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Take Home Message—Gastrointestinal conditions may be acute or chronic, painful or seemingly benign, have no systemic effects or be responsible for profound losses in condition or performance potential. When acute, establishing a diagnosis, preserving life and controlling pain are first and foremost. When chronic, the management plan may be difficult to formulate and achieve only intermittent success. Clinical findings should be interpreted in relation to the historical information available. Confirming a definitive diagnosis before action is required may not be obtainable.

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I. INTRODUCTION

Acute or chronic gastrointestinal pathology is a common and potentially life-threatening affliction of the horse, with the majority of horses suffering from one of these conditions sometime during their lives. The majority of gastrointestinal conditions are amenable to medical management and in some cases this may be chronic in nature, however acutely painful conditions may indicate an intestinal accident is present necessitating surgical intervention.

II. COLIC

The degree of pain shown during a gastrointestinal condition will largely determine the approach to diagnosis and management. The presence of intractable abdominal pain necessitates surgical exploration.

History

Signalment: Geriatric horses may have a similar admission cardiovascular status compared to mature horses (heart rate, packed cell volume, plasma creatinine and blood lactate concentration) but a more serious cause of colic underlying the episode. Increased occurrence of enteroliths in Arabians and miniature breeds has been demonstrated.

Management changes and potential triggers: transport, administration of medications for concurrent conditions (antimicrobials, anti-inflammatories), and changes in social structure by the addition or removal of other horses may precipitate colic. More frequently, recent feed or water changes may be incriminated.

Pattern of colic episode(s): How often and over what time period has the colic been apparent (isolated episode versus chronic intermittent over months)? Are episodes similar or signs random? Is there a temporal relationship to estrous cyclic activity (actual or perceived)?

Fecal production: Decrease in volume or frequency of fecal production is suggestive of decreased dietary intake (inappetence, inability to prehend food, competition), or prolonged intestinal passage (motility disorders, obstructive processes, increased fecal density).

Pain

Duration: The duration of signs of colic prior to evaluation has been associated with survival.

Severity and frequency: In many prognostic models, the degree of pain affects survival. Horses vary in their pain responses, so it is useful to know if this episode is consistent with other colic episodes (is it more or less severe?)

Response to analgesics: What is the duration and completeness of response to analgesics? Alpha-2 adrenergic agonists have a rapid onset and may provide visceral analgesia for prolonged periods. Flunixin meglumine has been shown to provide less visceral analgesia.

Clinical Examination

In one referral center colic study, there was a significant association between predicted survival and outcome based on clinical impression, and this correlation improved with increased case exposure.

Vital signs: Rectal temperature is widely variable, ranging from elevated with acute infectious involvement to hypothermic in the presence of severe hypovolemia or devitalized bowel. Cardiovascular parameters are significant predictors of mortality in multiple studies. Heart rate is variably elevated (individual pain tolerance) and alone cannot be relied upon to predict outcome. Hypovolemia may be responsible (are signs of circulatory compromise present?). Paradoxically, a normal heart rate may be present with significant gastrointestinal compromise. Pulse quality can be assessed peripherally at the distal extremities or the facial artery along the mandible. Poor pulse pressure is suggestive of endotoxemia before decompensation occurs. Respiratory rate and effort
when elevated may indicate pain, acid-base disturbances, physical impediment to the diaphragmatic excursion due to visceral dilation or pleural space disease (pleural pain can mimic gastrointestinal pain).

Mucous membranes: Color is anecdotally considered a reliable prognostic indicator, with some\textsuperscript{8,12} but not all retrospective studies agreeing.\textsuperscript{3} Capillary refill time was not shown a reliable indicator of the need for medical or surgical management in one study.\textsuperscript{13} However, a shorter capillary refill time was associated with increased likelihood of survival.\textsuperscript{12,14}

Abdominal size: Abdominal distension, rectal findings and peritoneal fluid color were the most discriminating deciding variables between medical and surgical management in one study.\textsuperscript{15} In another study, rectal examination findings, abdominal fluid composition, presence of intractable pain, and abdominal distention were most likely to differentiate between medical and surgical lesions.\textsuperscript{16}

Abdominal sounds: Increased gut activity is seen in conditions which irritate the intestinal tract. Decreased or absent gut activity may indicate a more serious situation, with decreased fecal production and signs of acute pain indicating a less favorable prognosis. Decreased borborygmi may also indicate sudden feed changes, carbohydrate overload, or infectious agents.

Oral examination: Decreased frequency of dental examinations and treatments are associated with increased risk of colonic distention and impaction.\textsuperscript{17}

Trauma signs: Duration and severity of unobserved pain may be indicated by skin abrasions, musculoskeletal injuries and damage to housing.

Nasogastric Intubation

Character and amount of nasogastric reflux, and the response of the horse to passage of the fluid yields valuable information. Amount, timing relative to the occurrence of colic, and character of reflux should be noted. A small intestinal problem is implied by the presence of significant amounts of fluid. When obstructed, the proximal small intestine yields a high volume of reflux. Duodenitis-proximal jejunitis (anterior enteritis) yields malodorous, sometimes hemorrhagic fluid. Lower small intestinal lesions are much less likely to reflux initially. Physical obstructions yield relatively fresh feed and intestinal fluids. Colonic distension may cause reflux by duodenal compression. Small intestinal ileus will experience a relief of pain and a decrease in heart rate with gastric decompression. Physical obstructive lesions are most likely to have no response to successful reflux.

Rectal Examination

Intestinal distension and disposition prompts categorization of diagnosis.\textsuperscript{10,18} Rectal examination has been reported as the single most useful pre-surgical diagnostic tool, allowing definitive diagnosis of many large intestinal conditions and providing non-specific information about small intestinal disease. Serosal surfaces are able to be felt and character assessed for signs of peritonitis. Fecal presence or absence and consistency can be evaluated. The spleen, left kidney and uterus can be assessed.

Concurrent Conditions

Fecal consistency gives insight to nutritional imbalances, the presence of infectious enteric agents, or changes in fecal density. Infectious and inflammatory conditions within the peritoneal cavity can produce signs consistent with an intestinal lesion. Pain similar to a colic episode can result from rhabdomyolysis, pneumonia, pleuritis, nphritis, nephrolithiasis, and cholestasis.

Ancillary Aids

Ultrasoundography: Rapid assessment of intestinal wall thickness, diameter, content and motility, stomach size, quantity and nature of peritoneal fluid, and position of the viscera and intestinal tract is possible. Comparing surgical and necropsy findings, ultrasonographic detection of abnormal small intestine that lacked motility was highly sensitive and specific, with high positive and negative predictive values for small-intestine strangulation.\textsuperscript{19} With large colon lesions, imaging via a ventral abdominal window was moderately sensitive and highly specific for diagnosis of large-colon torsion.\textsuperscript{20}

Peritoneal fluid: In the field, it is challenging to gain all the information contained in a sample in a timely fashion. Gross appearance is therefore the most valuable indicator of the presence of devitalized gut and is most likely to aid the clinician in the determination of the need for surgery. Normal fluid is clear and pale yellow-straw-colored. It does not clot in a plain tube. Yellow fluid that is slightly turbid suggests a medical colic, with dehydration or elevated bilirubin. Pink/orange fluid indicates hemolysis or hemorrhage. The presence of hemolysis increases the likelihood of the need for surgical correction.\textsuperscript{15} Hemolysis may be distinguished from iatrogenic hemorrhage by centrifugation or allowing time to settle as iatrogenic hemorrhage will form a pellet of red cells with a clear supernatant. Compromised vascular supply to the gut, however, will result in fluid that does not settle but instead remains uniformly pink/orange. Bloody to brown fluid indicates advanced ischemia, and the presence of ingesta indicates rupture or enterocentesis has occurred. Increased protein concentrations occur with peritonitis and surgical colic where inflammation and vascular compromise of the gut has occurred. Protein can be readily assessed with a hand-held refractometer. Volume of peritoneal fluid at the time of sample collection can be suggested by the flow rate: since fluid can pocket in the peritoneal cavity this is an unreliable indicator. Serial evaluation of abdominal fluid color and specific gravity has a high positive predictive value for type of intestinal lesion,\textsuperscript{21} whereas patient age and abdominal fluid color has a high positive predictive value for clinical outcome.\textsuperscript{21} The presence of gut content or urine may be
indicated by smell, subsequently peritonitis and presence of bacteria may be suggested.

Gross fluid analysis complements but does not replace information gained during a clinical examination and this must be communicated to the client. More importantly, changes in the peritoneal fluid may considerably lag the gastrointestinal or peritoneal pathology it is considered to reflect.

Regardless of the clinical examination findings other indications for surgery, especially unremitting pain, may become apparent before laboratory data is available. In these cases, referral for further evaluation or surgery should be undertaken immediately to expedite management of the case.

**Management**

Control of abdominal pain is often the most pressing issue in the management of colic as this allows safe examination, provides patient relief and provides both diagnostic and prognostic information (Table 1). Fluid and electrolyte therapy, laxatives and transition back to feeding following a period of fasting are commonly practiced.

**Recurrent Colic Episodes**

Causes of recurrent colic are many and diverse, and they provide a considerable challenge to diagnose and manage. Differential diagnoses include small intestinal stenosis or partial obstructions, ileal hypertrophy, large intestinal obstructions, neoplasia, parasitism, gastroduodenal ulceration and gastric distension. Peritoneal cavity problems that cause recurrent abdominal pain include abscessation and adhesions. Intermittent pain may result from disease of other intraabdominal organs – liver or the urogenital system. Long-term dietary control may be necessary in situations where there is no surgical correction or readily apparent medical management. In these situations, a decrease in fecal bulk, interval feeding, use of a complete ration, removal or addition of extra roughage, hind gut acid suppression, gastric ulcer treatment and prophylaxis, prokinetics where motility disturbances are confirmed, probiotics, parasite control, and scrupulous dental care are all components of a comprehensive candidate management program. When an inflammatory or infiltrative process is suspected or confirmed by diagnostic testing, chronic treatment with corticosteroids may be required.

**III. DIARRHEA**

With the exception of the typical ‘foal heat’ diarrhea, this condition is an indication for animal isolation. A diarrheic horse should be considered infectious and contagious until proven otherwise. While troublesome to take precautionary measures, the expense and potential for animal losses from a diarrhea outbreak outweighs the inconvenience of heightened hygiene measures and segregation of farm animals.

**Pathophysiology**

Defense mechanisms of the equine digestive tract include gastric acidity, a robust commensal intestinal flora, coordinated peristalsis, protection of cell surface binding sites by intestinal mucus, and inhibitors such as lactoferrin. Different mechanisms are involved in the development of diarrhea; these include hypersecretion, increased permeability (exudation), malabsorption, osmotic draw and abnormal motility.

Patients with severe acute diarrhea may show signs of an active inflammatory process centered in the large colon. Following disturbance of the cecocolic flora, bacterial toxins may be released (lipopolysaccharide, LPS) which promote inflammation via cytokines and other inflammatory mediators. This results in a net fluid loss into the intestinal lumen, with damage to the cecocolic mucosa and vascular supply. Edema, exudation, and toxin absorption into the bloodstream can then readily occur. As a result, fluid, electrolyte, protein, anticoagulant, and procoagulant substances are lost to the gut lumen. Hypovolemia, hypoalbuminemia, coagulopathy, acidemia, and shock (distributive and endotoxic) ensue.

There are many causes of acute diarrhea. Infectious etiologies include Salmonellosis, Clostridium difficile, C. perfringens, and Potomac Horse Fever (PHF). In younger horses, viral causes are possible. Non-infectious causes include antibiotic-associated diarrhea (AAD) and nonsteroidal anti-inflammatory (NSAID)–induced right dorsal colitis. In approximately 60% of acute diarrhea cases, the originating cause is never found. The underlying lesion is a disturbance of the normal fluid and electrolyte balance. As the absorptive capacity of the large colon is high, small intestinal disturbances do not usually cause diarrhea in adult horses as compared with other species.

**Infectious Agents**

**Bacteria**

*Salmonella*

Once *Salmonella* has overcome the host defense mechanisms bacteria are able to migrate through the enterocytes and reach the lamina propria where they stimulate an inflammatory response. Lymphatic spread to regional lymph nodes occurs, promoting further inflammation. Distribution of *Salmonella* organisms to the systemic circulation may occur via efferent lymphatics. In highly concentrated populations of horses, *Salmonella* is of particular concern. Stressors such as dietary changes, surgery, transport, heat exposure and concurrent illness increase the likelihood of infection.22-29

Clinical signs of salmonellosis are variable and can range from mild enteritis to severe septicemic shock. Intestinal fluid loss occurs due to active fluid loss through hypersecretion and passive fluid loss by malabsorption due to profound inflammation. Diagnosis of *Salmonella* is demonstrated by positive fecal or blood cultures. Treatment for *Salmonella* infection is nonspecific and is aimed at maintaining hydration and electrolyte balance. In neonates and younger foals, systemic antimicrobials are necessary to counter the potential for bacteremia and infection of physeal and synovial structures.
### Table 1. Drug Dosages

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose rate</th>
<th>Indication</th>
<th>Author comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Motility agents</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Parasympathomimetics</td>
<td></td>
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</tr>
<tr>
<td>Bethanechol chloride</td>
<td>0.025 mg/kg SQ q8h</td>
<td>Gastric and proximal small intestinal motility disorders.</td>
<td>Improves gastric emptying and small intestinal motility. May cause abdominal pain and hypersecretion in some horses (salivation, diarrhea). Author has found bethanechol useful in gastric emptying disorders (gastroduodenal ulcer disease in foals) and small intestinal motility disorders (with yohimbine when tolerated).</td>
</tr>
<tr>
<td>Neostigmine methylsulfate</td>
<td>0.01-0.02 mg/kg SQ q4h</td>
<td>Large intestinal motility disorder, pelvic flexure impactions. Avoid use in small intestinal disorders.</td>
<td>Suggested to improve motility of the pelvic flexure, cecum and areas of the colon. May disrupt gastric emptying and has variable effects on jejunum experimentally. Colic signs may worsen as contractility is stimulated in the area of the impaction. Author has not found this to be a predictable agent.</td>
</tr>
<tr>
<td><strong>Sympatholytics (alpha-2 adrenergic antagonists)</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Yohimbine hydrochloride</td>
<td>0.075-0.25 mg/kg IV q8h</td>
<td>Ileus</td>
<td>Suggested experimentally to shorten gastrointestinal transit time. Author uses in combination with bethanechol.</td>
</tr>
<tr>
<td><strong>Benzamides</strong></td>
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<td></td>
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<tr>
<td>Metoclopramide</td>
<td>0.04 mg/kg/h as CRI 0.25 mg/kg SQ or IV over 30 minutes q6h</td>
<td>Gastric emptying disorders and lack of motility at all levels of the intestinal tract.</td>
<td>Potent prokinetic agent. Potential for untoward neurological effects as a dopamine antagonist. CRI has most efficacy and least potential for side effects. Rapid metabolism means best administered as CRI. Author recommends close monitoring during usage and immediate cessation on first signs of neurological problems: administer diphenhydramine.</td>
</tr>
<tr>
<td>Cisapride</td>
<td>0.5 mg/kg PO q8h</td>
<td>Gastric emptying disorders, stimulates majority of gastrointestinal tract.</td>
<td>Unlikely to cause central nervous system side effects (compared with metoclopramide).</td>
</tr>
<tr>
<td>Lidocaine hydrochloride</td>
<td>1.3 mg/kg IV slow loading dose then 0.05 mg/kg/h IV CRI</td>
<td>Ileus, enteritis, low grade continuous pain</td>
<td>Very useful and predictable analgesia for enteritis/colic patients. Mode of action controversial: anti-inflammatory effect proven, in vitro studies do not show direct prokinetic effect.</td>
</tr>
<tr>
<td><strong>Analgesics</strong></td>
<td></td>
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<tr>
<td>Sympathomimetics (alpha-2 adrenergic agonists)</td>
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<tr>
<td>Xylazine hydrochloride</td>
<td>0.3-0.5 mg/kg IV PRN</td>
<td>Acute abdominal pain secondary to intestinal distension,</td>
<td>Suitable for control of pain during initial work-up as analgesic effects last up to 30 minutes. Author prefers dose range 0.3-</td>
</tr>
</tbody>
</table>
### Detomidine hydrochloride

- **Dosage:** 0.02-0.04 mg/kg IV PRN
- **Effect:** Acute abdominal pain secondary to intestinal distension, displacement or strangulation. Relaxation of gut at site of impaction.
- **Additional Notes:** More profound and long lasting analgesia than xylazine, with sedation lasting longer than analgesia. Author considers detomidine a more potent analgesic than flunixin, and when used in combination with butorphanol the most potent analgesic combination available for the acute abdomen patient.

### Ketamine hydrochloride

- **Dosage:** 0.3 mg/kg IV PRN
- **Effect:** ‘Ketamine stun’ – facilitates examination in situations of intractable abdominal pain.
- **Additional Notes:** Useful where other medications have failed to provide sufficient analgesia to establish a diagnosis and provide emergency care to the colicking horse. May provide up to 15 minutes of relief depending on causative lesion. The author considers this technique to provide excellent analgesia and aids surgical decision making.

### Hyoscine-N-butylbromide (Buscopan®)

- **Dosage:** 0.3 mg/kg IV PRN
- **Effect:** Spasmodic and flatulent colic signs.
- **Additional Notes:** May elevate heart rate, therefore this cannot be used as a valid indicator of severity of pain for 30 minutes following IV injection. Will not mask increase in pain associated with progression of disease. Author has found drug useful for passage of nasogastric tube in cases of severe gastric distension, esophageal obstruction and distal intestinal obstructions in foals. Useful during initial examination of acute abdomen patient to allow examination (after heart rate taken), duration of effect useful gauge of severity of causative lesion.

### Butorphanol tartate

- **Dosage:** 0.02-0.05 mg/kg IV or IM PRN
- **Effect:** Acute abdominal pain of any cause.
- **Additional Notes:** A useful agent alone or as an adjunct to alpha-2 adrenergic agonists. May induce profound sedation in addition to analgesia.

### Cimetidine HCl

- **Dosage:** 6.6mg/kg IV q8h, 20mg/kg PO q8h
- **Effect:** Reduction of gastric acid production. Prophylaxis or treatment of gastric ulceration.

### Ranitidine HCl

- **Dosage:** 2mg/kg IV q8h, 6.6mg/kg PO q8h
- **Effect:** Reduction of gastric acid production. Prophylaxis or treatment of gastric ulceration.

### Omeprazole

- **Dosage:** 4 mg/kg PO q24h
- **Effect:** Reduction of gastric acid production. Prophylaxis or treatment of gastric ulceration.
### Intestinal Clostridiosis

These gram-positive organisms can be found in the intestinal tracts of domestic animals and are widely distributed throughout the environment, including the soil. An obligate anaerobe, the vegetative form perishes under aerobic conditions; however, it can produce endospores which are capable of surviving under adverse environmental conditions in soil and water. They produce potent exotoxins that are responsible for a variety of intestinal diseases in domestic animals. The common causative agents of clinical disease are *Clostridium perfringens* biotypes A and C and *Clostridium difficile*. While most studies suggest that biotypes A and then C are the most important, there are reports of biotypes A through E being associated with enteric disease of foals. Diarrhea induced by *Clostridium sp.* is recognized more commonly during the early neonatal period. Disease induced by *C. perfringens* biotype C is associated with abdominal distention, colic, circulatory shock, hemorrhagic diarrhea and high mortality. The syndrome is commonly seen within the first 48 hours of life. Most often it occurs in vigorous foals with high levels of milk ingestion. Diagnosis is confirmed by identifying toxin within the feces or intestinal luminal content, and recovery of the organism and biotyping by polymerase chain reaction (PCR) for toxin gene identification is of use. Treatment is often unrewarding in established cases. Antimicrobial agents (potassium or sodium penicillin and metronidazole), NSAID, plasma, correction of fluid and electrolyte derangements, and *C. perfringens* biotype C antitoxin are indicated. Total parenteral nutrition allows nutritional support without worsening diarrhea. Probiotics may be useful as both treatment for the affected and as a preventative for other newborn foals on the property. Enteric disease associated with *C. perfringens* biotype A has been seen in newborn foals, with transient and variable clinical presentation. This biotype is however present in the feces of healthy young foals. Likewise, the role of *C. difficile* in juvenile diarrhea is not clear. Prevalence varies with geographic location but *C. difficile* appears to be a rare isolate in older suckling foals. Treatment is as above for *C. perfringens* without specific antitoxin.

Samples should be collected and shipped in an appropriate container. The isolation of *C. difficile* is usually considered significant in foals of all ages but it is not uncommon to identify foals that are culture positive but toxin-negative. Recovery of *C. perfringens* from diarrheic foals is also of questionable significance because the organism, particularly *C. perfringens* biotype A, is commonly present in the feces of healthy foals.

### Lawsonia

Equine proliferative enteropathy (EPE) is a fecal-oral transmissible enteric disease caused by *Lawsonia intracellularis*. The disease has a sporadic distribution, but has been reported in apparent outbreaks. Equine proliferative enteropathy predominantly affects weanling foals between the ages of three and six months although yearling and older cases occur. Depression, fever, weight loss, colic, diarrhea, and ventral edema are characteristic signs. Clinical pathology findings include leukocytosis, anemia, mild to severe hypoalbuminemia, and variably hyperfibrinogenemia. Hyponatremia, hypokalemia, hypocalcemia, and metabolic acidosis are present. Thickened small intestinal wall is typically noted on abdominal ultrasonography. In severe cases, the entire small intestine is affected with apparent large colon involvement.

Antemortem diagnosis of EPE depends on a combination of characteristic clinical findings and serological testing for specific antibodies to *L. intracellularis* and PCR detection in feces. Gross pathologic lesions in EPE are characteristic. Mucosal hypertrophy of the ileum and terminal jejunum, sometimes involving the entire small intestine is present. Histologic examination with Warthin-Starry silver staining or immunohistochemistry is positive for EPE. The presence of small intestinal hyperplasia with curved intracellular bacteria in the apical cytoplasm of crypt cells is confirmatory.

Treatment entails correction of fluid and electrolyte deficits, with a combination of crystalloid and colloid therapy, and sometimes intense supportive care is necessary for recovery. Anti-inflammatory, antidiarrheal, antiulcer therapy and pain management are required. A number of antimicrobials have proven effective in this condition including macrolides, chloramphenicol, oxytetracycline and doxycycline. Early administration of plasma or hetastarch is beneficial as foals usually become profoundly hypoalbuminemic. This hypoalbuminemia may increase plasma levels of oxytetracycline (further compromising renal function).

### Potomac Horse Fever (Neorickettsia risticii)

Equine monocytic ehrlichiosis is infectious but not contagious. It causes fever and colitis in horses. *Neorickettsia risticii*...
Enterococcus durans

Enterococcus durans has been isolated from diarrhea cases in many species. It is commonly isolated from the feces of young foals with diarrhea in association with other potential diarrhea pathogens. The organism is found to colonize the small intestinal mucosa and it was associated with moderate pathology. Diarrhea is likely to be age dependent in its severity.

Viruses

Rotavirus

The virus invades the intestinal epithelium on the lateral sides and the tips of the villi affecting the brush border epithelium, responsible for small intestinal degradation of disaccharides to monosaccharides for absorption. Loss of the brush border results in decreased lactase which results in lactose maldigestion. Lactose remains in the intestinal lumen, osmotically drawing fluid. Bacteria in the large intestine subsequently ferment the lactose to volatile fatty acids, increasing osmotic draw of the colonic content.

Clinical signs of disease occur most often in young foals, usually 3 months of age or less. Diarrhea, abdominal distension, colic, depression and anorexia occur. Rotavirus is reported as the most common cause of diarrhea in foals in a Kentucky study. Infected foals may shed rotavirus for up to 10 days. This may continue asymptptomatically up to 8 months following infection. The virus is environmentally persistent for 9 months.

Diagnosis requires detection of the virus in feces by ELISA, latex agglutination or electron microscopy. Treatment is generally empirical and symptomatic.

Protozoa

Cryptosporidia

The role of cryptosporidium in foal diarrhea remains controversial as infection appears widespread. Cryptosporidium has been implicated in the death of foals. However, in one study the number of foals with diarrhea or soft feces was not significantly different between positive and negative foals. C. parvum should be considered a zoonosis.

Detection of oocysts in fecal samples requires acid-fast staining, immunofluorescence assays, or flow cytometry. As considerable expertise is required to detect the small oocysts, submission of fecal samples to a laboratory should specifically state that detection of cryptosporidium is desired. Treatment is generally supportive and centers on fluid and electrolyte replacement. Prevention includes environmental disinfection and isolation of infected foals.

Giardia

Prevalence is considered to be as high as 35% but data associating shedding with disease are lacking. Reports of cases of suckling foals with diarrhea and high Giardia counts responding to a short course of metronidazole exist.

Parasitic

Strongyles are most often incriminated in cases of acute or chronic enterocolitis. Large and small strongyles are involved. Historically, large strongyle infections were considered an important source of colic and chronic diarrhea. However, with widespread use of effective anthelmintics, small strongyles have become a significant cause of diarrhea (acute and chronic) along with an association with colic signs.

Diagnosis of a prepatent infection is challenging, with potentially lethal burdens of encysted small strongyles possible.

IV. IATROGENIC

Disturbances of colonic flora – antimicrobial associated diarrhea

The development of C. difficile diarrhea has been associated with usage of several antibiotics. These include β-lactam antibiotics, gentamicin, potentiated sulfonamides and erythromycin. In these cases the risk factors for C. difficile-associated diarrhea are similar to those discussed for Salmonellosis including alterations in cecocolic microflora that allow colonization, proliferation, and toxin production. This organism is more frequently isolated from stud farm soil samples than farms with only mature horses. Clostridium difficile was also found in the feces of healthy horses and foals, implying healthy foals may function as a potential reservoir. At least five toxins have been identified. Toxin A is an enterotoxin which causes intestinal fluid accumulation and is pro-inflammatory. Toxin B is a potent cytotoxin. These toxins act synergistically to cause intestinal disruption. Both toxin genes are present in the majority of C. difficile isolates.
from horses with acute enteric disease; however, variations have been discovered.  

The relationship between antimicrobials and diarrhea is complex and inconsistent. In many clinical cases, it is difficult to establish a pathogen and the antibiotic as the linked causative factors in disease. Theoretically, any broad-spectrum antibiotic has the potential to upset the local protective flora and to allow potential pathogens to "overgrow" and cause disease. Contrary to some opinions, parenteral antimicrobials can cause diarrhea and favor the overgrowth of pathogenic organisms. Major causative factors in human cases have been identified as a loss of colonization resistance through alterations in the gastrointestinal microflora, altered colonic fermentation, and increased toxin production by overgrowing pathogenic organisms. Most classes of antibiotics have been implicated in human antimicrobial-associated diarrhea; however, cephalosporins, penicillins, and clindamycin have been implicated in horses. 

Right Dorsal Colitis – NSAID usage 

Two syndromes can result from NSAID usage: generalized NSAID toxicity and right dorsal colitis (RDC). All NSAIDs have the potential to cause toxicity. Localized ulcerative inflammation of the right dorsal colon has been associated with NSAIDs given in excessive amounts, or in the presence of dehydration. Clinical signs of RDC include fever, depression, inappetence, colic, diarrhea, dehydration, and evidence of endotoxia. Oral ulceration and peripheral edema may occur in severe toxicities. Those NSAIDs that indiscriminately block COX-1 and COX-2 have increased capacity to cause toxicity. The gastrointestinal lesions can result in mucosal ulceration, protein-losing enteropathy, gastrointestinal bleeding, and absorption of microbial toxins across the damaged colonic wall with systemic effects. 

Treatment of Diarrhea 

Acute Diarrhea 

Acute diarrhea tends to be rapid in onset and self-limiting in uncomplicated cases. The main goal of any treatment plan is to re-establish homeostasis, as fluid, electrolyte and protein losses can be substantial. Hypovolemia, circulatory shock and endotoxin absorption are possible. Fluid replacement is calculated by body weight multiplied by the estimated percentage of dehydration (Table 2). Depending on the status of the patient, this volume can be administered relatively rapidly (10-20 mL/kg/h). Shock doses of isotonic crystalloids can be administered up to 50 mL/kg/h, with higher rates possible when close monitoring is available and neither cardiovascular nor pulmonary concerns exist. Hypertonic saline 7.5% can be given (5–10 mL/kg over 20 minutes) for initial resuscitation during hypovolemia, but care should be exercised in colitis cases with severe hyponatremia. This should be followed as soon as practical with the administration of isotonic solutions, the required volume being the calculated loss plus daily maintenance requirements (up to 5% of bodyweight). Estimated ongoing losses must also be supplied. 

Table 2. Fluid Doses and Rates for Stabilization and Maintenance 

<table>
<thead>
<tr>
<th>Fluid Doses and Rates for Stabilization and Maintenance</th>
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<tbody>
<tr>
<td>Replacement Fluid Volume (Liters) = Body weight (kg) x % Dehydration.</td>
</tr>
<tr>
<td>Example: 500 kg horse x 0.05 (5%) Dehydration = 25 liters (this is the minimum dehydration that can be detected).</td>
</tr>
<tr>
<td>Rate of Replacement Fluid Administration: 10 – 20 mL/kg/hr.</td>
</tr>
<tr>
<td>Example: 500 kg horse at 10 ml/kg/hr = 5 L/hr until deficit is replaced (can safely double this rate, shock doses are possible).</td>
</tr>
<tr>
<td>Maintenance Fluid Volume = 50 mL/kg/24 hrs (adult), 100 mL/kg/24 hrs (neonate).</td>
</tr>
<tr>
<td>Example: 500 Kg horse x 50 ml/kg/24 hrs = 25 L per day (ongoing losses and insensible losses must be added).</td>
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</table>

Colloids can be used to maintain the fluid in the vascular space. Plasma supplies albumin for oncotic pressure and drug carriage, along with antithrombin and immunoglobulins. Hetastarch is less expensive, has only oncotic properties, and cannot be measured by refractometer. The author prefers to give both products in tandem. Sodium bicarbonate may be used in severe metabolic acidosis not corrected with volume expansion. Half the calculated dose is given slowly intravenously over 20 minutes, and the rest of the dose in crystalloid fluids over 4 hours. Oral supplementation is also possible. Hypokalemia can be managed by addition of KCl to intravenous replacement fluids (20-40 mEq/L) and can be administered safely if the rate of administration does not exceed 0.5 mEq/kg/h. KCl can also be administered orally for several days. Flunixin meglumine, ketoprofen, or firocoxib are appropriate choices for antiendotoxic effects and analgesia. Aspirin is also reported to prevent thrombus formation. Inhibition of the vasodilator prostaglandins occurs with some NSAIDs, therefore care must be exercised with usage with regard to renal damage. 

Hyperimmune antisera or plasma is used in endotoxia. Gram-negative bacteria vary widely; however, they share common core antigens, therefore therapeutic antibodies are aimed at the commonly held LPS core. Some studies have failed to show positive results. The use of antibiotics in treating acute enterocolitis is controversial; however, in severely neutropenic patients, broad-spectrum antimicrobials can be considered rational. Metronidazole is indicated in confirmed C. difficile cases. In cases of Salmonella, antimicrobial usage is controversial. Foals with salmonellosis are routinely treated with...
antimicrobial drugs, although adults are not. Reasons for using antimicrobials include removal of Salmonella sp. and prevention of spread to other tissues. However, some Salmonella sp. are highly resistant and antimicrobials can result in additional endotoxin release, and further derangement of the colonic flora. In one study, usage of antimicrobials was associated with an increased risk of death in hospitalized acute diarrhea cases.\(^{51}\)

**Chronic Diarrhea**

Chronic diarrhea may be progressive in onset, accompany a concurrent debilitating disease or be an extension of acute intestinal inflammation and scarring resulting in a disease process of extended duration. Colonic dysfunction is always present as the colon is the major organ responsible for water reabsorption.\(^{52}\) Conditions associated with chronic diarrhea and weight loss include dental conditions; esophageal conditions; gastric ulceration; malabsorption and maldigestion as a result of infectious, infiltrative and neoplastic disorders; sand enteropathy; motility disturbances; parasitism and abdominal abscessation.\(^{52,53}\) Where hypoproteinemia is present, small intestinal malabsorption may be investigated by D-xylose or glucose absorption testing.\(^{52-54}\) Glucose has the advantage of rapid availability of results and no specialized equipment is required.\(^{55}\)

Parasitic and infectious (if identified) causes are treated specifically. With larval cyathostomiasis intensive supportive therapy may be required in addition to repeated administration of appropriate anthelmintics. Both moxidectin and daily treatments of fenbendazole have been shown highly successful.

Iodochlorhydroxyquin has long been recommended for diarrhea treatment, with many horses having an improvement in consistency within days of initiating treatment, some having worsening of diarrhea, and others reverting to diarrhea once the drug is discontinued.

Many horses with chronic diarrhea do not have a readily achievable diagnosis. It is considered that the causative lesion in these horses is a disturbance of the normal colonic flora. Treatment often is unsuccessful with relapses common, most often following discontinuing treatments. Antimicrobial usage has been reported useful in individual cases.\(^{55,56}\) Only anecdotal evidence exists for supportive treatments including colonic and fecal transfaunation, and conflicting information exists regarding probiotics and decreasing roughage to minimize colonic fermentation. The presence of chronic infiltrative and/or inflammatory disease (histological diagnosis) associated with diarrhea is an unfavorable prognostic indicator.

V. **MOTILITY DISORDERS**

Post-operative ileus and gastric emptying disorders are challenging conditions to manage. Ileus may respond to supportive therapy alone, or may require intensive medical management to overcome. Gastric dilation is a serious problem and rupture can result.

**Motility Agents**

Adynamic ileus is a well-recognized condition that complicates case management; however, the mechanism by which it occurs is subject to controversy. Research into intestinal motility has included *in vitro* and *in vivo* mechanical studies, measurement of myoelectrical activity, transit time of nonabsorbable markers, and surgical harvesting of intestinal musculature. Difficulties have arisen in the extrapolation of findings in non-diseased to diseased horses.

Studies have shown that a relationship appears to exist between inflammation and the onset of ileus, with sufficient inflammation in the muscle layers and myenteric plexus disrupting motility. Another proposed mechanism is increased sympathetic stimulation (alpha-2 receptors) and norepinephrine-mediated inhibition of ACh release. Both inflammation and the listed receptors are therefore attractive targets for intervention.

**Cholimimetics or parasympathomimetic** drugs increase ACh by inhibiting cholinesterase (indirect action) or directly stimulating cholnergic receptors (direct action).

**Bethanechol chloride** is a direct acting muscarinic receptor agonist. Gastric emptying was significantly hastened in one study, with increased activity also noted in the ileum, right ventral colon and cecum.\(^{59}\)

**Neostigmine methylsulfate** is an indirect acting agent (cholinesterase inhibitor). Increased cecal emptying along with increased activity in the ileum, pelvic flexure, right dorsal colon and cecum have been reported.\(^{60,61}\)

**Alpha-2 adrenergic antagonists or sympatholytic** drugs block receptors within the enteric nervous system allowing release of ACh from cholnergic neurons promoting intestinal motility. They counteract the inhibitory effect of sympathetic nervous stimulation on intestinal smooth muscle.

**Yohimbine** has shown the ability to increase activity of the right ventral colon and cecum; however, cecal emptying was unaffected. In some studies normal motility was not restored. In other studies yohimbine increased activity of the upper intestinal tract with a milder transient effect on the large intestine.\(^{60}\)

The combination of yohimbine and bethanechol may be more effective than bethanechol alone. Gastric emptying was enhanced in one study.\(^{59}\)

**Benzamides** stimulate the release of ACh from post synaptic cholnergic neurons.

**Metoclopramide** activates 5-HT receptors, antagonizes dopamine and blocks alpha-2 adrenergic receptors.\(^{62}\)
Continuous rate infusion led to a decrease in the volume and duration of gastric reflux. This was more effective than administration in a bolus. Contractile activity in the pylorus, duodenum and jejunum was increased.

Cisapride increases ACh release in the myenteric plexus, antagonizes 5-HT₃ receptors, increases 5-HT₁ activity, and stimulates motilin production. As the parenteral form is no longer available administration to refluxing horses is challenging, with variable results per rectum. Increased jejunal motility is seen with usage.

Lidocaine while in widespread usage clinically has displayed variable efficacy in clinical studies and the mode of prokinetic action is controversial. During in vitro mechanical studies of intestinal muscle in horses free from gastrointestinal disease lidocaine usage increased contractility of the proximal jejunum but not adjacent regions. An in vivo study of healthy horses displayed similar findings in that overall proximal jejunal motility was unaffected. However, lidocaine has been shown beneficial in clinical studies of refluxing horses.

Prophylactic lidocaine treatment was significantly associated with a reduced incidence of post-operative ileus and enhanced short-term survival in one study. However, it has also been demonstrated that a continuous infusion of lidocaine increased transit time of feces in normal horses.

Other Agents

Alpha-2 adrenergic agonists are widely used and affect intestinal motility. Xylazine decreases motility in the jejunum and pelvic flexure. Detomidine, and to a lesser extent xylazine, have been shown to relax the entire large intestine with profound analgesic effects. Both xylazine and detomidine have significant suppressive effects on duodenal motility. Detomidine appears more potent and of longer duration of effect than xylazine. Relaxation of both the small and large colon may be of benefit during attempts to rehydrate and conservatively manage the colon impaction patient, with passage of laxatives around the impaction facilitated.

Hyoscine-N-butylbromide has been shown to have an immediate and profound but short-lived depressive effect on cecum and left ventral colon contractions, but a minor and longer duration of effect on duodenal contractions. Studies have shown little effect on small intestinal motility. Rectal pressure is reduced facilitating more complete and safer examinations per rectum. Experimentally, hyoscine-N-butylbromide produced rapid analgesia (within 30 seconds after injection) of at least 20 minutes duration.

REFERENCES AND FOOTNOTE


