For many equine diseases, the goal of treatment is to target the underlying etiology; however, in cases of acute colitis, a definitive diagnosis may not be determined and was made in only 35% of cases in one report.\(^1\) Appropriate diagnostic tests may identify specific infectious organisms, but often not in a timely manner. Because the condition can be fatal, treatment must be initiated before results are obtained. The primary treatment goals for acute colitis are as follows:

- Maintenance of fluid and electrolyte balance
- Preservation of colloid oncotic pressure and replacement of plasma protein
- Control of local and systemic inflammation
- Promotion of tissue perfusion
- Promotion of mucosal repair
- Nutritional management

All patients with acute colitis require intensive care, and even if the primary treatment is effective, sequelae associated with the disease can limit a horse’s future performance or can be severe enough to require euthanasia. The cost of treatment can rise quickly, and the total bill may easily approach that for colic surgery.

**Fluid Therapy**

When the integrity of the gastrointestinal (GI) barrier is compromised, fluid shifts from the intravascular compartment to the interstitial compartment, which can have catastrophic effects. Depending on time to clinical assessment and the severity of disease, signs of hypovolemia may be apparent only within the intravascular space (i.e., poor systemic perfusion and pulse quality) or may manifest as clinical hypovolemia (TABLE 1). In cases of mild to moderate hypovolemia of short duration...
without continued GI dysfunction and ongoing losses, simple replacement of the calculated fluid deficit with an isotonic crystalloid solution may be adequate to restore fluid and electrolyte homeostasis (BOX 1). Typically, a polyionic, isotonic, crystalloid fluid (e.g., Normosol-R [Baxter Healthcare], lactated Ringer’s solution) can be used as an initial replacement fluid. The volume of fluid to administer should be estimated based on the replacement deficit/degree of hypovolemia, maintenance requirements, and anticipated ongoing losses.

A general guide for fluid replacement during initial resuscitation is 10 to 20 mL/kg/h, but rates of 20 to 45 mL/kg/h might be indicated in a profoundly hypovolemic patient. These high rates of fluid administration should be used only for the first 2 to 3 hours of treatment, during which the goal is to replace the calculated fluid deficit. Once a fluid volume equal to the calculated fluid deficit has been administered, ongoing fluid losses must be assessed and the fluid rate switched to a maintenance rate that accounts for the patient’s basal metabolic needs and ongoing losses. Prolonged fluid administration at a rate above 10 to 20 mL/kg/h in a patient without substantial fluid losses may result in edema, especially because many colitis patients have hypoproteinemia and diminished colloidal oncotic pressure. During colitis, fluid is lost into the colon wall and the GI lumen, so there is no quantitative means of accurately determining ongoing fluid losses, which can be as high as 150 mL/kg/d. Frequency, consistency, and volume of diarrhea per episode are useful subjective indicators; high-volume, high-frequency, watery diarrhea indicates that a large volume of fluid has been lost from the colon.

Most cases of acute colitis require a fluid rate two to three times the maintenance rate once the deficit has been replaced due to ongoing water loss through diarrhea; however, this rate varies from patient to patient and can be reduced with clinical improvement. Close monitoring of the patient’s hydration status by assessing capillary refill time and cardio-

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
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<tbody>
<tr>
<td>Skin turgor</td>
<td>Good to fair</td>
<td>Fair</td>
<td>Poor</td>
</tr>
<tr>
<td>Mucous membrane moisture</td>
<td>Good to fair</td>
<td>Tacky</td>
<td>Dry</td>
</tr>
<tr>
<td>Capillary refill time</td>
<td>1–2 sec</td>
<td>2–4 sec</td>
<td>&gt;4 sec</td>
</tr>
<tr>
<td>PCV&lt;sup&gt;a&lt;/sup&gt;</td>
<td>40%–50%</td>
<td>50%–65%</td>
<td>&gt;65%</td>
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<tr>
<td>Total plasma protein&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.5–7.5 g/dL</td>
<td>7.5–8.5 g/dL</td>
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<tr>
<td>Heart rate&lt;sup&gt;b&lt;/sup&gt;</td>
<td>40–60 bpm</td>
<td>60–80 bpm</td>
<td>&gt;80 bpm</td>
</tr>
<tr>
<td>Pulse quality&lt;sup&gt;a&lt;/sup&gt;</td>
<td>Good (easily palpated and turgid)</td>
<td>Fair (slightly weak with decreased tone)</td>
<td>Poor (weak/thready and difficult to palpate)</td>
</tr>
</tbody>
</table>

<sup>a</sup>Normal packed cell volume (PCV) depends on the horse’s breed and level of athletic training. Thoroughbreds and Standardbreds in training have normal PCVs up to 45%. The normal PCV of draft breeds can be 25%–30%. Splenic contraction and hypoproteinemia may affect PCV and total protein, respectively.

<sup>b</sup>Heart rate is also affected by the horse’s pain level.

### BOX 1

**Calculating Fluid Deficit and Formulating a Fluid Replacement Plan**

**Replacement fluid volume (L) = Bodyweight (kg) × % Hypovolemia ÷ 100 (L/kg)**

**Replacement fluid rate = 10–20 mL/kg/hr**

**Maintenance fluid volume = 50–100 mL/kg/day**

**Rate of maintenance fluid administration = 2–4 mL/kg/hr**

**Example:**

A 500-kg horse is 5% hypovolemic.

**Replacement fluid volume (L) = 500 kg × 5% Hypovolemia ÷ 100 = 25 L**

**Replacement fluid rate (hr) = 10–20 mL/kg/hr × 500 kg/1000 mL/L = 5–10 L/hr for 2.5–5 hr**

**Maintenance fluid volume (L/day) = 50–100 mL/kg/24 hr × 500 kg/1000 mL/L = 25–50 L/day**

**Maintenance rate (L/hr) = 2–4 mL/kg/hr × 500 kg/1000 mL/L = 1–2 L/hr**
vascular parameters (e.g., heart rate, pulse quality) is essential to ensure an appropriate fluid rate. Indirect blood pressure monitoring is also useful for assessing response to treatment. Measurement of packed cell volume (PCV) and total protein concentration (TP) is quick, inexpensive, and clinically useful for monitoring hydration status. However, while increased PCV and TP suggest hypovolemia, these values may be lower than expected due to intravenous fluid therapy and hypoproteinemia secondary to colitis. In very mild cases of acute equine colitis, the volume of each fecal episode and the frequency (e.g., five to six times per day) of diarrhea are only moderate, so the patient can maintain an acceptable hydration state without supplementary intravenous crystalloid fluid therapy.

When a fluid therapy plan is designed, it is important to remember that sodium-containing fluids rapidly exit the vasculature to equilibrate with extracellular fluid, and only 25% of these fluids remain in the intravascular space. Therefore, if ongoing fluid losses are significant, adequate colloidal support (e.g., plasma) and electrolyte supplementation must be provided. In severely hypovolemic equine patients, hypertonic saline (1 to 2 L of 7% sodium chloride [NaCl]) can expand intravascular blood volume, increasing systemic blood pressure and cardiac output. Circulating fluid volume can be rapidly increased as fluid moves from the extracellular space into the vascular compartment in response to hypertonic solution in the vascular space. However, after administration of hypertonic saline, it is imperative to quickly administer crystalloid fluids to replenish fluid from the extracellular space and maintain intravascular fluid volume.

Electrolyte Supplementation and Correction of Acid–Base Derangements
Horses with colitis often have marked electrolyte deficiencies, which can be exacerbated by aggressive fluid therapy. Diminished absorption and increased secretion via the GI tract lead to a net loss of serum sodium, chloride, potassium, calcium, and bicarbonate into the colonic lumen. Clinical signs do not adequately predict the patient’s need for electrolyte supplementation, so this should be determined from measured plasma concentrations. Ongoing monitoring throughout the course of the disease is also essential. The recommended supplementation rate differs for each ion. Potassium is typically supplemented using potassium chloride (KCl). The starting supplementation rate should be determined from the serum potassium concentration after clinical rehydration of the patient, and the clinician should be careful to account for the ongoing loss of potassium, especially in an inappetent patient. In most cases, adding KCl to intravenous fluids at a concentration of 20 mEq/L is appropriate for replacement therapy. More severe hypokalemia can be treated by increasing the KCl concentration to 40 mEq/L, but care must be taken not to exceed an administration rate greater than 0.5 mEq/kg/h.

Hypocalcemia is usually treated with intravenous administration of 23% calcium gluconate solution. Daily administration of 100 to 300 mL (2.14 to 6.42 g) is typically required in equine patients with ongoing GI losses, but the amount depends on the severity of disease and the patient’s nutritional intake. The calcium gluconate solution is typically added to the intravenous fluids that the patient is already receiving, but calcium-containing solutions cannot be used concurrently with bicarbonate-containing solutions because this results in formation of calcium carbonate precipitate. Some horses with severe colitis remain hypocalcemic despite aggressive calcium supplementation, so ongoing monitoring of the ionized calcium concentration is indicated. Excessively rapid calcium administration may result in cardiovascular complications, particularly in septic horses, which may be more vulnerable to toxic effects of calcium. However, a calcium dose of 1 to 2 mg/kg/h is considered safe, and Toribio et al have induced hypercalcemia in horses with rapid administration of calcium gluconate with no obvious complications. Concurrent administration of fresh-frozen plasma or blood results in a transient fall in the number of divalent cations as a result of chelation by citrate.

Magnesium is typically supplemented using magnesium sulfate (MgSO₄). A recommended dosage of intravenous MgSO₄ in adult horses is 25 to 150 mg/kg/d (12.5 to 75 g/d for a 1100-lb [500-kg] horse), and MgSO₄ can be added to the crystalloid fluids that the horse is already receiving. Magnesium is already added to some
crystalloid fluids (e.g., Normosol-R), so this must be considered when calculating the rate of magnesium supplementation.

When sodium is supplemented beyond what is contained in primary resuscitation fluids, it is important to remember that in other species, rapid correction of sodium deficits has been shown to cause demyelination of pontine and extrapontine neurons, resulting in severe neurologic dysfunction. This concern is greatest when the patient is profoundly hyponatremic (serum Na+ concentration: ≤120 mEq/L). A simple guideline for clinical use is to calculate the sodium deficit using the following equation:

\[
\text{Normal serum sodium concentration} - \text{Actual serum sodium concentration} = \text{Sodium deficit (mEq/L)}
\]

The result can be used as a guideline; for example, a deficit of 20 mEq/L should be replaced over no fewer than 20 hours and a deficit of 15 mEq/L over no fewer than 15 hours. Hypertonic saline (5%) or sodium bicarbonate (5% or 8.5%) solutions are used for sodium supplementation; NaCl may be used when hyponatremia is accompanied by hypochloremia, and sodium bicarbonate may be used when the serum chloride concentration is normal or increased. To avoid excessive or overly rapid correction of the serum sodium concentration, the plasma sodium concentration should be carefully monitored. We recommend taking a repeat blood sample 6 to 8 hours after initiating corrective fluid therapy and every 12 hours thereafter until the sodium concentration is within normal limits.

Acid–base disturbances in colitis patients are primarily corrected by addressing the primary disease process and the associated sequelae, such as hypovolemia. Metabolic acidosis frequently accompanies acute colitis due to (1) the colon’s failure to resorb bicarbonate and (2) lactate buildup in the tissues secondary to poor tissue perfusion and anaerobic metabolism associated with endotoxemia. Correction of hypovolemia is an important component of therapy for acid–base disorders in diarrheic patients. If correction of hypovolemia is not accompanied by correction of acid–base abnormalities, additional therapy will be required to correct plasma imbalances.

For example, hyponatremia leads to metabolic acidosis, so sodium supplementation should help resolve acidosis. Similarly, albumin is a weak acid, so severe hypoalbuminemia may contribute to metabolic alkalosis or mask concurrent metabolic acidosis; therefore, protein supplementation should correct the acid–base imbalance. Persistence of acidosis despite correction of oral or intravenous supplementation should prompt the clinician to reassess the source of the pH abnormality (i.e., Is it respiratory or metabolic acidosis?). In addition, the clinician should address the underlying cause of this physiologic imbalance by treating the primary disease process.

Oral administration of fluids is an effective and economical adjunct to intravenous fluid administration. Once severe electrolyte or acid–base disturbances have been corrected, horses, if given a choice, often elect to drink a solution containing the electrolyte in which they are deficient. The following “water buffet,” including an assortment of electrolyte-supplemented water solutions, can be offered free choice:

- Plain water
- Water with 6 to 10 g/L Lite Salt (iodized NaCl and KCl; Morton)
- Water with 10 g/L baking soda (sodium bicarbonate)
- Water with 6 to 10 g/L plain salt (NaCl)

**Colloidal Support**

Because of GI losses and serum albumin catabolism, many horses with acute colitis are hypoproteinemic. Additionally, absorption of bacterial products may induce systemic inflammatory response syndrome (SIRS), leading to increased vascular permeability and low plasma oncotic pressure. A large volume of crystalloid fluids causes hemodilution, contributing to decreased plasma oncotic pressure. In contrast, colloids preserve colloidal oncotic pressure, resulting in more effective volume expansion. Commercial colloids include plasma, dextran 40, dextran 70, hydroxyethyl starch (hetastarch), and polymerized bovine hemoglobin. The duration of oncotic support provided by a colloid is affected by the severity of inflammation in the colon and by resultant molecular loss from the circulation. Hetastarch (up to 10 mL/kg/d) has been shown to increase colloidal oncotic pressure for up to 24 hours in

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**Critical Point**

Many adjunctive anecdotal therapies are available for treating acute colitis, but clinical evidence supporting their use is limited.
hypoproteinemic horses. Experimental studies suggest hetastarch may be superior to plasma in stopping endotoxin-induced increases in vascular permeability. This is due to the larger molecular size of polymers in hetastarch and, perhaps, to its ability to attenuate permeability dysfunction associated with endotoxemia. Caution is indicated, as higher doses of hetastarch (>20 mL/kg total cumulative dose) may prolong bleeding by altering von Willebrand’s factor function.

Intravenous administration of plasma provides protein (albumin and globulins) as well as other beneficial elements, such as coagulation factors. Acute colitis is probably the most common disease associated with disseminated intravascular coagulation (DIC) in horses. In one study, the 1-year incidence of subclinical DIC in horses with acute colitis was 32%. There is no published dose of plasma for correction of hypoproteinemia in adult horses; however, as a guideline, equine patients with acute colitis require 10 to 15 mL/kg of plasma to raise their TP approximately 1 g/L. Close monitoring of the patient’s plasma protein is essential, as ongoing GI losses may require repeated doses of plasma. Fresh-frozen commercial equine plasma is readily available from various sources. Adverse effects of plasma administration in horses are uncommon but may include immune-mediated reactions and changes in hemostatic variables.

**Antiinflammatory Therapy**

The NSAIDs typically used in equine patients inhibit cyclooxygenase (COX) 1 and 2, which are responsible for producing vasoactive and proinflammatory prostanoid compounds, such as prostaglandin I₂ (PGI₂), prostaglandin E₂ (PGE₂), and thromboxane. Suppression of this process can help break the self-perpetuating inflammatory cycle within inflamed colonic mucosa and allow GI mucosa to heal. However, evidence suggests that certain prostaglandins, such as PGI₂ and PGE₂, are cytoprotective to GI mucosa and critical for mucosal repair; therefore, using COX inhibitors to block prostaglandin production in these tissues may exacerbate GI pathology. It is unclear whether the benefits of NSAID therapy outweigh the limitations; however, many horses with colitis may initially require analgesia. Similar to the standard dose of flunixin meglumine (1.1 mg/kg IV q12h), the low dose (0.25 mg/kg IV q8h) has been shown to decrease eicosanoid production and ameliorate some clinical signs of systemic inflammation following exposure to intravenous endotoxin. The low dose is also thought to be less likely to impair tissue blood flow in the GI mucosa; therefore, the low dose may be safer than the standard dose. Other antiinflammatory therapies for equine acute colitis include the free radical scavenger dimethyl sulfoxide (DMSO; 1 g/kg IV q12–24h as a 10% solution), the antimicrobial metronidazole (20 mg/kg PO q8h; 10–25 mg/kg PO q6–12h⁹; 15–25 mg/kg PO q6h¹⁰), the prokinetic and analgesic lidocaine (1.3 mg/kg over 15 min, then 0.05 mg/kg/min), and the PGE₁ analogue misoprostol (5 µg/kg q8h). Experimental evidence for these therapies is limited, but they may be of clinical value.

**Analgesic Therapy**

Many horses with acute colitis develop mild to severe signs of abdominal discomfort from gas and fluid distention of the colon, colonic ischemia, and infarction. NSAIDs are commonly used, particularly flunixin meglumine (1.1 mg/kg IV or PO q12h) because of its antiinflammatory effects compared with the effects of phenylbutazone or ketoprofen. However, NSAIDs impair prostaglandin production by the kidneys, inhibiting normal renal blood flow. Overdosage or misuse of NSAIDs, especially in hypovolemic patients, can lead to renal crest necrosis and may increase the risk of acute tubular nephrosis during hypovolemia or aminoglycoside administration. Therefore, low-dose flunixin meglumine may be better to use than the standard dose in some patients. Alternative analgesics include lidocaine, α₂-agonists (e.g., xylazine, detomidine), and opioids (e.g., butorphanol). These are all considered to be short acting, but constant-rate infusion may be used to provide sustained clinical analgesia. However, opioids and α₂-agonists decrease GI motility, so patient comfort and fecal output should be monitored.

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**Critical Point**

Prevention and treatment of the devastating sequelae of acute colitis are crucial to optimizing a patient’s chance of survival.
Antidiarrheal Therapy
Bismuth Subsalicylate

Bismuth subsalicylate is commonly administered orally to equine patients with colitis to decrease inflammation and secretion in the colon. In a double-blind, placebo-controlled study in children with acute diarrhea, this drug was shown to significantly decrease the time to the last watery stool.20 The precise mechanism of action of bismuth subsalicylate in any species is unclear, but the salicylate moiety may have an antisecretory action in the large colon, where it stimulates fluid and electrolyte absorption.21 In addition, salicylic acid inhibits prostaglandin synthesis, which is responsible for intestinal inflammation and hypermotility, and modulates oxidative stress in colonic mucosal cells.22 Furthermore, the drug and its intestinal reaction products, bismuth oxychloride and bismuth hydroxide, appear to be bacteriocidal in vivo and in vitro.23 Based on extrapolation from human medicine, the dose required in adult horses is large (3 to 4 L by stomach tube q4–6h)20; however, the drug is also available as a concentrated paste. In humans, bismuth subsalicylate is considered extremely safe. A feeding trial in which mice were fed 60 times the maximal recommended human dose did not result in adverse effects, and no histopathologic lesions were noted in the brains on postmortem examination.24 Bismuth subsalicylate toxicosis in horses has not been reported.

Di-Tri-Octahedral Smectite

Di-tri-octahedral smectite is a natural hydrated aluminomagnesium silicate with a lamellar structure. It binds to digestive mucus and increases intestinal resistance to bacterial damage.24 The drug has been shown to increase water and electrolyte absorption in rabbit intestinal loops in the presence of Escherichia coli infection; a preliminary study in horses reported that administration of di-tri-octahedral smectite prevented lincomycin-induced colitis in four horses, whereas four untreated horses died or were euthanized due to severe colitis.24,25 In vitro studies have shown that di-tri-octahedral smectite can bind Clostridium difficile toxins A and B as well as Clostridium perfringens enterotoxin and endotoxin.26 The current recommendation for a 1000-lb (454.5-kg) horse is administration of a solution of 1 lb of Bio-Sponge (Platinum Performance) and 3 L of water via nasogastric tube q6–12h.

Probiotics

Restoring the microbial ecology of the colon has recently attracted experimental interest, leading to the use of many different agents (e.g., commercial probiotic pastes, live-culture yogurt) and techniques (e.g., transfaunation). In transfaunation, freshly harvested colonic or cecal contents (5 to 6 L) or a slurry of fresh feces from a healthy horse is administered via nasogastric tube to the patient to restore normal GI flora. Transfaunation has had reported clinical success in cattle but has not been reported in diarrheic horses.27 There is little supportive evidence for the use of commercial probiotic pastes in horses, and one study in foals found the pastes to be detrimental.26 In comparison, Saccharomyces boulardii is a nonpathogenic yeast that has been used prophylactically and therapeutically as an antidiarrheic agent in humans since 1962.29 Experimentally, the yeast has been found to survive within the equine GI tract, and the severity and duration of acute enterocolitis were significantly decreased in horses that received S. boulardii compared with horses that received a placebo.29 S. boulardii has been found to release a protease that can digest C. difficile toxins A and B; additional mechanisms of action include an immunoprotective effect attributed to promoting the release of secretory immunoglobulins within the intestine or activation of the reticuloendothelial and complement systems.30,31 Because pharmacokinetic studies with S. boulardii have not been performed in horses, the equine dose is extrapolated from the human literature. One study reported the use of an S. boulardii dose of 25 g (10 × 10⁹ yeast cells) PO q12h for 14 days in horses with acute colitis, resulting in no clinical adverse effects and in significant improvement in the severity and duration of GI disease compared with a placebo group of horses.29

Antimicrobial Therapy

Antimicrobial therapy is hotly debated in many cases of acute colitis. In cases with concurrent neutropenia, it is thought that the host’s defenses may be weakened sufficiently to render the horse susceptible to organisms

Critical Point

In a double-blind, placebo-controlled study in children with acute diarrhea, this drug was shown to significantly decrease the time to the last watery stool.
that breach the mucosal barrier; therefore, some clinicians recommend broad-spectrum antimicrobial coverage. However, antimicrobials can have adverse GI effects in horses. Antimicrobial administration has been reported to prolong shedding of Salmonella spp in experimentally infected ponies, and there are many reports of antimicrobial-induced diarrhea in horses.

For certain infectious causes of acute colitis in horses, such as Potomac horse fever (equine monocytic ehrlichiosis) and Clostridium infections, antimicrobial therapy is required to address the underlying cause. Neorickettsia risticii, which causes Potomac horse fever, is highly sensitive to tetracyclines (e.g., oxytetracycline dosed at 6.6 mg/kg IV q24h for 5 days). Clostridium spp, namely C. difficile and C. perfringens, have been shown to be eradicated using metronidazole (minimum inhibitory concentration: ≤4 mg/mL); commonly used dosage: 20 mg/kg PO q8h, and in vitro evidence suggests efficacy of chloramphenicol (50 mg/kg PO q6h). Antimicrobial use must be decided on a case-specific basis, with consideration for the most likely etiology, including the most common agents in the area, the season of the year, and the history and clinical presentation.

Critical Point
Experimnetally, the yeast has been found to survive within the equine GI tract, and the severity and duration of acute enterocolitis were significantly decreased in horses that received S. boulardii compared with horses that received a placebo.

Treating Acute Colitis

Because of the severity of systemic illness associated with acute colitis, complicating issues are frequently encountered, the most common of which are endotoxia, thrombophlebitis, and laminitis. Preventive or therapeutic measures must address these sequelae to optimize the patient’s chance of survival.

Endotoxia is a well-recognized complication of GI disease, particularly diseases involving GI inflammation or ischemia, such as acute colitis. A horse’s intestinal tract normally contains a large number of gram-negative bacteria that release endotoxin when they die or multiply rapidly. The endotoxin is normally restricted to the intestinal lumen by an efficient intestinal mucosal barrier; however, if some endotoxin crosses the mucosal barrier, it enters the portal system and is removed by hepatic mononuclear phagocytes without initiating systemic signs in the horse.

If the intestinal barrier is impaired (e.g., inflammation, ischemia), endotoxin translocates more significantly into the circulation, and the hepatic clearance mechanism becomes overwhelmed, leading to endotoxiaemia as well as synthesis and release of inflammatory mediators.

Four therapeutic approaches should be considered when addressing endotoxiaemia. The first approach is to prevent absorption of endotoxin into the circulation by treating the primary cause of the GI disease. The second approach is neutralization of endotoxin before it interacts with inflammatory cells. Polymyxin B has shown some promising endotoxin-neutralization effects in vitro and in vivo, appearing to be clinically useful in decreasing the inflammatory response to endotoxin exposure. However, polymyxin B must be used judiciously due to its inherent toxic effects on neural and renal tissues. Polymyxin B is administered at a rate of 1 mg (6000 U)/kg diluted in 1 L of sterile saline solution IV q8h and is typically discontinued after 1 or 2 days of therapy; however, many clinicians administer the drug every 12 hours to prevent adverse effects. Doses below 6000 U/kg may be effective and less nephrotoxic. Although few experimental data are available to support the in vivo use of antilipopolysaccharide (anti-LPS) hyperimmune equine plasma, it provides antibodies that target the endotoxin and appears to have bacteriocidal activity. The third approach is prevention of the synthesis, release, or action of inflammatory mediators that follow endotoxin exposure and are responsible for SIRS. This approach has included the use of NSAIDs, corticosteroids, monoclonal antibodies directed against cytokines, platelet-activating factor receptor antagonists, pentoxifylline, and naturally occurring nontoxic endotoxins. However, all of these interventions have demonstrated only limited efficacy, and only flunixin meglumine has become clinically accepted.

The fourth and most clinically important approach to endotoxiaemia is appropriate supportive care. This helps minimize organ dysfunction secondary to severe SIRS, which is characteristic of endotoxiaemia.

Abdominal pain associated with colitis may result in stall rolling, excessive catheter movement, and contamination or disconnection of the intravenous line. Patients with acute colitis can be at high risk for thrombophlebitis (inflammation of the vein with thrombus for-
Treating Acute Colitis

because of associated general debilitation, lowering of the head for prolonged periods, and placement of an indwelling catheter for several days or weeks. In addition, a large volume of intravenous fluids and intravenously administered drugs can cause turbulent blood flow and irritate vascular endothelium at the catheter tip. Patients with colitis may be predisposed to venous thrombosis because of endotoxemia-associated loss of anticoagulants in the bloodstream and systemic activation of procoagulants. No studies have proven that type of catheter material is a risk factor for thrombophlebitis in horses, but polyurethane, over-the-wire catheters are assumed to have less risk. If thrombophlebitis develops, the use of topical antiinflammatory therapies (e.g., DMSO), systemic anticoagulants (e.g., aspirin), and antimicrobials is advisable but has not been experimentally confirmed to be beneficial.

Laminitis is the primary reason for euthanasia in many colitis cases. Prevention and treatment of laminitis are controversial, primarily because the exact pathophysiology of the condition is unclear. To effectively prevent laminitis, clinicians must be aware that patients with colitis are at high risk of developing it. Preventives include vasodilator administration, corrective hoof trimming and shoeing, deeply bedded stalls, and frog support. Many of these measures are also used therapeutically.

Venodilatory therapy, namely low-dose acepromazine (0.03 to 0.06 mg/kg IM q6–8h), isoxsuprine hydrochloride (1.2 mg/kg PO q12h), or topical glyceryl trinitrate cream (2-cm strip applied to the back of the pattern of the affected feet q6–8h), should theoretically reduce venous resistance in the feet, decrease capillary pressure, and diminish abnormal fluid movement within laminar tissue. Conversely, blood flow to the feet can be decreased or altered by dramatically cooling the feet with ice-packed boots. The associated mechanism of action is thought to be induction of hypometabolism of laminar tissue, thereby decreasing proteolytic and neutrophil enzyme activities, inflammatory

Critical Point

For certain infectious causes of acute colitis in horses, such as Potomac horse fever (equine monocytic ehrlichiosis) and Clostridium infections, antimicrobial therapy is required to address the underlying cause.
cytokine activity, and metalloproteinase activity, which have been found to be increased in laminitic feet. Applying supportive pads to the feet, trimming the hooves, and using soft bedding may decrease mechanical shear forces on the laminae, limiting the predisposition to or exacerbation of laminitis. Inhibiting neutrophil adhesion to endothelial cells with the use of pentoxifylline, lidocaine, or flunixin meglumine could also have some prophylactic value. If a horse with acute colitis develops laminitis, treatment should be directed at pain control and ongoing correction of the underlying cause. Historically, phenylbutazone (2.2 to 4.4 mg/kg IV or PO q12h) has been regarded as the best NSAID for treating pain and inflammation associated with laminitis; however, it is important to consider using available COX-2–selective NSAIDs such as firocoxib (Equioxx, Merck) and meloxicam (Metacam, Boehringer Ingelheim). Further research may show COX-2 inhibitors to be superior and safer. Lateral radiographs of the feet illustrating rotation or sinking of the pedal bone in addition to response to analgesic therapy are key prognostic indicators in laminitic cases.

Nursing Care and Nutrition
Good nursing care and nutrition are essential to a successful outcome for equine patients with acute colitis (BOX 2). The goal of enteral nutrition should be to avoid overloading the poorly functioning colon; this can be achieved by feeding small, frequent meals with a predominantly pellet base. Feeds with greater digestibility decrease the amount of undigested concentrate that reaches the cecum, where fermentation may exacerbate diarrhea. Hay can be fed but should be high-quality grass hay. Feeding hay may help produce volatile fatty acids (propionate, butyrate, acetate) within the colon; these compounds are important for normal functioning of colonic mucosa. Because many colitis patients are anorectic and the severity of the disease can result in catabolism, corn oil can be added to the diet to increase caloric intake. If a patient remains anorectic for longer than 3 to 4 days despite therapy, parenteral nutrition should be provided. In addition, gastroprotectants such as sucralfate (1 g/45.5 kg PO q6–8h) and omeprazole (4 mg/kg PO q24h) are useful for improving appetite and treating gastric ulcers, if present, due to inappetence.

Response to Therapy
Response to therapy is determined by frequent monitoring of clinical signs, clinicopathologic data, and fecal water content. Signs of improvement are decreased fever, stability of serum electrolyte concentrations, acid–base balance, and improved appetite. Clinicopathologically, one of the earliest signs of improvement can be a decreased number of morphologically “toxic” neutrophils. Decreases in fecal water content and frequency of diarrhea suggest clinical improvement. Acute colitis can have an infectious cause, so equine patients may continue shedding the infectious agent even when diarrhea has resolved, thereby putting other horses at risk. Repeated diagnostic testing should be considered before removing a horse from isolation. Because salmonellosis patients shed Salmonella spp intermittently, a series of five fecal cultures obtained on different days should have negative results before isolation protocols are discontinued. Alternatively, three consecutive fecal samples can be obtained 24 hours apart and submitted for polymerase chain reaction (PCR) testing to test for Salmonella spp. This method can provide a level of confidence similar to that obtained by testing five samples by culture. Because watery feces are difficult to culture
for *Salmonella* spp due to a dilutional effect, samples for culture or PCR testing should contain at least 5 to 10 g of feces. The usefulness of repeat testing in clostridiosis cases has not been critically evaluated, but obtaining negative results from a fecal sample following resolution of diarrhea helps ensure that the risk of shedding has decreased. Because salmonellosis is highly contagious, many hospitals have an infectious disease, environmental monitoring protocol.

**Prognosis and Outcome**

The substantial mortality rate and treatment expense associated with acute enterocolitis underscore the importance of identifying equine patients with a poor prognosis for survival. Patients that recover from acute colitis typically show clinical improvement within 3 to 6 days after treatment begins. The following clinical signs suggest a guarded prognosis: azotemia, clinicopathologic findings consistent with hemoconcentration and hypoproteinemia (e.g., a persistent PCV >50% and TP <6.2 g/dL), and failure to show demonstrable signs of improvement after 10 days of therapy. Certain types of colitis, including necrotizing enterocolitis and antimicrobial-associated diarrhea, have been associated with low survival rates. If a horse survives acute colitis without developing sequelae such as laminitis, ongoing health issues are unlikely.

**Conclusion**

Managing equine patients with acute colitis can be challenging due to the intensity of care involved and the concerns regarding disease transmission. Many different treatments are available for this condition; although the efficacy of some treatments is unclear, clinicians have the opportunity to explore different therapeutic approaches, making acute colitis rewarding to treat. It is important to remember that if the patient responds to therapy in the first few days, the prognosis for a full recovery is favorable.
References

Treating Acute Colitis

1. Which fluid would not be appropriate to administer to a hypovolemic equine patient?
   a. hypertonic saline
   b. lactated Ringer’s solution
   c. plasma
   d. dextrose 50%
   e. 0.9% saline

2. Which serum level is not typically low in an equine patient with acute colitis?
   a. sodium
   b. chloride
   c. lactate
   d. potassium
   e. bicarbonate

3. Which statement regarding equine acute colitis is false?
   a. Prostaglandins such as PGL and PGE may be cytoprotective to GI mucosa.
   b. Flunixin meglumine must be administered at 1.1 mg/kg IV q12h to decrease production of tumor necrosis factor and other inflammatory cytokines in the GI mucosa.
   c. Horses with severe colitis have profound hypoproteinemia.
   d. Metronidazole use is indicated in cases of C. difficile infection.
   e. DMSO and lidocaine may be used as anti-inflammatories in horses with acute colitis.

4. Which statement regarding bismuth subsalicylate is true?
   a. The anecdotal dose of liquid bismuth subsalicylate for horses is large, requiring passage of a stomach tube every 6 to 8 hours. Alternatively, a concentrated paste can be administered.
   b. Several cases of associated toxicosis in horses have been reported.
   c. Adverse cardiac signs were reported in a person with suspected bismuth subsalicylate toxicity.
   d. It has a prokinetic mechanism of action.
   e. No studies support its use in humans.

5. Which commonly used therapy lacks experimental support as a beneficial treatment of equine acute colitis?
   a. bismuth subsalicylate
   b. metronidazole
   c. di-tri-octahedral smectite
   d. S. boulardii
   e. probiotic pastes

6. Which disease and treatment combination for equine patients is incorrect?
   a. C. difficile infection; metronidazole
   b. Potomac horse fever; oxytetracycline
   c. C. difficile infection; S. boulardii
   d. antimicrobial-associated colitis; ceftiofur sodium
   e. right dorsal colitis; misoprostol

7. Which statement regarding colloid therapy in equine patients is true?
   a. Administering plasma is beneficial only if the volume is adequate to replace all protein loss.
   b. Hydroxyethyl starch should not be administered to horses with diarrhea.
   c. Hypertonic saline has short-term colloidal action.
   d. Plasma administration reduces the chance of developing laminitis.
   e. Colloid administration decreases plasma oncotic pressure.

8. Which statement regarding fluid therapy for equine acute colitis is false?
   a. Fluids should not be administered until clinicopathologic test results are available.
   b. Calcium supplementation is often required due to reduced feed intake and increased GI loss.
   c. Ongoing fluid losses must be considered in addition to maintenance requirements and replacement of lost fluid when determining an appropriate fluid plan.
   d. Potassium should be supplemented carefully.
   e. Sodium bicarbonate administration is appropriate for correcting hyponatremia in some cases.

9. Which statement regarding equine acute colitis is false?
   a. Many components of treatment are the same regardless of etiology.
   b. Fluid therapy is crucial to all treatment plans.
   c. Antimicrobial therapy should not be routinely administered in colitis cases.
   d. If complications do not occur and diarrhea resolves, the patient should have no long-term effects.
   e. Laminitis is an uncommon sequela of acute colitis.

10. Which statement regarding treatment of equine acute colitis is false?
    a. Treatment with COX inhibitors should be routine because they limit the chance of developing intestinal ulceration.
    b. The volume and frequency of diarrhea are good subjective indicators of fluid loss and should be considered when devising a fluid therapy plan.
    c. There is limited evidence to support the use of transfaunation.
    d. Antimicrobial therapy may prolong bacterial shedding in salmonellosis cases.
    e. Oxytetracycline is an appropriate therapy for Potomac horse fever.