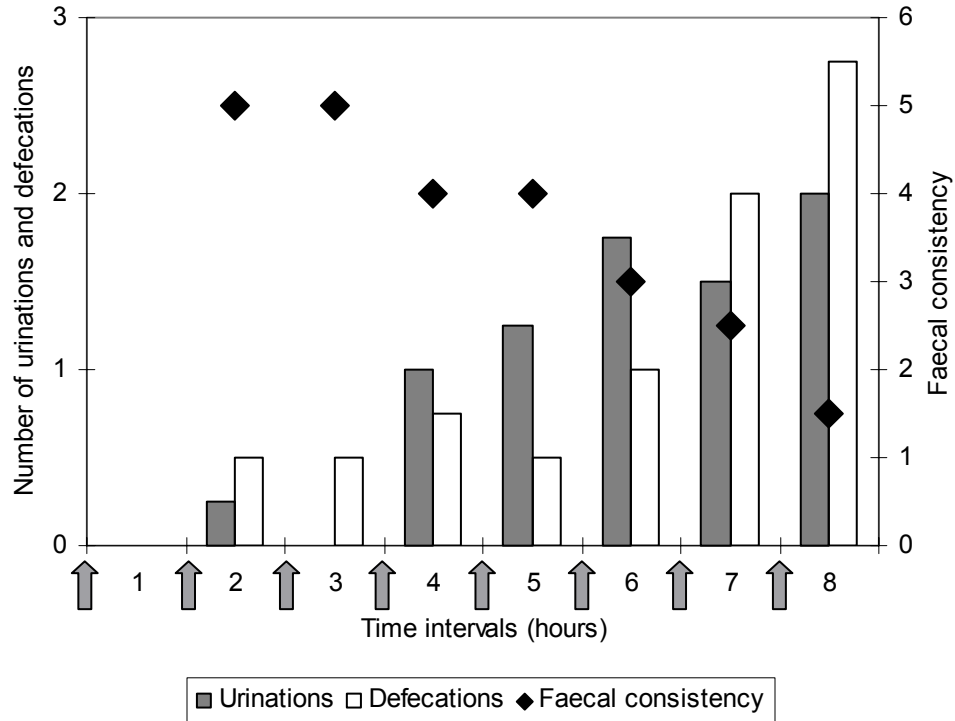


Figure 5.1 – Mean number of defecations and urinations and median of faecal consistency rating (6=dry, 5=normal, 4=normal/cow pie, 3=cow pie, 2=cow pie/watery, 1=watery) of four normal horses treated with 80 L of NaCl 0.9% divided in 10 L doses administered hourly for 8 h. In two horses, the fluid doses were administered slowly (10 L in 40 to 50 min), while in two horses, the fluid doses were administered as boluses (10 L in three to four min). The arrows indicate the moments when the administration of each 10 L dose was started.

Table 5.1 – Effects of administration of 80 L of NaCl 0.9% at a rate of 10 L / h to four horses during two periods one week apart. Fluid therapy was administered as boluses through a large-bore nasogastric tube or slowly through a small-bore nasogastric tube (means and p-values).

	Treatment	Before start (9:00)	After 40 L (13:00)	After 80 L (17:00)	Next day (7:00)	Repeated measures ANOVA	
						Effects	P-value
Body weight (kg)	Boluses	529.5	-	558.1	528.2	Treatment	0.20
	Slow infusion	534.7	-	542.3	531.2	TreatmentXtime interaction	0.11
Abdominal circumference (cm)	Boluses	203.8	214.6	218.4	201.3	Treatment	*
	Slow infusion	209.5	216.5	218.4	207.6	TreatmentXtime interaction	*
Fecal hydration (%)	Boluses	80.0	-	94.0	84.1	Treatment	0.59
	Slow infusion	83.7	-	94.3	83.2	TreatmentXtime interaction	0.22
PCV (%)	Boluses	35.7	-	31.0	34.5	Treatment	0.88
	Slow infusion	35.0	-	29.5	36.2	TreatmentXtime interaction	0.31
Plasma protein (g / L)	Boluses	65.7	-	64.2	69.0	Treatment	0.96
	Slow infusion	66.2	-	64.0	69.0	TreatmentXtime interaction	0.96
Na+ (mmol / L)	Boluses	143.5	-	148.2	143.5	Treatment	0.31
	Slow infusion	143.3	-	147.5	141.8	TreatmentXtime interaction	0.53
K+ (mmol / L)	Boluses	3.5	-	4.2	4.2	Treatment	1.00
	Slow infusion	3.9	-	3.9	4.1	TreatmentXtime interaction	0.06
Cl- (mmol / L)	Boluses	103.5	-	118.5	111.0	Treatment	0.34
	Slow infusion	104.0	-	116.8	109.8	TreatmentXtime interaction	0.38

* Not compared. Only recorded during the second period (each treatment was administered to two horses).

Table 5.2 – Effects of enteral administration of 80 L of NaCl 0.9% (10 L / h for 8 h) to four horses during two periods one week apart (mean± standard error).

	Before start (9:00)	After 40 L * (13:00)	After 80 L (17:00)	Next day (7:00)
Body weight (kg)	533.2 ± 14.8 ^a	-	551.4 ± 15.2 ^b	530.8 ± 15.1 ^a
Abdominal circumference (cm) **	206.7 ± 4.4 ^a	215.6 ± 4.6 ^b	218.4 ± 4.7 ^b	204.5 ± 3.8 ^a
Fecal hydration (%)	81.8 ± 1.3 ^a	-	94.2 ± 1.6 ^b	83.7 ± 1.2 ^a
PCV (%)	35.4 ± 1.0 ^a	-	30.2 ± 0.6 ^b	35.4 ± 1.0 ^a
Plasma protein (g / L)	66.0 ± 1.7 ^a	-	64.1 ± 1.6 ^a	69.0 ± 1.6 ^a
Na+ (mmol / L)	143.4 ± 0.3 ^a	-	147.9 ± 0.6 ^b	142.6 ± 0.9 ^a
K+ (mmol / L)	3.7 ± 0.1 ^a	-	4.1 ± 0.1 ^{ab}	4.2 ± 0.1 ^b
Cl- (mmol / L)	103.7 ± 0.7 ^a	-	117.6 ± 0.8 ^b	110.4 ± 0.7 ^c

^{a, b, c} Means for each analyte accompanied by different letters are significantly different at $\alpha = 0.05$ according to the Tukey's HSD.

* Before starting the fifth 10 L dose of fluids, only abdominal circumference was measured.

** Only recorded during the second period (each treatment was administered to two horses).

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Chapter 6

Hydration of colonic ingesta and feces in fistulated horses treated with enteral fluids, magnesium sulfate, sodium sulfate and intravenous fluids

Abstract

Objective – To assess changes in systemic hydration, plasma electrolytes, ingesta and fecal hydration and gastrointestinal transit in horses treated with enteral fluids, magnesium sulfate, sodium sulfate and intravenous fluids.

Animals – Seven horses with an experimental fistula in the right dorsal colon.

Methods – The horses were subjected to 6 experimental conditions: 1) Control (no treatment administered); 2) MgSO_4 (1 g/kg) by nasogastric tube; 3) Na_2SO_4 (1 g/kg) by nasogastric tube; 4) IV lactated Ringer's (5 liters/h for 12 h); 5) Water (5 liters/h for 12 h) through a nasogastric tube; 6) Enteral electrolyte solution (5 liters/h for 12 h) through a nasogastric tube. Cobalt EDTA (0.04 g/kg) was administered via nasogastric tube at the beginning of each experimental period. A clinical examination was performed and blood, ingesta and fecal samples were collected every 6 hours for 48 h.

Results – The enteral electrolyte solution was the most effective treatment in promoting ingesta hydration, followed by Na_2SO_4 and water. IV fluids or MgSO_4 did not affect ingesta hydration. Na_2SO_4 was the most effective treatment in promoting fecal hydration followed by MgSO_4 and enteral electrolyte solution. Water and IV fluids did not affect fecal hydration. Na_2SO_4 produced marked hypocalcemia and hypernatremia and water produced hyponatremia. Fasting delayed gastrointestinal transit.

Conclusions – Enteral administration of electrolyte solutions can be used to promote ingesta hydration and correct dehydration and electrolyte imbalances. Enteral administration of water and saline cathartics can produce severe electrolyte imbalances and have limited effect on ingesta hydration, while intravenous fluid therapy (5 L/h/12 h) does not promote ingesta hydration.

Introduction

Large colon impaction is one of the most common causes of colic (White 1990, Cohen et al. 1999, Hudson et al. 2001). However information based on controlled studies is scarce and current recommendations for treatment are mostly based on anecdotal observations and tradition. The standard treatment for large colon impaction is the administration of laxatives and analgesics. For severe cases intravenous (IV) administration of large volumes of fluids is also recommended (White and Dabareiner 1997, Dabareiner 1998, Sullins 1999). This treatment appears to be effective for most horses, although, in some cases, surgery is necessary (Dabareiner and White 1995). It has been suggested that the systemic overhydration produced by IV fluid therapy plus the increase in intraluminal osmolality produced by magnesium sulfate promotes secretion of fluids into the gastrointestinal lumen, which promote ingesta hydration and subsequent resolution of the impaction (White and Dabareiner 1997). However the effects of these treatments on water content of colonic ingesta have not been evaluated in horses.

Absorption of MgSO_4 after nasogastric administration is thought to be limited, although magnesium toxicosis after MgSO_4 administration has been reported (Henninger and Horst 1997). Kidney excretion of the Mg^{++} is thought to prevent hypermagnesemia and the recommended dose of MgSO_4 (1.0 g/kg, enterally) appears to be safe. A single dose of MgSO_4 not followed by fluid therapy is a common treatment for horses with impaction and other conditions (Dabareiner and White 1995). Alternatively another saline cathartic such as sodium sulfate can be used, which would not have the risk of producing hypermagnesemia. Furthermore, in the only reported study, a single dose of sodium sulfate (1.0 g/kg, enterally) was more effective than a single dose of magnesium sulfate (0.8 g/kg, enterally) in promoting fecal hydration in 2 fistulated horses (Meyer et al. 1986).

Enteral fluid therapy has been used as an effective and inexpensive treatment for horses with large intestine impaction (Lopes et al. 1999). Since fluids administered intragastrically can rapidly reach the large intestine (Alexander and Benzie 1951, Argenzio et al. 1974), it is reasonable to expect that fluids administered by nasogastric tube will promote hydration of large intestine ingesta shortly after administration. Enteral administration of large volumes (10 liters / h) of an electrolyte solution has been shown to be more effective in promoting ingesta hydration than IV fluid therapy (10 liters / h) and enteral MgSO_4 (1 g/kg) combined (Lopes et al. 2002c). However the effects of fluids administered enterally at a slower rate have not been investigated nor compared to IV fluid therapy or laxatives. Furthermore the effects of an electrolyte solution have not been compared to the effects of plain water administered enterally.

The objective of this experiment was to assess the systemic and gastrointestinal effects of IV administration of lactated Ringer's solution and nasogastric administration of an electrolyte solution, water, MgSO_4 and NaSO_4 to horses with a right dorsal colon fistula. It was hypothesized that enteral administration of an electrolyte solution with sodium, potassium and chloride concentrations similar to those of plasma would produce maximal hydration of ingesta with minimal changes in plasma electrolytes, plasma protein and PCV. It was also hypothesized that IV fluid therapy, enteral water and laxatives would be less effective in promoting ingesta and fecal hydration.

Material and methods

This study was approved by the Virginia-Tech Animal Care Committee. Seven horses with an experimental right dorsal colon fistula were used in this study: 6 geldings and 1 mare; 5 Thoroughbred and 2 Quarter Horse; 6 to 17 years old (mean, 10 years old); weighing 447 to 581 kg (mean, 514.7 kg). The right dorsal colon fistula had been created 32 to 75 days (mean, 48.7 days) before the start of the experiment. The technique to create the right dorsal colon fistula has been described elsewhere (Lopes et al. 2002a). For at least 5 days before each trial the horses were kept in stalls and had free access to water, a salt block ^a and orchard grass hay. A single batch of hay was used for the entire study.

Experimental conditions - The study was conducted in a crossover design to compare 6 experimental conditions: 1) Control – no treatment was administered, but a nasogastric tube ^b was passed; 2) MgSO_4 – treated with 1 g/kg $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ ^c dissolved in 1 liter of water by nasogastric tube at time 0; 3) Na_2SO_4 – treated with 1 g/kg Na_2SO_4 anhydrous ^d dissolved in 3 liters of water by nasogastric tube at time 0; 4) IV fluid therapy – treated with 60 liters of

lactated Ringer's solution ^e through a catheter ^f in the jugular vein for the first 12 h (5 liters/h); 5) Enteral water – treated with 60 liters of tap water through a nasogastric tube for the first 12 h (5 liters/h); 6) Enteral electrolyte solution – treated with 60 liters of an electrolyte solution made of 5.27 g of NaCl ^g, 0.37 g of KCl ^h and 3.78 g of NaHCO₃ ⁱ per liter of tap water through a nasogastric tube for the first 12 h (5 liters/h). The order of the treatments was randomized. The electrolyte loads provided by the laxatives and by the enteral electrolyte solution and intravenous lactated Ringer's are shown in Table 6.1. For all experimental conditions the observation period lasted 48 h: The nasogastric tube was kept in place with the horses muzzled for the initial 24h (no access to hay or salt, but free access to 2 buckets each containing 12 liters of water); then the nasogastric tube and the muzzle were removed and the horses had free access to orchard grass hay, a salt block ^a and water for the last 24 h.

Clinical assessment and sample collection - For clinical examination and sample collection the horses were restrained in stocks. Starting immediately before and during the 48 h observation period, a clinical examination was performed every 6 hours. At the same times, blood was collected from a catheter in the jugular vein, ingesta was collected from the right dorsal colon fistula and fecal samples were collected from the rectum or from the floor immediately after defecation. Immediately after performing the first clinical examination and collecting the first set of samples (time 0), cobalt-EDTA (0.04 g / kg) dissolved in 1 liter of water was administered by nasogastric tube ^c. Cobalt-EDTA was prepared as previously described (Uden et al. 1980). The water loads provided by all experimental conditions are shown in Table 6.1. During the 48 h trials environmental temperature and humidity were recorded every 6 h ^j, and water consumption was estimated by measuring the volume needed to refill the buckets.

Sample analyses - Blood samples were immediately analyzed for PCV using the microhematocrit technique and plasma protein using a refractometer ^k. Plasma was pipetted off after 10 minutes of centrifugation at 1124 xg and frozen at -70°C for posterior analysis of electrolytes. Automatic analyzers with ion specific electrodes were used to measure plasmatic concentration of sodium, potassium, chloride, total calcium, total magnesium ^l and ionized calcium ^m. Ingesta and fecal samples were divided in 2 aliquots. From one aliquot, the liquid phase was extracted by filtration in gauze and frozen at -70°C for posterior analysis of cobalt concentration. The other aliquot was weighed immediately after collection, dried in an oven ⁿ at 90° C and repeatedly weighed until no change in the weight was detected. Ingesta and fecal hydration was calculated using the formula: water content = (wet weight – dry weight) / wet weight. Cobalt concentration of the liquid phase of ingesta and feces was measured by atomic absorption spectrophotometry ^o. Sodium and magnesium concentration of the liquid phase of ingesta and feces was measured by inductively coupled plasma atomic emission spectrometry ^p.

Statistical analysis - The MIXED procedure of the SAS System ^q was used to perform a mixed model repeated measures analysis of variance to test for effects of treatment, time and treatment by time interaction while controlling for horse and period effects. Significant interactions were further investigated using the SLICE option to test for the simple main effect of treatment within each time. The mean retention time of cobalt in ingesta and feces was calculated using the noncompartmental approach ($MRT = \sum t_i c_i / \sum c_i$ where t_i = time and c_i = cobalt concentration) (Riviere and Williams 1999). For this variable the MIXED procedure of the SAS System ^q was used to perform a mixed model analysis of variance to test for effects of treatment.

Results

Due to problems with the colostomy, two horses were removed from the study and could not be submitted to all experimental conditions. Intravenous fluid therapy was only administered to 5 horses, while all other treatments were administered to 6 horses. Complications associated with the treatments were only seen in the largest horse of this group. After receiving 50 liters of electrolyte solution via nasogastric tube this horse had moderate colic (pawing, recumbency and rolling), which was managed by interrupting fluid therapy and administering flunixin meglumine (250 mg IV). It was observed in all the trials that this horse would be recumbent most of the time whenever he was muzzled. A few weeks later, the same horse had severe colic (pawing and rolling violently), gastric reflux, reduced intestinal sounds and synchronous diaphragmatic flutter 19 h after the administration of sodium sulfate. The horse was treated with flunixin meglumine (500 mg IV), xylazine (300 mg IV), 0.9% sodium chloride (10 L IV), lactated Ringer's solution (60 L IV) and 23% calcium gluconate (240 ml IV). Seventeen hours later the horse appeared normal and had good appetite and was returned to free choice hay. Both the enteral electrolyte solution and the sodium sulfate trials were repeated one week after the original trial and no complication was seen.

During the trials, environmental temperature ranged from 10 to 32° C and environmental humidity ranged from 34 to 90%. During the first 6 h of the fasting period, water consumption was higher when the horses were treated with sodium sulfate, while no difference between the other experimental conditions could be detected. During the remainder of the fasting period and after feed was offered, no effect of treatment on water consumption could be detected. A marked increase in water consumption was seen after the horses were returned to free choice hay (Figure 6.1). PCV and plasma protein increased after feed was offered, but there was no effect of treatment or treatment by time interaction (Figure 6.2).

Enteral administration of sodium sulfate and water were the only treatments that affected plasma sodium concentration: sodium sulfate produced hypernatremia, while water produced hyponatremia. With all treatments, plasma sodium concentration tended to be lower after the horses had free access to hay (Figure 6.3). All treatments affected plasma chloride concentration except enteral administration of magnesium sulfate and water: enteral electrolyte solution and IV lactated Ringer's solution produced an increase in plasma chloride, while enteral water and sodium sulfate produced a decrease in plasma chloride. With all treatments, plasma chloride concentration returned to baseline levels after the horses had free access to feed (Figure 6.3). Sodium sulfate was the only treatment that affected plasma potassium concentration, which was low throughout the initial 24 h. With all treatments an increase in plasma potassium concentration was seen after feed was offered (Figure 6.4). All treatments except enteral administration of MgSO₄ produced hypomagnesemia. This effect was more pronounced with IV fluid therapy, enteral Na₂SO₄ and enteral electrolyte solution. When feed was offered there was an increase in plasma magnesium concentration except when MgSO₄ was administered (Figure 4). All treatments produced a drop in plasma total calcium concentration, but hypocalcemia was only seen after sodium sulfate administration (during the entire fasting period) and 6 h after the termination of the enteral administration of the electrolyte solution. Total calcium concentration returned to baseline levels after feeding (Figure 6.5). Plasma ionized calcium also dropped during fasting and only sodium sulfate administration produced changes relative to the control. From 6 to 30 h after sodium sulfate administration, plasma ionized calcium concentration was

lower than the control and below the normal limit. A rise in plasma ionized calcium was seen after feeding (Figure 6.5).

Only the enteral electrolyte solution and Na_2SO_4 produced a marked increase in ingesta water content, while enteral water produced a transient increase in ingesta water content. Magnesium sulfate and IV fluid therapy did not have any effect. No difference between treatments could be detected after feed was offered, which produced an increase in ingesta water content except when enteral electrolyte solution and Na_2SO_4 were administered (Figure 6.6). Enteral administration of the electrolyte solution, Na_2SO_4 and MgSO_4 produced an increase in fecal hydration. This increase occurred sooner with the enteral electrolyte solution, but was more pronounced with Na_2SO_4 . Changes produced by MgSO_4 could not be detected before 30 h. Enteral water and IV fluid therapy did not have any effect on the water content of the feces. An increase in water content was seen after feed was offered with all treatments except sodium sulfate (Figure 6.6).

There was no difference in ingesta cobalt concentration between the control and any treatment or between treatments during the fasting period. When the enteral electrolyte solution was administered, ingesta cobalt concentration was lower than in the control 6 h after feed was offered. Ingesta cobalt concentration was higher at 36 h with intravenous fluid therapy than with sodium sulfate, enteral electrolyte solution and water administration. When the enteral electrolyte solution was administered, fecal cobalt concentration at time 18 h was higher than with the control and magnesium sulfate administration. When sodium sulfate was administered, fecal cobalt concentration at time 30 h was lower than with both enteral administration of electrolyte solution and water, although at that time no treatment was different from the control. Six hours later, with the sodium sulfate treatment fecal cobalt concentration was lower than with the control and enteral administration of water. Although at that time no other treatment was different from the control, with magnesium sulfate and intravenous fluid therapy the fecal cobalt concentration was lower than with enteral water. Fasting shifted the curves of cobalt concentration in ingesta and feces to the right (Figure 6.7). When enteral fluid therapy was administered, the MRT of cobalt in ingesta was shorter than when the horses were in the control trial or treated with magnesium sulfate or IV fluid therapy. When enteral fluid therapy and sodium sulfate were administered, the MRT of cobalt in feces was shorter than when the horses were in the control trial. No other difference on MRT of cobalt in ingesta or feces was detected (Figure 6.8).

Fasting produced a gradual increase in ingesta sodium concentration which fell after feed was offered. Only sodium sulfate administration produced sodium ingesta concentrations higher than the control. However with the enteral electrolyte solution ingesta sodium was not different than with the sodium sulfate treatment at any time. With both enteral administration of electrolyte solution and sodium sulfate fecal sodium concentration was higher than the control. However, with the enteral electrolyte solution, fecal sodium concentration was lower than with sodium sulfate from time 24 h until the end of the observation period. No other treatment produced fecal sodium concentration different than the control at any time (Figure 6.9).

Only magnesium sulfate administration produced an increase in ingesta and fecal magnesium concentration relative to the control. When the enteral electrolyte solution was administered, fecal magnesium concentration was lower than with the control at time 24 h. When sodium sulfate was administered, fecal magnesium concentration was lower than with the control at times 24 h and 30 h and lower than with enteral water at time 36 h (Figure 6.10).

Discussion

In this study, enteral administration of an electrolyte solution containing 135 mmol of Na⁺, 5 mmol of K⁺ and 95 mmol of Cl⁻ per liter was effective in promoting hydration of colonic ingesta. The rapid transit of the fluid phase of ingesta through the small intestine (Alexander and Benzie 1951, Argenzio et al. 1974) can explain why enteral fluid therapy is so effective. Evidence of the laxative effect of enteral fluid therapy has been shown in previous studies in normal horses (Freeman et al. 1992, Lopes et al. 2002b, Lopes et al. 2002c) and in horses with large colon impaction (Lopes et al. 1999). The ingesta hydration and ingesta cobalt concentration curves and the MRT suggest that enteral fluid therapy was more effective than sodium sulfate in increasing the water content of ingesta and promoting transit of water through the gastrointestinal tract. The observation of less pronounced changes on plasma electrolyte concentrations with enteral fluid therapy further indicate that this treatment was advantageous over saline cathartics. It may be possible to adjust fluid composition and rate of administration of enteral fluid therapy in order to produce even less variation in plasma electrolytes, to compensate for ongoing losses or to correct pre-existing electrolyte imbalances.

The effectiveness of enteral administration of the electrolyte solution contrasts with the modest changes in ingesta hydration produced by enteral administration of water. This difference can be explained by the electrolyte loads provided by these treatments. Studies in other species have shown that the proximal small intestine is highly permeable to water and functions as an equilibration chamber. The net movement of fluids across the gastrointestinal mucosa is determined by the osmolality of ingesta in this segment of the gastrointestinal tract (Johnson 2001). Thus, a major portion of the tap water should be expected to be rapidly absorbed from the proximal gastrointestinal tract and not reach the large intestine. However it was not possible to detect any difference in PCV and plasma protein after water administration when compared to electrolyte solution administration, as would be expected with an increase in plasma volume. Perhaps the fluid load was insufficient to change PCV and plasma protein, which are not sensitive or specific indicators of plasma volume (George 1994). Furthermore these variables were only evaluated every 6 h and transient increase in plasma volume may have occurred. Alternatively, rapid elimination of the absorbed water could have happened through the kidneys, which might have minimized the changes in plasma volume. To better investigate these mechanisms it would be necessary to assess changes in PCV and plasma protein more frequently, to use more specific and sensitive methods to detect changes in plasma volume (i.e., indocyanine green dilution method) (Clarke et al. 1990), and to measure urine production. In addition to being less effective in promoting ingesta hydration, enteral water produced hyponatremia, which indicates that large volumes of water should not be used for enteral fluid therapy. Severe hyponatremia caused by water intoxication is known to cause life-threatening neurologic dysfunction (Fraser and Arieff 1997, Androque and Madias 2000). The findings of this study as well as the results of a previous study (Monreal et al. 1999) demonstrate that, even in normal horses, enteral administration of large volumes of water can produce hyponatremia.

Although increased fecal hydration was seen after enteral administration of the electrolyte solution, diarrhea, as reported in a previous study (Lopes et al. 2002b), was not consistently observed. This can be explained by the different fluid composition and rate of administration used in the two studies. In the previous experiment, within 8 h, the horses had been treated with 1.52 times the amount of sodium (12320 mmol) and 2.16 times the amount of chloride (12320 mmol) administered in this study over 12 h, and the volume administered per

hour was twice (10 liters) the volume used in this study. However, in the earlier study, the high electrolyte load resulted in hypernatremia and hyperchloremia, which was not seen in this study.

Abdominal discomfort seen in one horse after the administration of 50 liters of the electrolyte solution suggested intolerance to enteral fluid therapy. This was the largest horse of the group and thus received the smallest dose of fluids relatively to body size. Later this horse did not demonstrate abdominal pain when treated twice with 60 liters of enteral fluid therapy. Since this horse tended to become recumbent as soon he was muzzled and he spent most of the time in lateral recumbency while enteral fluid therapy was being administered, it is possible that gastric emptying had been compromised due to his posture. Alternatively, this horse may have been simply less tolerant to enteral fluid therapy. Thus whenever enteral fluid therapy is used, careful introduction and continuous assessment is indicated.

The effectiveness of sodium sulfate in promoting ingesta and fecal hydration was likely a result of the high sodium and sulfate load provided by this treatment. Because these horses had free access to water, the high sodium load was followed by consumption of large volumes of water. It is likely that the addition of about 20 liters of water (3 liters administered with the laxative plus the mean voluntary consumption of 17 liters) contributed to the increase in ingesta and fecal hydration seen after sodium sulfate administration. The large sodium load followed by hypernatremia can explain the high water consumption during the first 6 h, since plasma osmolality is one of the main thirst stimuli in horses (Sufit et al. 1985). In this group of healthy horses the large electrolyte load provided by sodium sulfate produced hypernatremia, hypocalcemia and hypochloremia, which was followed by clinical signs characteristic of hypocalcemia in one case. The occurrence of hypocalcemia in these horses is likely to be explained by binding of calcium ions to sulfate, a mechanism that has been demonstrated in humans (Cole et al. 1989). Unfortunately plasma concentration of sulfate was not measured, but, in humans, it has been demonstrated that sulfate can be absorbed after oral administration leading to an increase in plasma sulfate (Cocchetto and Levy 1981). Although acid-base status was not assessed in this study, it is likely that the hypernatremia and hypochloremia caused by sodium sulfate administration could have produced alkalosis (Whitehair et al. 1995), which would also contribute to reduce the concentration of ionized calcium (Kohn and Brooks 1990). Considering the importance of calcium for several physiologic mechanisms including gastrointestinal motility (De Ponti et al. 1993, Fenger 1998) and because hypocalcemia is frequently observed in horses with gastrointestinal disease (Dart et al. 1992, Garcia-Lopez et al. 2001), it does not seem appropriate to administer 1 g/kg of anhydrous sodium sulfate as a laxative for horses with colic. Or if this dose of sodium sulfate is administered, close monitoring of plasma electrolytes is mandatory.

In contrast to what was observed with sodium sulfate administration, treatment with magnesium sulfate did not produce any change in ingesta hydration and produced less pronounced changes in fecal hydration. Furthermore magnesium sulfate did not produce hypocalcemia. These differences may be explained by the different amount of electrolyte provided by magnesium sulfate. The amount of sulfate provided by 1 g/kg of $\text{MgSO}_4 \cdot 7\text{H}_2\text{O}$ was about 57.52% of that provided by 1 g/kg of anhydrous Na_2SO_4 . Although hypermagnesemia has been reported in dehydrated horses treated with high doses of magnesium sulfate (Scarratt and Swecker 1999), in this study only an increase in plasma magnesium concentration within normal limits was produced.

Most changes in ingesta and fecal electrolyte composition can be explained by the different electrolyte loads provided by each of the treatments. However, despite the higher

sodium load provided with enteral administration of the electrolyte solution, higher concentrations of sodium in ingesta and feces were seen after sodium sulfate administration. This finding suggests that more sodium was absorbed when the enteral electrolyte solution was administered. It has been suggested that sulfate may impair sodium absorption in the large intestine, which may contribute to the laxative effect of sodium sulfate (Meyer et al. 1986). It is likely that the larger volume of water provided with the enteral electrolyte solution may have prevented an increase in plasma sodium due to dilution or by facilitating water excretion. The fair temporal coincidence of the changes in electrolyte concentration, water content and cobalt concentration in ingesta and feces and the ordinal manifestation of changes (first in colonic ingesta and later in feces) suggests that these treatments acted predominantly by direct intraluminal effects and not by reflex mechanisms (e.g., gastrocolic response).

Although it has been suggested that the overhydration status produced by IV fluid therapy may promote water secretion into the gastrointestinal tract (White and Dabareiner 1997), in this study IV administration of 5 liters of lactated Ringer's solution every hour for 12 hours to normally hydrated horses did not have any effect on ingesta or fecal hydration. In normally hydrated dogs IV fluids administered at an extremely high rate (150 ml/kg/h) for one hour were found to increase fluid secretion into the gastrointestinal tract (Duffy et al. 1978). To achieve this rate of infusion in the group of horses used in this study, about 76 liters of fluids would have to be administered every hour which would make this treatment extremely expensive and could have life-threatening side effects such as pulmonary edema (Cornelius et al. 1978, Gabel et al. 1986).

In this study fasting and feeding had major effects on water intake, hydration status, plasma electrolytes and ingesta and fecal composition. These effects of fasting and refeeding may have masked some treatment effects. The current recommendation for large intestine impaction is to fast the horse until the impaction is completely resolved to avoid further increase in the impacted mass (White and Dabareiner 1997, Dabareiner 1998, Sullins 1999). However some effects of fasting demonstrated in this study (i.e., delay in gastrointestinal transit, reduction in ingesta hydration and electrolyte imbalances) cannot be ignored. Although it is obvious that feed deprivation is appropriate for many horses with colic, offering feed early may be beneficial in some situations. The practice of offering feed with low fiber content to horses with large colon impaction as soon as pain is no longer observed, but before complete resolution of the impaction has been reported (Lopes et al. 1999).

Several benefits of enteral fluid therapy have been demonstrated in this and in other studies, but many aspects related to the effects of enteral fluid therapy have still to be investigated. In the meantime, it may be appropriate to recommend that enteral fluid therapy be used with caution. There is evidence that it is safe to administer large volumes of isotonic or slightly hypotonic electrolyte solutions through a nasogastric tube to horses with no sign of severe compromise of the gastrointestinal transit (i.e., gastric reflux) and that this treatment can produce significant increase in ingesta hydration and contribute to correct dehydration and restore plasma electrolytes. This study also demonstrated that enteral administration of water saline cathartics can produce severe electrolyte imbalances and have limited effect on ingesta hydration, while intravenous fluid therapy (5 L/h/12 h) does not promote ingesta hydration in normal horses.

Manufacturer's address

- ^a Rotomin, Roto Salt, Co. Penn Yan, New York.
- ^b 18 French Equine Enteral Feeding Tube, Mila International, Inc. Florence, Kentucky.
- ^c Epsom Salt, Bindley Western Industries, Indianapolis, Indiana.
- ^d Sodium Sulfate Anhydrous 99%, Fisher Scientific,
- ^e Veterinary Lactated Ringer's, Abbott Laboratories Inc., North Chicago, Illinois.
- ^f 14G Abocath-T, Abbott Laboratories Inc., North Chicago, Illinois.
- ^g Mix-N-Fine Salt, Cargill, Inc., Minneapolis, Minnesota.
- ^h Potassium Chloride, ICN Biomedicals, Inc., Aurora, Ohio.
- ⁱ Baking Soda, Church & Dwight, Co., Princeton, New Jersey.
- ^j Thermometer / Humidity Metter, Springfield Precision Instruments, Wood-Ridge, New Jersey.
- ^k Hand-Held Refractometer, Westover Scientific, Woodenville, Washington.
- ^l Olympus AU400, Olympus America, Inc., Melville, New York.
- ^m Rapidlab 348, Bayer, Co., East Walpole, Massachusetts.
- ⁿ Precision Mechanical Convection Oven, Precision Scientific, Chicago, Illinois.
- ^o SpectrAA 220FS, Varian Inc., Walnut Creek, California.
- ^p SpectroFlame Modula Tabletop ICP, Spectro Analytical Instruments, Inc., Fitchburg, Massachusetts.
- ^q SAS System – 8e, SAS Institute, Inc., Cary, North Carolina.

Table 6.1 – Mean electrolyte loads and fluid loads during a 24 h fasting period in horses muzzled and treated with MgSO₄ (1 g / kg in 1 liter of water) via nasogastric tube (Mg), Na₂SO₄ (1 g / kg in 3 liters of water) via nasogastric tube (Na), lactated Ringer’s (5 liters / h for 12h) administered IV (IV), water (5 liters / h for 12h) via nasogastric tube (W) and an electrolyte solution (5 liters / h for 12h) via nasogastric tube (E) or not treated (C). For all trials including the control, 0.04 mg/kg of CoEDTA in 1 liter of water was also administered via nasogastric tube.

Variables	C	Mg	Na	IV	W	E
Na+ (mmol)	0	0	7221	7800	0	8100
K+ (mmol)	0	0	0	240	0	300
Ca++ (mmol)	0	0	0	81	0	0
Mg++ (mmol)	0	2077	0	0	0	0
Cl- (mmol)	0	0	0	6540	0	5700
SO ₄ -- (mmol)	0	2077	3610	0	0	0
Lactate (mmol)	0	0	0	1680	0	0
HCO ₃ - (mmol)	0	0	0	0	0	2700
Water intake (L)	0.60	6.70	17.92	2.20	0.33	0.25
Total fluids (L)	1.60	8.70	21.92	63.20	61.33	61.25
Total fluids/kg (ml/kg)	3.10	17.03	43.39	125.30	122.24	119.92

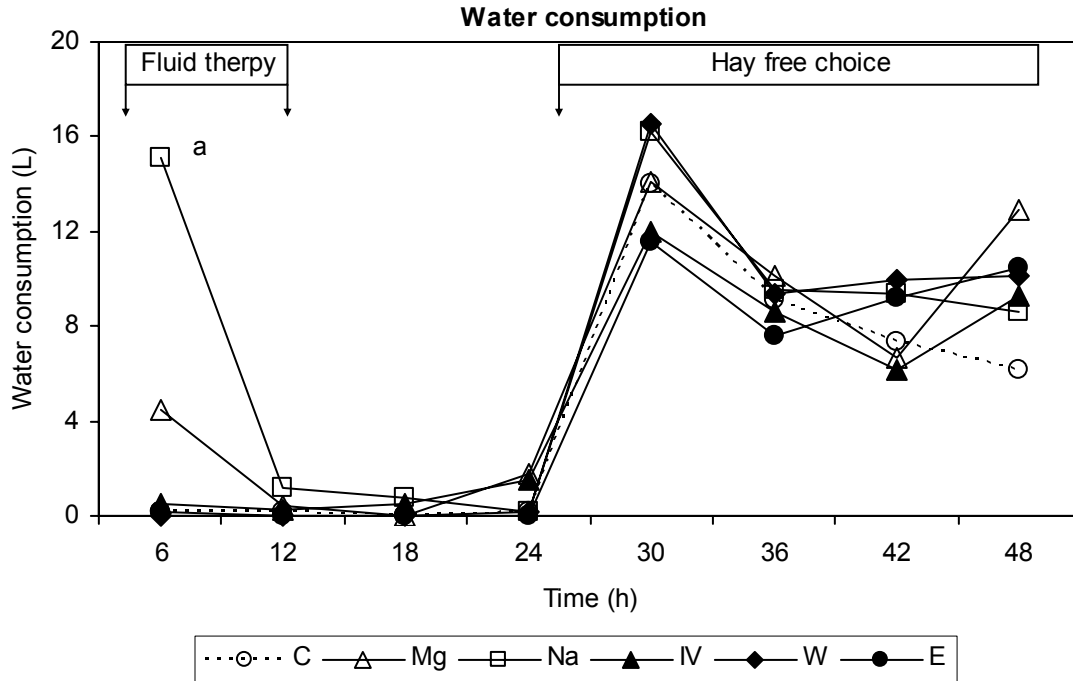


Figure 6.1 – Water consumption in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). There was time effect (P<0.01) and treatment by time interaction (P=0.01), but no treatment effect (P=0.12). Within one time, means with letters are significantly different from the control at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

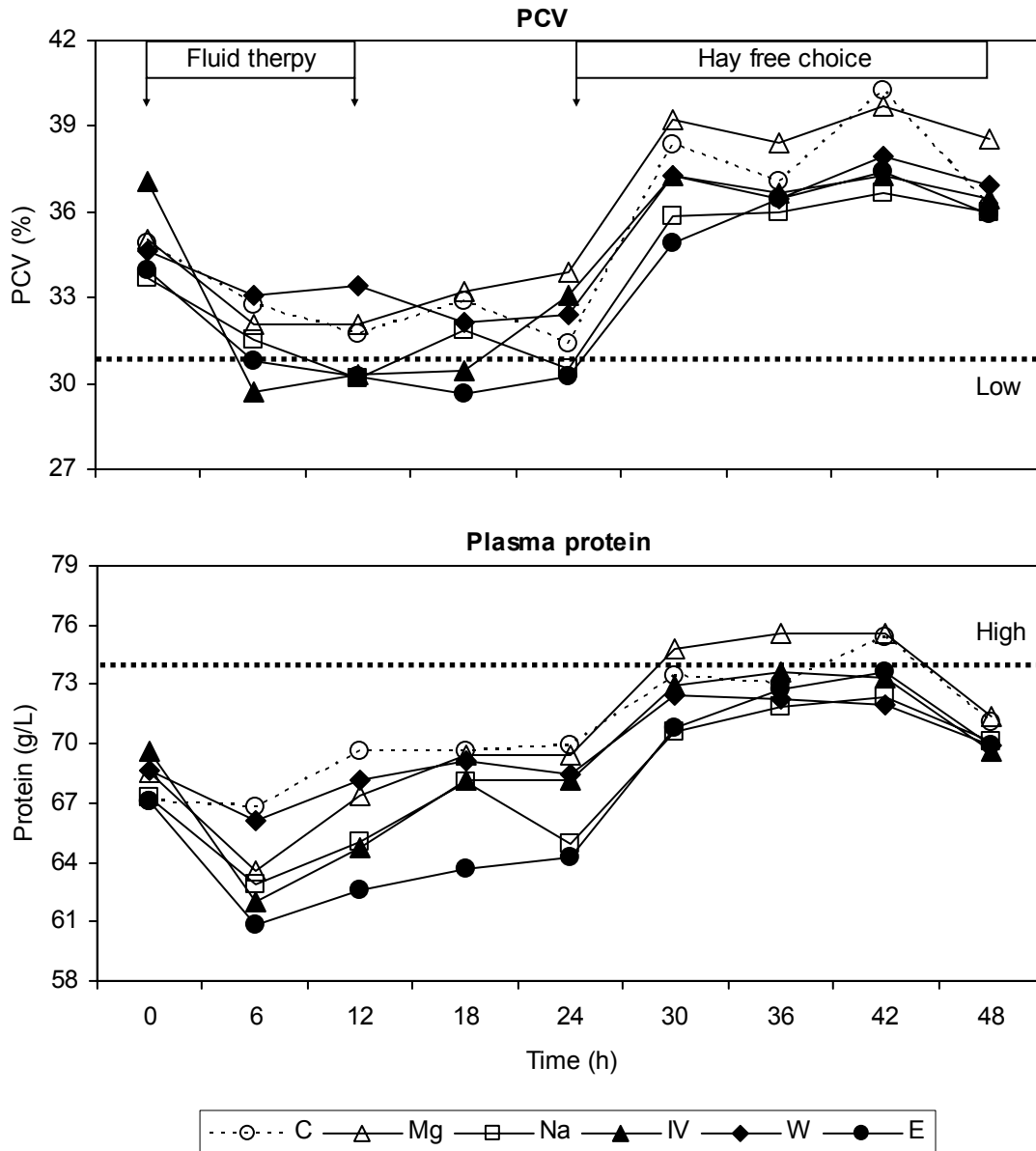


Figure 6.2 – PCV and plasma protein in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of $MgSO_4$ via nasogastric tube (Mg), 1 g / kg of Na_2SO_4 via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). The dotted lines indicate the normal limits (Parry and Brobst 1997). For both PCV and plasma protein there was time effect ($P < 0.01$), but no treatment effect ($P = 0.30$ and $P = 0.11$) or treatment by time interaction ($P = 0.90$ and $P = 0.35$).

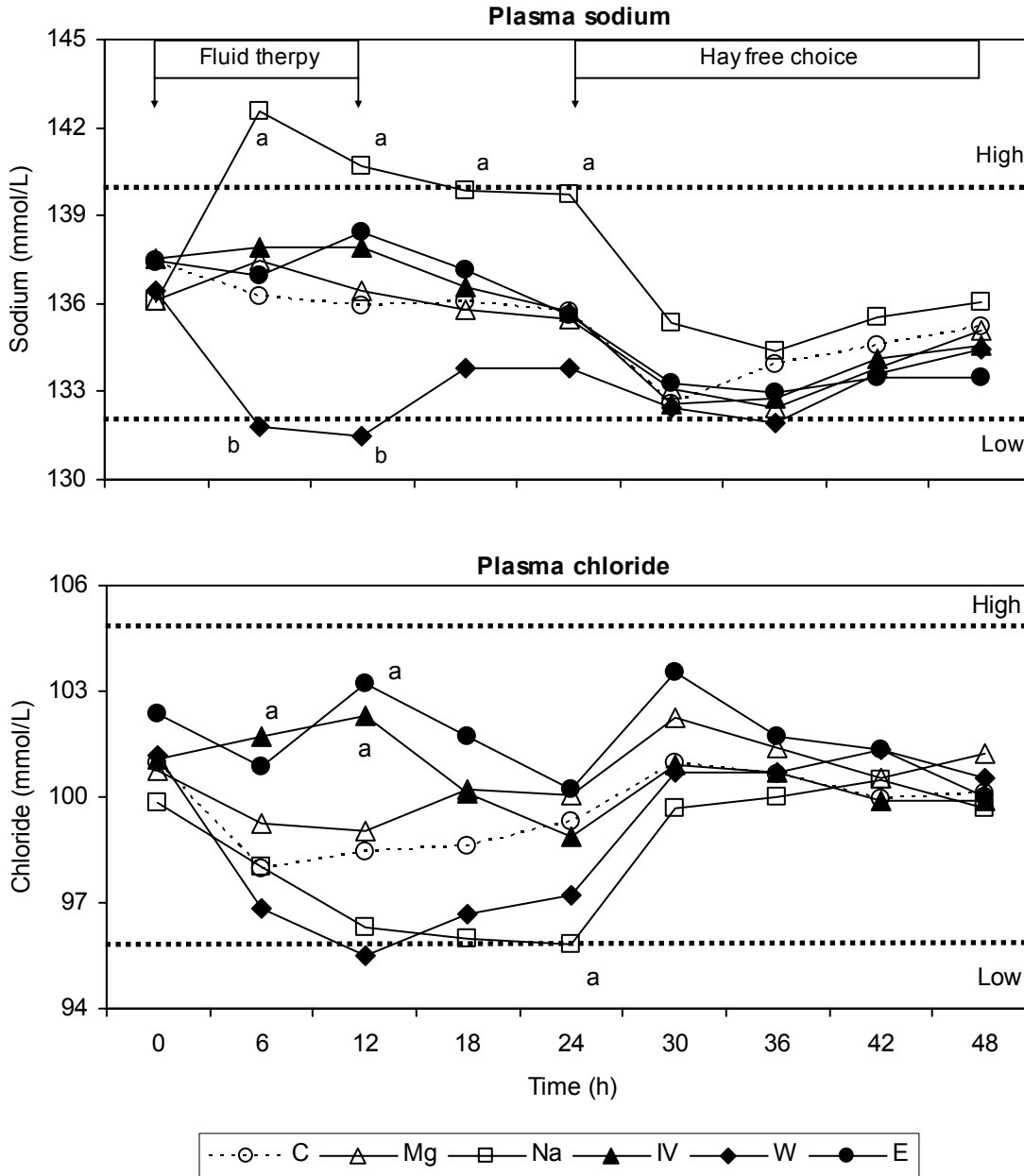


Figure 6.3 – Plasma sodium and chloride concentrations in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of $MgSO_4$ via nasogastric tube (Mg), 1 g / kg of Na_2SO_4 via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). The dotted lines indicate the normal limits of the laboratory. There was time effect ($P < 0.01$), treatment effect ($P < 0.01$) and treatment by time interaction ($P < 0.01$). Within one time, means with letters are significantly different from the control and different letters indicate difference between treatments at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

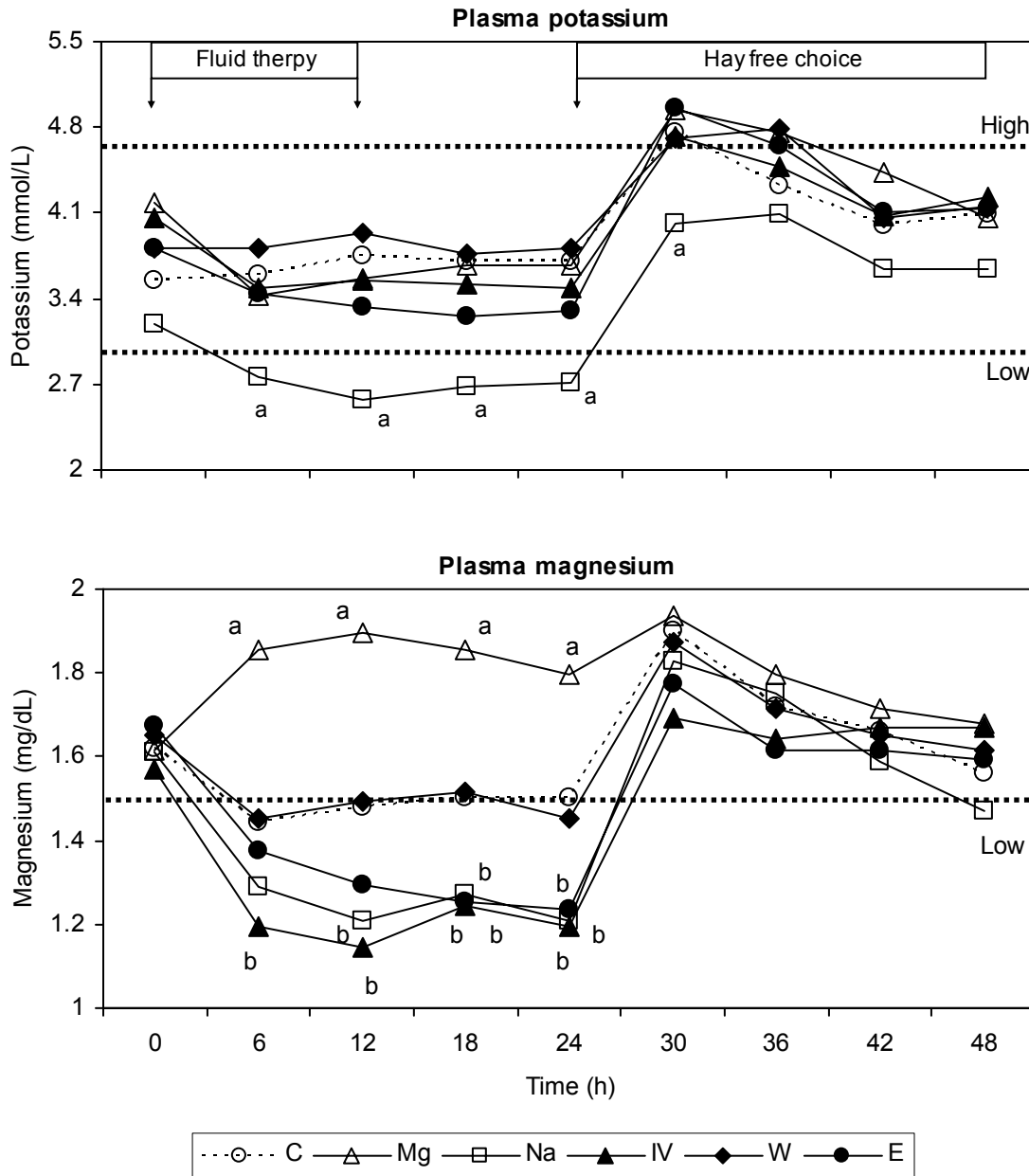


Figure 6.4 – Plasma potassium and magnesium concentrations in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). The dotted lines indicate the normal limits of the laboratory. For both variables there was time effect ($P < 0.01$) and treatment effect ($P < 0.01$). For magnesium ($P < 0.01$) but not for potassium ($P = 0.43$) there was treatment by time interaction. Within one time, means with letters are significantly different from the control and different letters indicate difference between treatments at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

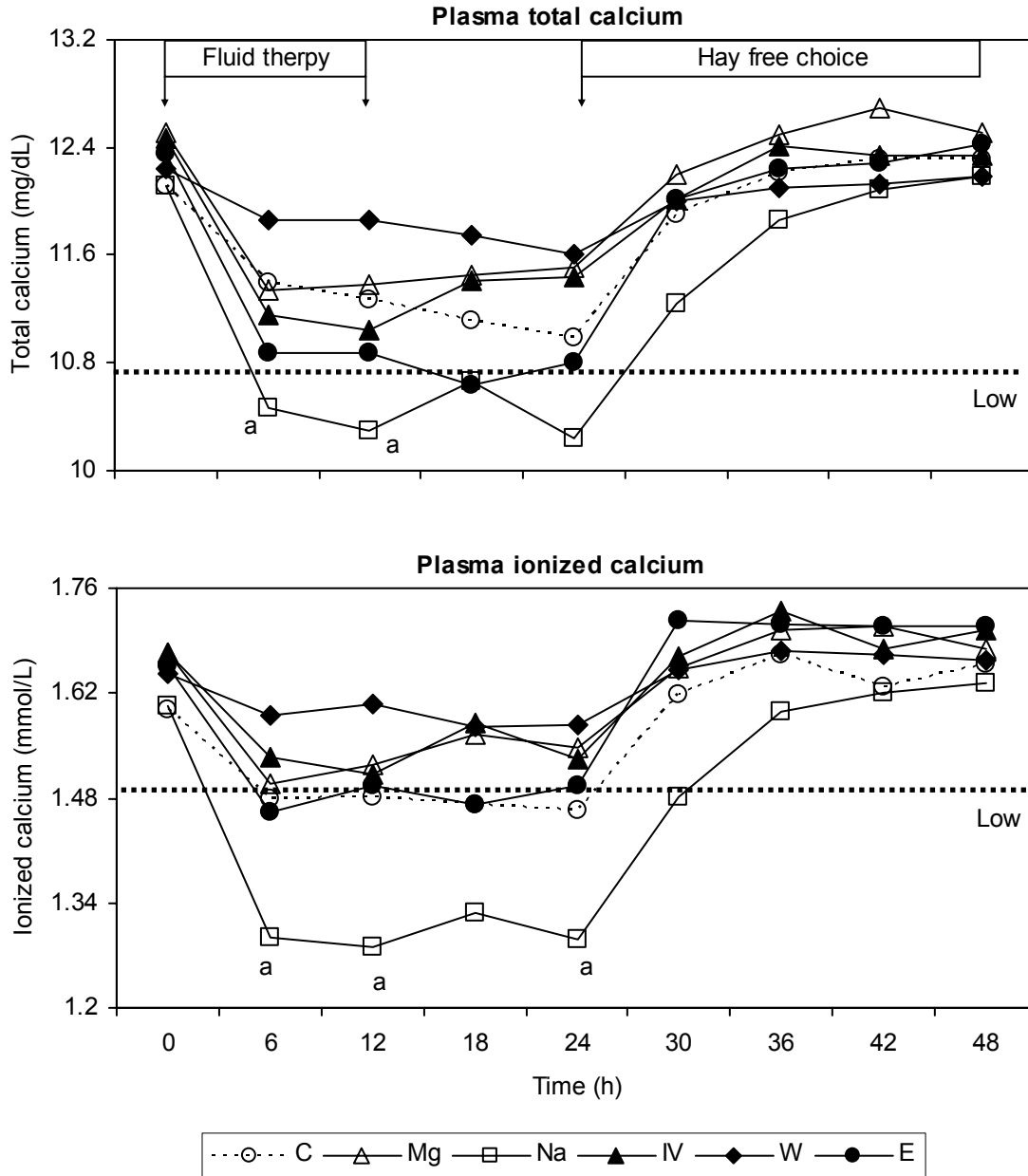


Figure 6.5 – Plasma total calcium and ionized calcium concentrations in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer's IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). The dotted lines indicate the lower limits of the laboratory (total calcium) or from the literature (Parry and Brobst 1997) (ionized calcium). There was time effect (P<0.01), treatment effect (P=0.02 and P<0.01) and treatment by time interaction (P<0.01). Within one time, means with letters are significantly different from the control at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

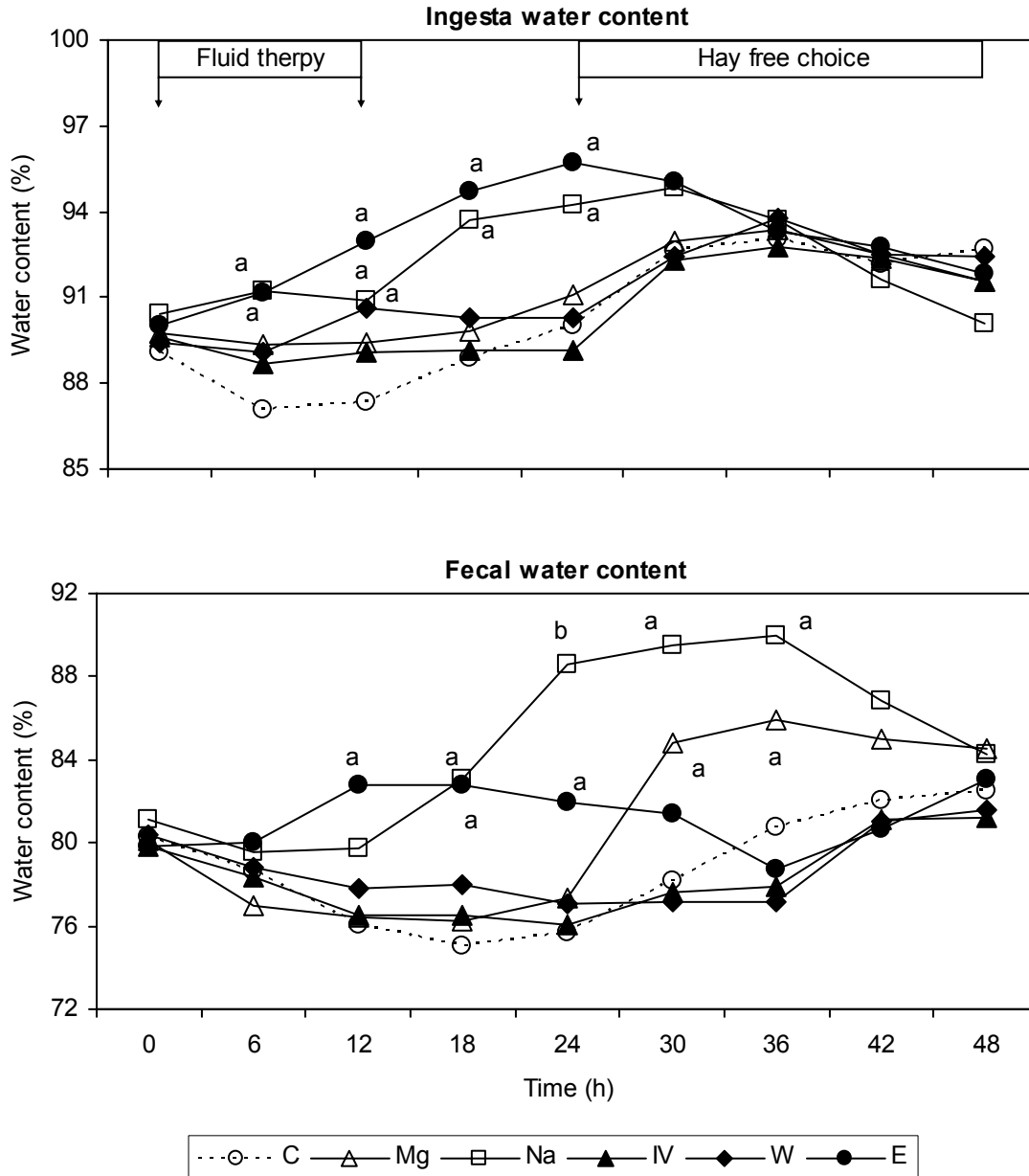


Figure 6.6 – Water content of colonic ingesta and feces in horses muzzled for 24h and not treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer's IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). There was time effect (P<0.01), treatment effect (P<0.01) and treatment by time interaction (P<0.01). Within one time, means with letters are significantly different from the control and different letters indicate difference between treatments at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

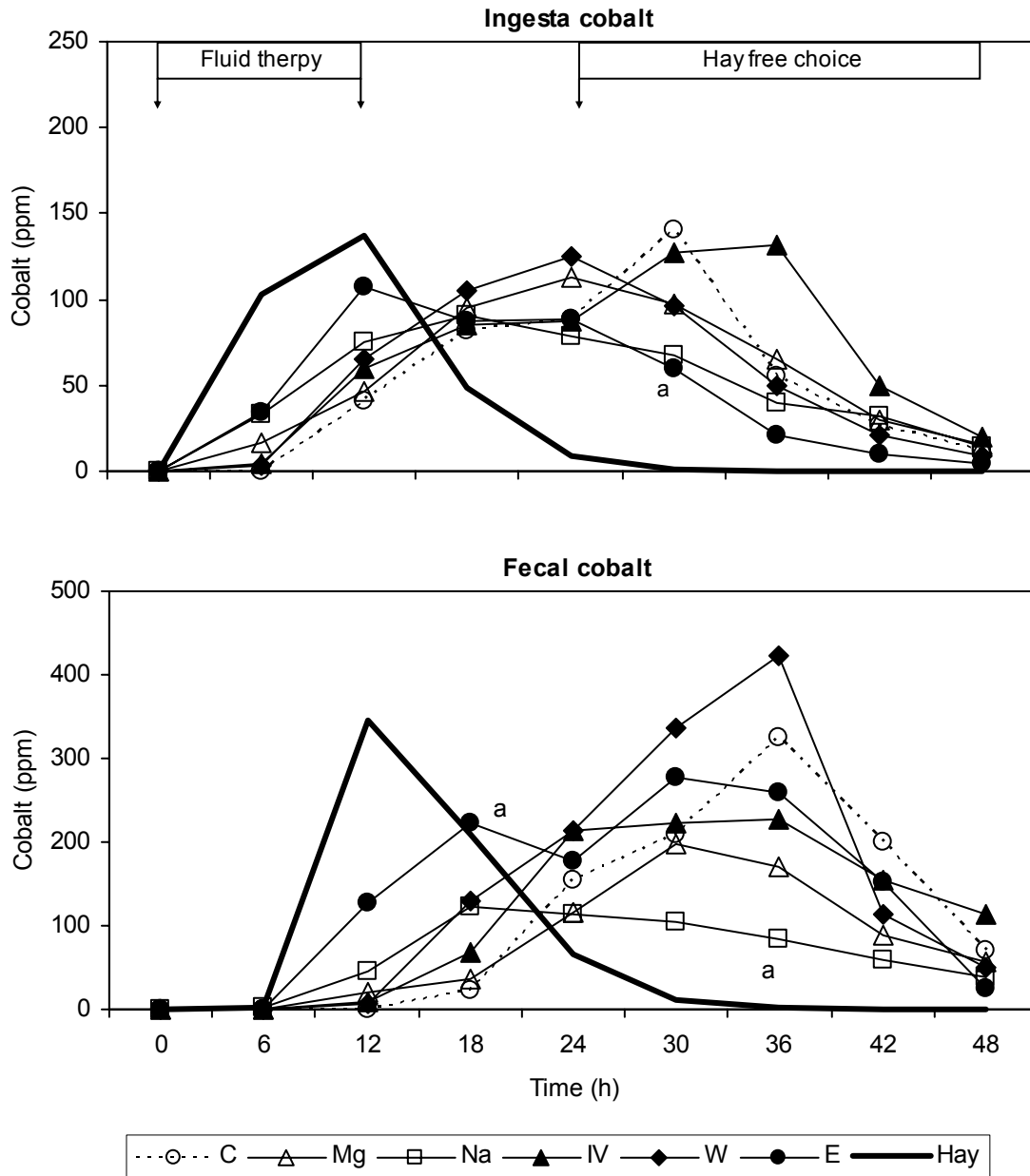


Figure 6.7 – Cobalt concentration in colonic ingesta and feces in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). Cobalt EDTA (40 mg/kg) was administered by nasogastric tube at time 0. For both variables there was time effect (P<0.01) and treatment by time interaction (P<0.05 and P<0.01). There was an effect of treatment for fecal cobalt concentration (P<0.05) but not for ingesta cobalt concentration (P=0.87). Within one time, the mean with the letter is significantly different from the control at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons. The cobalt concentration curves from the same horses while fed free choice hay (Hay) were added for comparison.

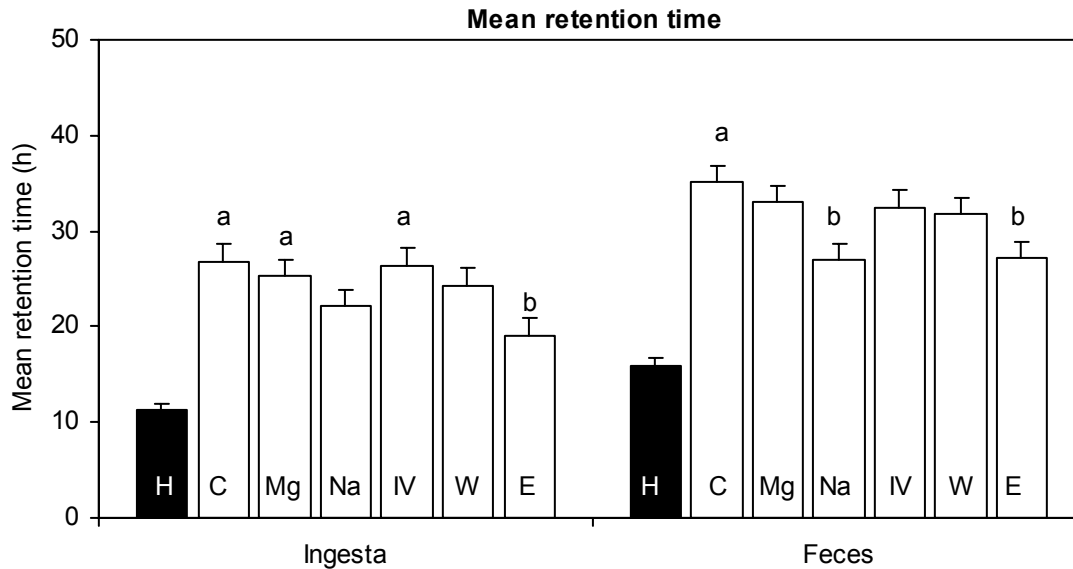


Figure 6.8 – Mean retention time (MRT) of cobalt in colonic ingesta and feces in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). Cobalt EDTA (40 mg/kg) was administered by nasogastric tube at time 0. Vertical bars represent the standard errors of the means. There was treatment effect on MRT of cobalt in ingesta (P<0.01) and feces (P<0.01). Different letters on top of the columns indicate significantly difference at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons. Mean retention time of cobalt in ingesta and feces from the same horse while fed free choice hay (H) was added for comparison.

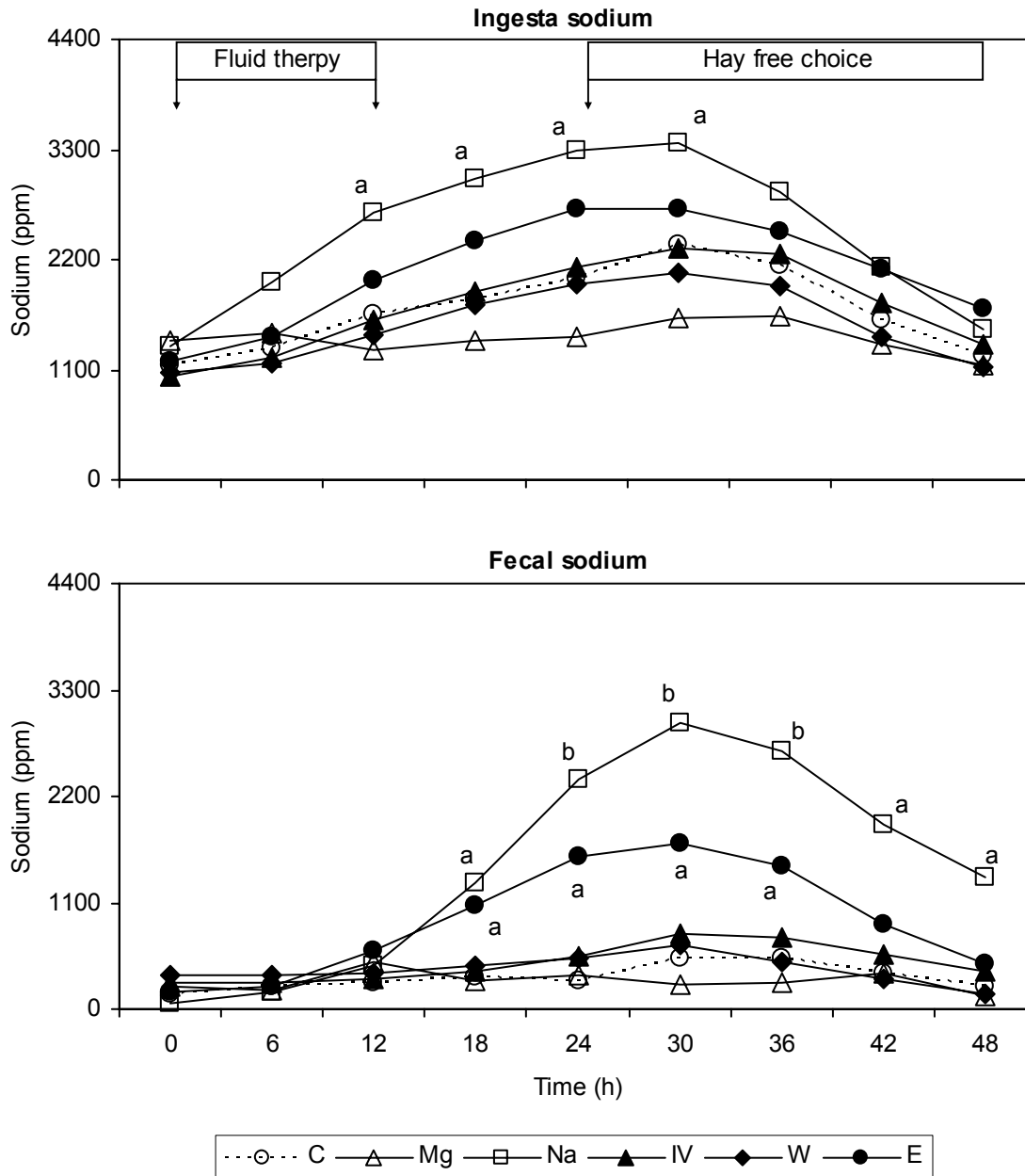


Figure 6.9 – Sodium concentration in colonic ingesta and feces in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of MgSO₄ via nasogastric tube (Mg), 1 g / kg of Na₂SO₄ via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer’s IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). There was time effect (P<0.01), treatment effect (P<0.01) and treatment by time interaction (P<0.01). Within one time, means with letters are significantly different from the control and different letters indicate difference between treatments at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

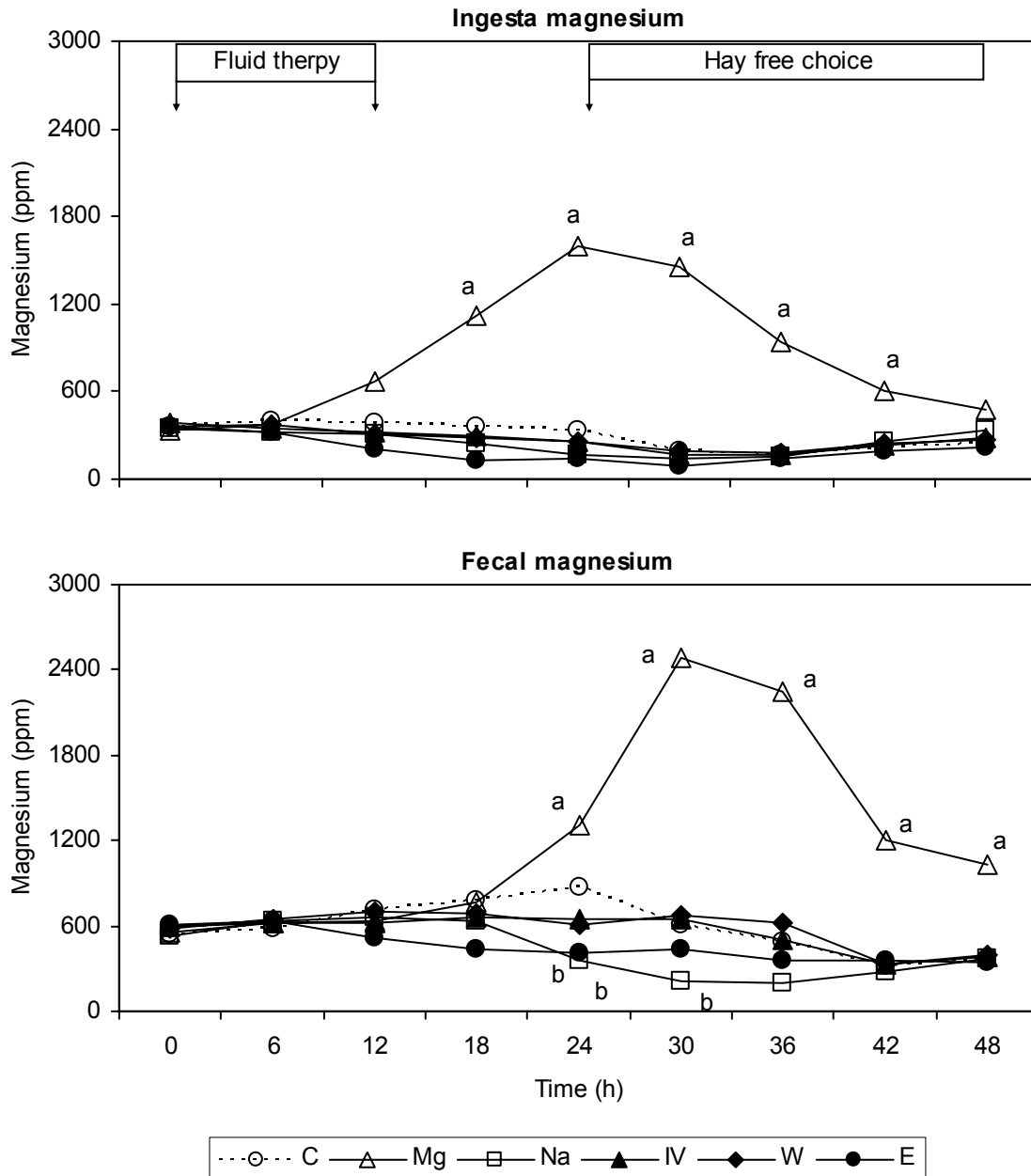


Figure 6.10 – Magnesium concentration in colonic ingesta and feces in horses muzzled for 24h and not treated (C) or treated with 1 g / kg of $MgSO_4$ via nasogastric tube (Mg), 1 g / kg of Na_2SO_4 via nasogastric tube (Na), 5 liters / h for 12h of lactated Ringer's IV (IV), 5 liters / h for 12h of water via nasogastric tube (W) and 5 liters / h for 12h of an electrolyte solution via nasogastric tube (E). For both variables there was time effect ($P < 0.01$), treatment effect ($P < 0.01$) and treatment by time interaction ($P < 0.01$). Within one time, means with letters are significantly different from the control and different letters indicate difference between treatments at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

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Chapter 7

Treatments to promote colonic hydration: Enteral fluid therapy versus intravenous fluid therapy plus magnesium sulfate

Abstract

Objective – To compare systemic and gastrointestinal effects of enteral fluids with the changes produced by intravenous fluids combined with MgSO₄.

Animals – Four horses with a fistula in the right dorsal colon.

Methods – All horses alternately received both treatments in 2 periods one week apart. Sixty liters of fluids were administered continuously (10 liters/h) through a venous catheter (lactated Ringer's) or a nasogastric tube (home made electrolyte solution containing 135 mmol/liter of Na⁺, 95 mmol/liter of Cl⁻, 5 mmol/liter of K⁺ and 45 mmol/liter of HCO₃⁻). Magnesium sulfate (1g/kg of body weight) was administered via nasogastric tube before intravenous fluid therapy.

Results – Two horses had mild abdominal discomfort at the end of enteral fluid therapy. Pollakiuria, hyposthenuria, increased body weight, increased fecal and ingesta hydration, and decreased PCV, plasma protein and plasma magnesium were produced by both treatments. Abdominal distention and more pronounced changes in body weight and ingesta hydration were seen with enteral fluids. Intravenous fluids plus MgSO₄ produced hypocalcemia and more pronounced changes in plasma protein.

Conclusions – Enteral fluid therapy was more effective in promoting ingesta hydration and produced less pronounced systemic effects than intravenous fluid therapy plus MgSO₄.

Introduction

Ingesta dehydration is a feature of large intestine impaction, the most common form of colic (White and Dabareiner 1997). Dehydrated ingesta has higher viscosity, which is associated with an increased resistance to flow (Morel et al. 1990). Thus one of the aims of the treatment of large intestine impaction is to promote ingesta rehydration with fluid therapy and/or laxatives. The combination of intravenous (IV) fluid therapy with the osmotic cathartic magnesium sulfate administered by nasogastric tube is commonly used to treat large intestine impaction. Although the effects of these treatments on ingesta hydration have not been measured, it is thought that the state of systemic overhydration produced by IV fluid therapy combined with the increase in intraluminal osmolality produced by the cathartic promotes ingesta rehydration (Freeman et al. 1992) (White and Dabareiner 1997).

Enteral fluid therapy is barely mentioned in the current literature as an alternative for horses with large intestine impaction, but it is tempting to give fluids directly into the gastrointestinal tract to promote ingesta hydration. In the horse, transit through the small intestine is rapid and fluids can quickly reach the large intestine after nasogastric administration (Alexander and Benzie 1951, Argenzio et al. 1974). Expensive sterile fluids with precisely adjusted compositions are not required for enteral fluid therapy. Nasogastric administration of fluids may stimulate colonic motility through the gastrocolic reflex, which may also contribute to relieve impaction (Freeman et al. 1992, Lopes et al. 1999). Additionally, systemic effects are

limited and electrolyte imbalances are less likely with enteral administration of fluids than with IV fluid therapy. During dehydration the renin-angiotensin-aldosterone system is activated, promoting sodium and water absorption by the gastrointestinal mucosa, while in the hydrated horse this mechanism is not active (Clarke et al. 1990, Clarke et al. 1992). Additionally, studies in other mammals have shown that the release of atrial natriuretic peptide (ANP) by the atrial myocardium, in response to stretch produced by overhydration, may contribute to decreased sodium and water absorption by the gastrointestinal mucosa (Pettersson and Jonsson 1989, Moriarty et al. 1990, Argenzio and Armstrong 1993). Although enteral fluid therapy has been reported to be an effective treatment for large intestine impaction (Lopes et al. 1999), most features associated with its use in horses have yet to be investigated including the effects on large intestine ingesta.

This experiment was conducted to compare systemic and gastrointestinal effects of enteral fluid therapy with IV fluid therapy combined with magnesium sulfate in normally hydrated horses. We hypothesized that enteral fluid therapy would be more effective in promoting ingesta hydration while producing less pronounced systemic effects than IV fluid therapy combined with enteral magnesium sulfate administration.

Materials and methods

Horses - Four mature horses (Quarter Horse gelding age 10 years, Thoroughbred mare age 17 years and 2 Thoroughbred geldings age 7 years) with an experimental fistula in the right dorsal colon were used. Bodyweight immediately before treatment were 439-595 kg (mean 497.96 kg). The technique to create the right dorsal colon fistula has been described elsewhere (Lopes et al. 2002a). Horses were maintained in stalls with free access to water, orchard grass hay and a salt block for at least a week before the experiment.

Fluids - For enteral fluid therapy the following solution was used: 5.27 g of sodium chloride (Mix-N-Fine Salt ¹), 0.37 g of potassium chloride (Potassium chloride ²) and 3.78 g of sodium bicarbonate (Baking Soda ³) per liter of tap water. The approximate calculated concentration of each ion in mmol/liter was 135 of Na⁺, 95 of Cl⁻, 5 of K⁺ and 45 of HCO₃⁻, and the calculated osmolality in mOsmol/liter was 280. Two samples (one of each of two batches) of the solution were evaluated by freezing point osmometry (Osmette A ⁴). For IV fluid therapy a lactated Ringer's solution (Veterinary Lactated Ringer's Injection, USP ⁵) was used.

Period I - Immediately before starting the treatment period the following procedures were performed in this order: 1- blood samples were collected from the jugular vein in a tube with lithium heparin; 2- ingesta samples were collected from the cannula; 3- fecal samples were collected from the rectum; 4- body weight was measured using a scale; 5- abdominal circumference was measured at the end of expiration with a graduated tape at the site with the apparent largest diameter (this site was marked by clipping the hair of 4 small areas located in the dorsal and ventral midline and in the sides of the abdomen); 6- heart rate and rectal temperature were recorded; 7- a catheter was inserted in the jugular vein (14 G Abocath-T ⁵); 8- a small bore nasogastric tube (18 French Equine Enteral Feeding Tube ⁶) was passed; 9- a muzzle was applied; 10- environmental temperature and humidity measured by a thermometer and humidity meter (Thermometer / Humidity Meter Model 90116 ⁷) were recorded.

The microhematocrit technique was used to measure PCV and plasma protein was measured with a refractometer (Hand-Held Veterinary Refractometer)⁸. Within 10 minutes after collection blood was centrifuged at 1124 xg for 10 minutes. Plasma was stored at -70° C until analysis for Na⁺, K⁺ and Cl⁻ concentration, which was performed with selective electrodes (Vet Ace⁹). Ingesta and feces were weighed (Harvard Trip Balance¹⁰), dried in an incubator (Precision Mechanical Convection Oven¹¹) at 90-100° C and weighed again until there was no longer a change in dry weight. Ingesta and fecal hydration were calculated by dividing the difference between wet weight and dry weight by the wet weight.

Two horses were randomly assigned to receive enteral fluid therapy, while the other 2 horses received IV fluid therapy plus magnesium sulfate. Fluid therapy was administered continuously at the rate of 10 liters / h for 6 h through a coiled line (STAT Large Animal IV Set¹²) connected to the nasogastric tube (18 French Equine Enteral Feeding Tube⁶) or to the catheter in the jugular vein (14 G Abocath-T⁵). Fluids were kept in a container (Nalgene¹³) (for enteral fluids) or in plastic bags (for IV fluids) hung on the ceiling about 100 cm above the horses' withers. Magnesium sulfate (Epsom Salt¹⁴) (1 g / kg of body weight) was dissolved in one liter of tap water and administered by nasogastric tube immediately before starting IV fluid therapy.

During fluid therapy the horses were allowed to move freely in the stall with free access to water and were continuously monitored for frequency of urinations and defecations, and signs of abdominal discomfort. A urine sample was collected from each urination and specific gravity was measured with a refractometer (Hand-Held Veterinary Refractometer⁸).

Blood, ingesta and fecal samples were collected, clinical variables were measured and environmental temperature and humidity were recorded again at 2 other occasions during fluid therapy (2 and 4 h after fluid therapy was initiated), after discontinuing fluid therapy, and 6, 12 and 18 h after the end of fluid therapy. Body weight and abdominal circumference were not measured during fluid therapy. After collecting the samples at the end of fluid therapy, the muzzle and nasogastric tube were removed and the horses were allowed to have free access to hay, water and a salt block.

Period II - One week after the first period all procedures were repeated, but the horses that had been treated with enteral fluid therapy received IV fluid therapy plus magnesium sulfate and vice versa.

Statistical analysis - The MIXED procedure of the SAS System – 8e¹⁵ was used to perform a mixed model repeated measures analysis of variance to test for effects of treatment, time and treatment by time interaction while controlling for horse and period effects. Significant interactions were further investigated using the SLICE option to test the simple main effect of treatment within each time.

Results

The measured osmolalities of the two samples of the solution for enteral fluid therapy were 253.7 and 255.0 mOsm/l (mean, 254.3). The temperature and relative humidity recorded during the experiment ranged from 11 to 32° C and 53 to 100% respectively. Considering the body weights

immediately before fluid administration, the rate of infusion ranged from 16.8 to 22.6 ml/kg/h (mean, 20.4 ml/kg/h).

Two horses presented signs of mild abdominal pain when treated with enteral fluid therapy. The largest horse in this group (BW, 595 kg) was observed pawing a few times after receiving 55 liters of fluids. This horse did not receive any treatment and no sign of abdominal discomfort was seen after fluid therapy. The other horse (BW, 447 kg) started pawing immediately after fluid therapy. No medication was administered and the horse was walked for 10 minutes. When returned to the stall the horse started eating and did not show any more signs of abdominal discomfort.

During fluid therapy, no horse drank and all horses had pollakiuria and hyposthenuria (urine specific gravity ranging from 1.003 to 1.008 g/ml). The mean number of urinations was 8.50 during enteral fluid therapy and 15.75 during IV fluid therapy, and there was no significant difference between treatments ($P=0.14$). The mean number of times the horses passed feces was 4.25 during enteral fluid therapy and 2.00 during IV fluid therapy, and there was a trend towards a significant difference between treatments ($P=0.06$). There was an increase in body weight at the end of both treatments ($P<0.01$), but the increase in body weight was more pronounced with enteral fluid therapy ($P=0.01$). Body weight returned to the pre-treatment values 6 h after the end of fluid therapy (Figure 7.1). An increase in abdominal circumference was seen at the end of enteral fluid therapy ($P<0.01$), while no abdominal distention was seen when the horses were treated with IV fluid therapy + $MgSO_4$ (Figure 7.1). When the horses were treated with enteral fluid therapy, larger abdominal diameter could still be detected 6 h after the end of fluid therapy ($P=0.03$). Fecal hydration was significantly increased 6 h after the end of fluid therapy with both treatments ($P<0.01$), and no difference could be seen between treatments ($P=0.75$) (Figure 7.2). Both treatments produced an increase in ingesta hydration ($P<0.01$), but more pronounced changes were seen with enteral fluid therapy after the administration of 40 liters ($P=0.02$), at the end of fluid therapy ($P<0.01$) and 6 h after the end of fluid therapy ($P=0.04$) (Figure 7.2). No difference between treatments could be detected for PCV ($P=0.31$), which was markedly reduced during and at the end of fluid therapy ($P<0.01$), and returned to the pre-treatment values 6 h after the end of fluid therapy (Figure 7.3). With both treatments plasma protein fell during fluid therapy ($P<0.01$) and recovered after fluid therapy, but 12 h after the end of fluid therapy plasma protein was higher when the horses received IV fluids + $MgSO_4$ ($P<0.05$) (Figure 7.3). Plasma sodium, potassium and chloride were not different between treatments ($P=0.28$, $P=0.61$, $P=0.90$ respectively) and changes within the normal limits were observed over time ($P<0.01$, $P=0.02$, $P<0.01$ respectively). With both treatments plasma calcium concentration fell at the end of fluid therapy ($P<0.01$) and recovered after fluid therapy, but these changes were more pronounced ($P<0.01$) and the mean was below the normal limit when IV fluid therapy + $MgSO_4$ were administered (Figure 7.4). With both treatments plasma magnesium concentration fell at the end of fluid therapy ($P<0.01$) and recovered after fluid therapy, but higher values were seen 6 h after the end of treatment with IV fluid therapy + $MgSO_4$ ($P=0.01$) (Figure 7.4).

Discussion

This study confirmed that both enteral fluid therapy and IV fluid therapy plus $MgSO_4$ increase hydration of ingesta and feces, although enteral fluid produced more marked changes in ingesta hydration. This study also supported the hypothesis that enteral fluid therapy can produce

significant hydration of the gastrointestinal contents, with less systemic effects than IV fluid therapy plus MgSO₄. The significant abdominal distention and the more pronounced change in body weight produced by enteral fluid therapy also suggest that a major part of the fluids administered was kept within the gastrointestinal lumen. The trend towards a greater number of defecations during enteral fluid therapy suggests that the gastrocolic response was activated by enteral fluid therapy. Activation of this mechanism in horses receiving fluids by nasogastric tube have been previously suggested (Freeman et al. 1992, Lopes et al. 1999). Although no treatment effect on frequency of urinations could be detected, urine excretion of fluids given intravenously was probably faster due to direct effect on plasma volume. Unfortunately urine volume was not measured, but a more pronounced plasma expansion with IV fluids was suggested by the changes in plasma protein and calcium. The observation of higher values for plasma magnesium with the IV fluid therapy + MgSO₄, may be explained by magnesium absorption. Other authors have documented an increase in plasma magnesium in normal horses (Scarratt and Swecker 1999) and even signs of magnesium toxicosis in dehydrated horses (Henninger and Horst 1997) after nasogastric administration of MgSO₄. The electrolyte imbalances seen in this study seemed to have no consequence since plasma concentration of calcium and magnesium returned to pre-treatment values after the end of treatment and no clinical sign of hypocalcemia or hypomagnesemia was observed. However to better understand the significance of these electrolyte changes it would have been better to measure ionized calcium and magnesium (Rosol et al. 2000). The composition of the electrolyte solution used for enteral fluid therapy seemed appropriate to prevent the marked hypernatremia and hyperchloremia that was reported when large volumes of 0.9% NaCl solution were administered to normal horses (Lopes et al. 2002b). The difference between measured and calculated osmolality of the electrolyte solution for enteral fluid therapy was expected, since the calculation ignores interionic or interparticulate attractions (Murty et al. 1976).

The signs of abdominal discomfort seen in 2 horses at the end of enteral fluid therapy suggest intolerance to the large volumes of fluids administered in a short period of time. No colic had been observed in another study when horses were treated with 10 liters fluids / h for 8 h (Lopes et al. 2002b), but those horses had been fasted for 9 h before fluid therapy, which was not done in this study. The rate of administration was probably too fast for horses having a significant amount of ingesta in the gastrointestinal tract. Alternatively, individual variability for tolerance to enteral fluid therapy could explain the occurrence of abdominal discomfort in this study. Although in both cases abdominal discomfort remitted without treatment, drainage of part of the fluids using the nasogastric tube could have been performed if necessary. As previously reported in horses with impaction (Lopes et al. 1999) and in normal horses (Lopes et al. 2002b), abdominal distention produced by enteral fluid therapy was well tolerated. However horses with conditions such as an extremely large impaction or advanced pregnancy may not tolerate further abdominal distention produced by enteral fluid therapy.

This study demonstrated that both IV fluid therapy plus MgSO₄ and enteral fluid therapy can increase colonic and fecal hydration and produce plasma expansion. More pronounced systemic effects were seen with IV fluid therapy plus MgSO₄, while more pronounced changes in ingesta hydration and abdominal distention were seen with enteral fluid therapy. However even if these differences prove not to be clinically significant, the use of enteral fluid therapy for conditions such as large colon and small colon impaction may be justified based solely on the lower cost. Despite the treatment chosen for large intestine impaction, rational judgment of

clinical condition based on clinical and laboratory findings should be used to adjust fluid composition and rate of administration according to the horse's needs.

Manufacturers' addresses

- ¹ Cargill, Inc. Minneapolis, Minnesota 55440 USA.
- ² ICN Biomedicals, Inc. Aurora, Ohio 44202 USA.
- ³ Church & Dwight Co., Inc. Princeton, NJ 08543 USA.
- ⁴ Precision Systems, Inc. Natick, Maryland 01760 USA.
- ⁵ Abbott Laboratories. North Chicago, Illinois 60064 USA.
- ⁶ Mila International, Inc. Florence, Kentucky 41042 USA.
- ⁷ Springfield Precision Instruments. Wood-Ridge, New Jersey 07075 USA.
- ⁸ Westover Scientific. Woodenville, WA 98072 USA.
- ⁹ Alfa Wassermann, Inc. West Caldwell, New Jersey 07006 USA.
- ¹⁰ Ohaus. Florham Park, New Jersey 07932 USA.
- ¹¹ Precision Scientific. Chicago, Illinois 60647 USA.
- ¹² International Win, Limited. Kennett Square, Pennsylvania 19348 USA.
- ¹³ Nalge Nunc International, Co. Rochester, New York 14602 USA.
- ¹⁴ Bindley Western Industries. Indianapolis, Indiana 46268 USA.
- ¹⁵ SAS Institute, Inc. Cary, North Carolina 27513 USA.

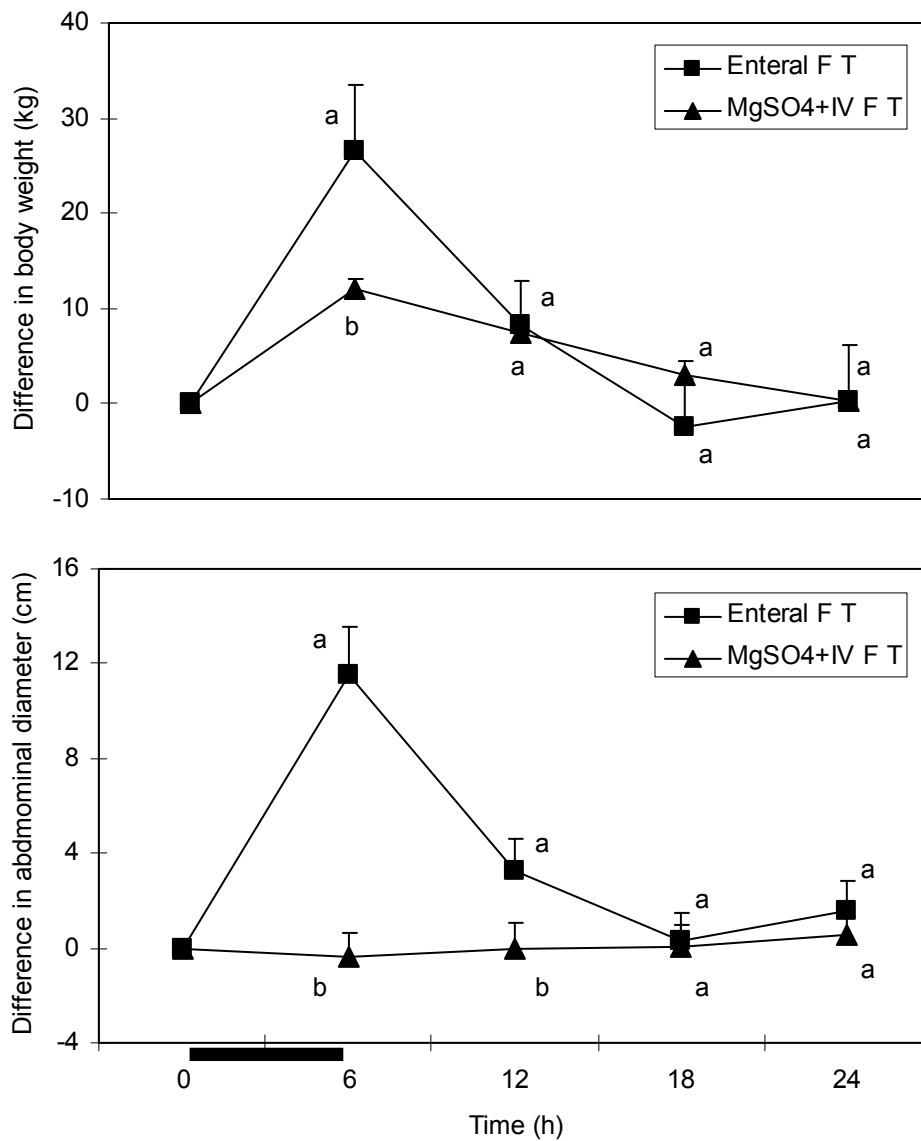


Figure 7.1 – Means of the changes (difference from time 0) in body weight and abdominal circumference of 4 fistulated horses treated with enteral fluid therapy (10 liters/h/6h) or enteral MgSO₄ (1 g/kg) and IV fluid therapy (10 liters/h/6h). The horizontal bar below the graph indicates the time when the horses were receiving fluid therapy. Vertical bars represent the standard errors of the means. Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

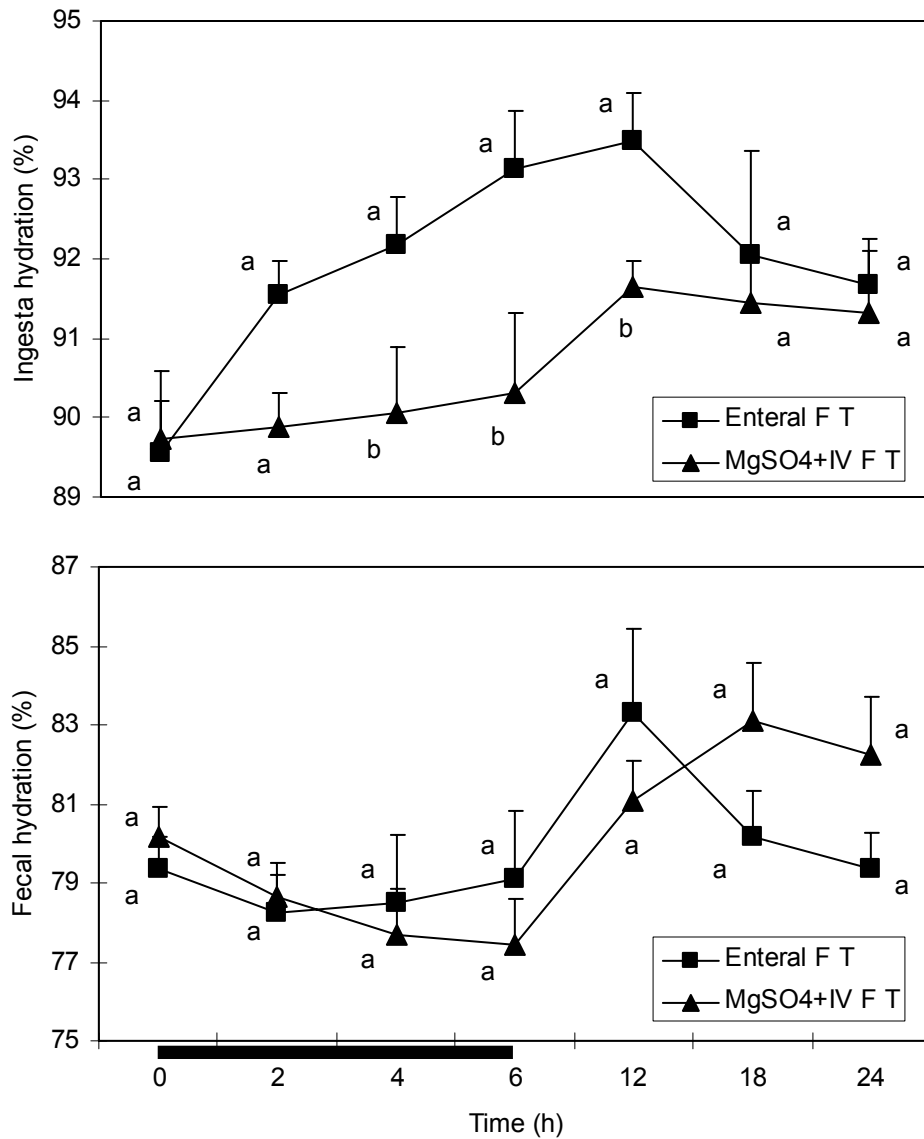


Figure 7.2 – Means of ingesta and fecal hydration of 4 fistulated horses treated with enteral fluid therapy (10 liters/h/6h) or enteral MgSO₄ (1 g/kg) and IV fluid therapy (10 liters/h/6h). The horizontal bar below the graph indicates the time when the horses were receiving fluid therapy. Vertical bars represent the standard errors of the means. Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

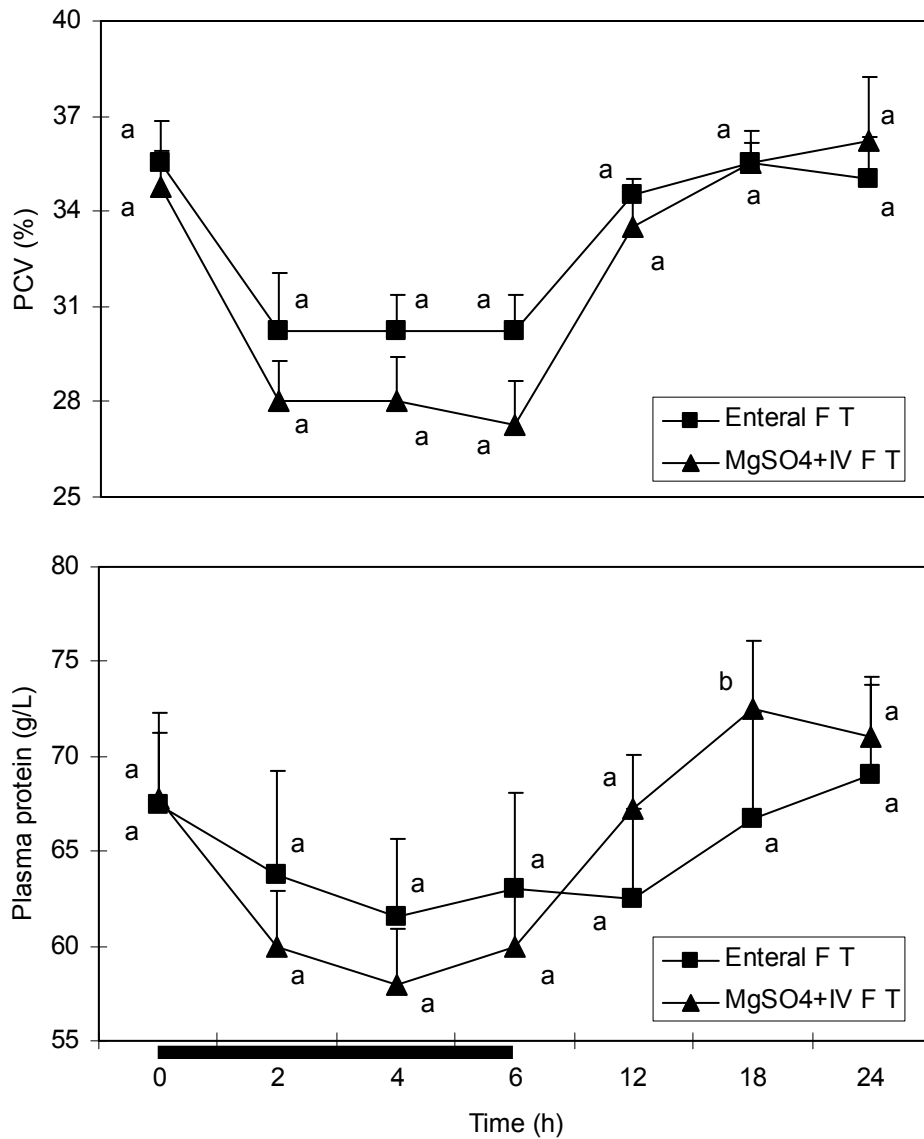


Figure 7.3 – Means of PCV and plasma protein of 4 fistulated horses treated with enteral fluid therapy (10 liters/h/6h) or enteral MgSO₄ (1 g/kg) and IV fluid therapy (10 liters/h/6h). The horizontal bar below the graph indicates the time when the horses were receiving fluid therapy. Vertical bars represent the standard errors of the means. Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons.

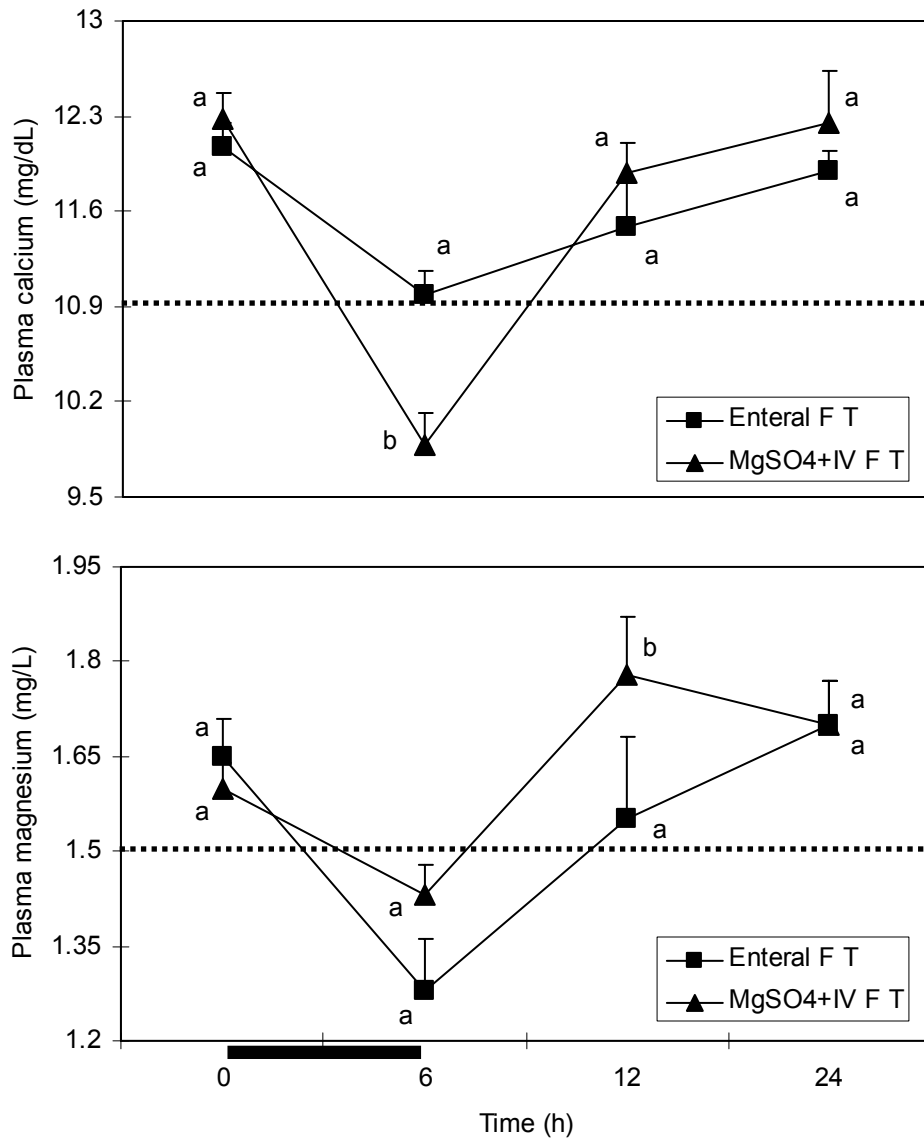


Figure 7.4 – Means of plasma concentration of calcium and magnesium of 4 fistulated horses treated with enteral fluid therapy (10 liters/h/6h) or enteral MgSO₄ (1 g/kg) and IV fluid therapy (10 liters/h/6h). The horizontal bar below the graph indicates the time when the horses were receiving fluid therapy. Vertical bars represent the standard errors of the means. Within one time, means with different letters are significantly different at $\alpha = 0.05$ according to the Bonferroni corrected multiple comparisons. Values above the dotted lines are within normal limits.

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Chapter 8

General Conclusions

1- A large right dorsal colon fistula can be created in horses using a two step surgical technique (1- colopexy and 2- stoma creation and cannula implantation performed 2 to 6 weeks later) for implantation of a specially designed cannula. Due to the relatively small size of the appropriate site for access to the right dorsal colon, precise identification of the surgical site is mandatory. Both surgical procedures can be performed with the horse standing restrained in stocks and treated with sedatives and local anesthetics. Although the cannula design was appropriate it may be better to use a cannula made of a less reactive material (e.g., silicon). The large diameter of the fistula allows large samples of ingesta to be collected without major manipulation, which is essential for studies where frequent sampling is necessary for assessing ingesta hydration.

2- Large grain meals can produce ingesta dehydration and formation of frothy ingesta. It is likely that mechanisms other than the proposed post-prandial activation of the renin-angiotensin-aldosterone system are involved in ingesta dehydration induced by grain ingestion. For the first time formation of frothy ingesta similar to what has been reported in ruminants with frothy bloat was documented in horses. Formation of gassy ingesta may predispose to tympany and large intestine displacements. However it is unlikely that the association between colic and grain ingestion can be explained solely by what had been reported previously and by the findings of this study. Other effects of large meals rich in hydrolyzable carbohydrates on the equine gastrointestinal tract (e.g., water secretion and absorption by the gastrointestinal mucosa, gastrointestinal motility, ingesta flow) need to be investigated.

3- Enteral fluid therapy can be conveniently administered continuously or intermittently using a large bore or small bore nasogastric tube via a coiled line to horses unrestrained in the stall or using a funnel. Enteral fluid therapy can be used in horses to promote ingesta and fecal hydration, as well as to produce plasma expansion, alter plasma electrolyte concentration and promote diuresis. The importance of the electrolyte composition of the solution for enteral fluid therapy can be illustrated by four findings of these studies: 1- enteral administration of plain water produced minimal effects on ingesta hydration and produced hyponatremia; 2- administration of an electrolyte solution with sodium, potassium and chloride concentration similar to plasma was more effective in promoting ingesta hydration than IV fluid therapy, saline cathartics and the combination of fluid therapy administered IV and magnesium sulfate administered through a nasogastric tube; 3- large volumes of this type of electrolyte solution produced minimal changes in plasma electrolytes; 4- administration of 0.9% sodium chloride was the only treatment that consistently produced watery diarrhea but produced hypernatremia and hyperchloremia. Although it is likely that the effects on ingesta and fecal hydration can be maximized by increasing the electrolyte load provided by enteral fluid therapy, this strategy can lead to severe plasma electrolyte imbalances. To establish guidelines for the appropriate fluid composition and rate of infusion for each clinical situation, further studies are necessary.

4- The fact that some horses may be less tolerant to enteral fluid therapy does not outweigh the benefits of this simple and inexpensive treatment. In the absence of a clear contraindication to

enteral fluid therapy (e.g., gastric reflux), this treatment can be started at a low infusion rate while watching for signs of intolerance (e.g., increased heart rate, abdominal discomfort). Gradual change up to the desired rate of infusion should follow. Signs of intolerance do not always mean that enteral fluid therapy is contraindicated, but may be a sign that a slower rate of infusion or temporary interruption of enteral fluid therapy is necessary. In extreme situations analgesics and drainage of gastric content using the nasogastric tube can be used to control pain produced by enteral fluid therapy.

5- At least in normally hydrated horses, intravenous fluid administration (at 5L/h for 12 h) does not increase ingesta or fecal hydration. In other species rapid administration of extremely large volumes of fluids intravenously has been shown to produce net fluid secretion into the gastrointestinal tract. The risk of severe complications and the expense preclude the use of this approach in horses with colic. Furthermore intravenous fluid therapy should not be seen as the only option for fluid administration in sick horses. Indeed it is better to limit the use of IV fluid therapy to situations where enteral fluid therapy is contraindicated or unable to provide the desired effects.

6- The saline laxative sodium sulfate can increase ingesta and fecal hydration, but may produce hypocalcemia and hypernatremia. These findings agree with other reports: a study in horses where this laxative was found to be effective; studies in humans documenting sulfate absorption after oral administration of this laxative and a decrease in plasma calcium concentration after IV administration of sodium sulfate. On the other hand magnesium sulfate alone does not change ingesta hydration or cause plasma electrolytes imbalances. Due to the differences in the molecular composition of these salts and due to the difference in hydration of the commercial products used, in this study the amount of electrolytes provided when sodium sulfate was administered was about twice than when magnesium sulfate was administered. Thus, similarly to what was observed with enteral fluid therapy, the amount of electrolytes provided may be a major determinant of the effects of saline cathartics. However high electrolyte loads may lead to severe plasma electrolyte imbalances.

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