Electrolyte abnormalities are extremely common in horses presenting to referral hospitals with acute abdominal pain. Ninety-three percent of 30 horses treated medically and 98% of 59 horses treated surgically had at least one electrolyte level outside the normal range during hospitalization when sodium, potassium, chloride, ionized calcium, and ionized magnesium concentrations were measured at regular intervals for the first 3 days after admission. 

If sodium, potassium, calcium, and magnesium concentrations were outside the normal range, they were usually low. If abnormal, the chloride concentration was usually high. Clinicians must decide which electrolyte abnormality to treat as well as how and when to do so. This requires a basic understanding of the physiologic role of these electrolytes and the consequences, if any, of treatment. The electrolyte abnormalities that I most commonly treat are low potassium and magnesium concentrations. Abnormalities of calcium, sodium, and chloride concentrations are common but require much less frequent treatment.

**POTASSIUM**

Forty-seven percent of horses treated medically and 85% of horses treated surgically have a potassium concentration below the reference range during their hospitalization. Even after aggressive therapy for hypokalemia in these horses, we still regularly find their potassium concentrations to be less than 2.7 mmol/L after surgery. There are two major reasons for this high incidence of hypokalemia in horses with colic: First, the stress of disease increases glucocorticoid and mineralocorticoid release, promoting excretion of potassium in the urine. Second, the infusion of large amounts of sodium-containing fluids increases distal tubular flow and, therefore, renal potassium loss. Horses undergoing surgery for colic are likely to have greater glucocorticoid and mineralocorticoid release and to be treated with greater volumes of intravenous fluids, which explains these horses’ increased likelihood of developing hypokalemia.

In humans and small animals, intestinal ileus is a major clinical sign of hypokalemia. In horses, although this association has not been proven, clinical experience suggests that hypokalemia is also related to ileus. Other clinical signs of hypokalemia include muscle weakness, lethargy, and the inability to concentrate the urine.

I supplement potassium, even if it is only slightly below the normal range, in colic patients because of the presumed association with ileus and the fact that the blood potassium concentration only partly reflects the total body potassium deficit. Because of the high incidence of hypokalemia, some clinicians choose to routinely supplement potassium, without measuring it, following colic surgery. Treatment of hypokalemia involves potassium replacement, either intravenously or orally (see box on p. 17). Rapid administration of intravenous potassium can lead to cardiac arrhythmias, so potassium should not be infused at a rate above 0.5 mmol/L of K+/kg/hr. Potassium chloride is the
Treatment of Electrolyte Abnormalities in Colic Patients

**Supplementation Guidelines for Common Electrolyte Abnormalities in Colic Patients**

**POTASSIUM** (Maintain a concentration of 3.5–5 mmol/L)

**Hypokalemia**
- Never infuse intravenously at >0.5 mmol/kg/hr
- **Rule of thumb**: Treat horses weighing >880 lb (>400 kg) and when the fluid rate is no faster than 5 L/hr (continuous fluid infusion; periodically check K⁺ concentration and adjust)
- If K⁺ is <2.5 mmol/L, add 40 mmol/L (= 77 ml of 20% KCl per 5-L bag of crystalloid fluids) and (if appropriate) 0.1–0.2 g/kg PO
- If K⁺ is <2.9 mmol/L, add 30 mmol/L (= 58 ml of 20% KCl per 5-L bag of crystalloid fluids) and (if appropriate) 0.1–0.2 g/kg PO
- If K⁺ is <3.2 mmol/L, add 20 mmol/L (= 39 ml of 20% KCl per 5-L bag of crystalloid fluids)
- If K⁺ is <3.5 mmol/L, add 10 mmol/L (= 19 ml of 20% KCl per 5-L bag of crystalloid fluids)
  — 20% KCl contains 13.4 mEq of potassium per 5 ml; adjust the volume of KCl added for different concentrations of solution
- If the horse remains hypokalemic despite aggressive potassium supplementation, supplement magnesium (unless the patient is hypermagnesemic)

**MAGNESIUM** (Maintain ionized magnesium at a concentration of 0.35–0.7 mmol/L [0.85–1.7 mg/dl])

**Hypomagnesemia**
- Supplement with 4–16 mg/kg magnesium sulfate (≈16 ml of 250-mg/ml solution) per 5-L bag of crystalloid fluids
- Remeasure magnesium within 12 hr

**Hypermagnesemia**
- Usually iatrogenic in colic patients (overadministration of magnesium sulfate [Epsom salts] by nasogastric tube)
- Treat the horse if it is symptomatic (weakness, recumbency)
- Treat with 125–250 ml of 40% calcium gluconate IV over 15 min
- Investigate renal function

**CALCIUM** (Maintain ionized calcium at a concentration of 0.9–1.8 mmol/L [3.6–7.2 mg/dl])

**Hypocalcemia**
- Treat with 0.1–0.5 ml/kg of 40% calcium gluconate solution IV over 2–3 hr
- Consider vitamin D₃ supplementation, especially in foals
- Evaluate the magnesium concentration, which may require concurrent treatment

**SODIUM** (Maintain a concentration of 128–150 mmol/L)

**Hyponatremia**
- Initially treat the horse with crystalloid fluids (lactated Ringer’s solution or Normosol-R) and remeasure the sodium concentration after 24 hr
- If hyponatremia persists or worsens:
  — Treat with 1.3% sodium bicarbonate (if there is not concurrent hypochloremia) or sodium chloride (if the horse is hypochloremic)
  — Frequently measure the sodium concentration and do not increase it faster than 1 mmol/L/hr

**CHLORIDE** (Maintain a concentration of 92–115 mmol/L)

**Hyperchloremia**
- Treat with 1.3% sodium bicarbonate (if there is not concurrent hypernatremia) or 5% dextrose solution (if the horse is hypernatremic)
- If concurrent hypernatremia is treated with 5% dextrose, the sodium concentration must not be lowered faster than 0.5 mmol/L/hr

**Hypochloremia**
- Treat with sodium or potassium chloride, to effect, depending on the status of these electrolytes
- KCl must not be administered faster than 0.5 mmol/L of potassium/kg/hr

Ideal replacement salt, especially in horses with gastric reflux that are, therefore, concurrently hypochloremic. High crystalloid flow rates result in increased urine production and kaliuresis, making it more difficult to replace potassium intravenously. Oral supplementation is more effective in horses without ileus, making this route available. The dose is 0.1 to 0.2 g/kg PO or by nasogastric tube. In foals, this is usually divided into three or four doses given at intervals of at least 4 hours. Horses with hypomagnesemia may be refractory to potassium replacement therapy unless the magnesium deficit is simultaneously corrected. In the absence of clinical signs, hyperkalemia can be treated by intravenous administration of polyionic fluids. Severe hyperkalemia (i.e., potassium concentration: >7 mmol/L) associated with clinical signs may be treated with calcium gluconate (1 ml/kg IV over 10 minutes), sodium bicarbonate (1 to 2 mmol/L IV over 15 minutes), and 50% dextrose solution (2 ml/kg IV over 5 minutes).
Therapeutics in Practice

Key Points

- Electrolyte abnormalities are extremely common in referred horses with colic.
- Major changes in electrolyte concentration can be associated with major morbidity and increased mortality.
- The safest way to supplement electrolytes is to measure and replace them according to the results rather than to give standard electrolytes to all horses recovering from colic surgery.

MAGNESIUM

In a recent study, 54% of horses that underwent colic surgery had low plasma ionized magnesium concentrations. There was a statistical association between low ionized magnesium concentrations and ileus in these patients. Causes of hypomagnesemia include decreased intake, gastrointestinal losses (e.g., prolonged nasogastric reflux, malabsorption), alterations in distribution (e.g., due to endotoxemia), renal losses (e.g., prolonged administration of lactated Ringer’s solution or other magnesium-free fluids; hypophosphatemia; acidemia), and excessive sweating. In horses, severe hypomagnesemia can result in ventricular arrhythmias, muscle tremors, ataxia, seizures, and calcification of elastic tissue. Clinical manifestations of hypomagnesemia reported in humans include supraventricular tachycardia, atrial fibrillation, thrombosis, anemia, decreased muscle strength, increased nephrotoxicity of aminoglycosides, and sudden death. Hypomagnesemia can also result in hypokalemia that is refractory to potassium supplementation.

I supplement magnesium if it falls below 90% of the bottom of the normal range (i.e., <0.35 mmol/L [0.85 mg/dl]) because of the association with ileus, cardiac dysrhythmia, and gentamicin-induced nephrotoxicity. Intravenous magnesium sulfate is the most common way that magnesium is supplemented (see box on p. 17). Oral magnesium supplementation can be problematic because many preparations are either laxative (e.g., magnesium sulfate) or have poor bioavailability (e.g., magnesium oxide) when administered orally.

Hypermagnesemia developed in two horses with large colon impactions after oral administration of approximately 2 lb (1,000 g) of magnesium sulfate. These
horses developed flaccid paralysis with recumbency and tachycardia but responded to treatment with crystalloid fluids and calcium gluconate\(^9\) (see box on p. 17).

**CALCIUM**

Eighty-six percent of horses that underwent colic surgery had low plasma ionized calcium concentrations at admission.\(^6\) Possible causes of hypocalcemia include lactic acidosis, endotoxin-induced changes in calcium homeostasis, loss of calcium in sweat, and functional disturbances of the small intestine. Clinical signs of hypocalcemia in horses include synchronous diaphragmatic flutter, tetany, muscle spasm, and seizures. After colic surgery in horses, decreasing ionized calcium concentrations were correlated with the following changes on the electrocardiogram\(^6\):

- Increased heart rate
- Increased QT interval corrected for heart rate
- Decreased PR interval
- Decreased QRS interval

Although hypocalcemia has been associated with post-operative ileus in horses,\(^6\) this has not been formally investigated.

Routine calcium supplementation of horses following colic surgery is very controversial. Proponents suggest that calcium supplementation reduces the incidence of ileus. Opponents point to research in rats demonstrating increased mortality from endotoxemia with infusions of moderate doses of calcium, which increased the plasma calcium concentration from 4.6 to 4.9 mg/dl.\(^10\) I supplement calcium if the ionized calcium concentration falls below 0.9 mmol/L (3.6 mg/dl; e.g., 60% below the bottom of its normal range) because cardiac dysrhythmias have been demonstrated in horses with calcium concentrations less than 0.83 mmol/L (3.52 mg/dl).\(^11\) The ionized calcium concentration rarely falls below 0.9 mmol/L in colic patients\(^6\); therefore, treatment is not frequently required. Details of calcium supplementation are provided in the box on p. 17.

**SODIUM AND CHLORIDE**

Sodium and chloride disturbances rarely require specific treatment in horses with colic because treatment with balanced electrolyte solutions often results in reso-
olution of the disturbance. Hyponatremia (51% of horses) and hyperchloremia (74%) are the most common imbalances. Hypochloremia also occurs, especially in horses with strangulating small intestinal lesions in which chloride is lost in refluxed gastric acid. In species other than the horse, rapid correction of sodium deficits has been shown to cause demyelination of the pontine and extra pontine neurons, resulting in severe neurologic dysfunction. Whether this is a risk in horses has not been established, but judicious guidelines for intravenous treatment of sodium and chloride disorders are provided in the box on p. 17. Sodium bicarbonate must be used with caution in horses with respiratory dysfunction, hypocalcemia, and severe hypokalemia.

Sodium can often be successfully replaced in oral fluids. Some horses with hyponatremia preferentially drink electrolyte-supplemented water, which should be isotonic or slightly hypotonic. Although ideally the amount of salt to be added should be weighed, I find it more practical to measure it out in milliliters in a syringe barrel. The volume in milliliters given in the next two sentences should match the weight of the preparation of sodium chloride or bicarbonate being used. For concurrent hyponatremia and hypochloremia, 20 to 30 ml (20 to 28 g) of sodium chloride granules should be added per 4 L of water. For hyponatremia without hypochloremia, 40 to 50 ml (40 to 50 g) of sodium bicarbonate powder should be added per 4 L of water. Fresh water should always be provided in addition to electrolyte-supplemented water. These sodium-supplemented solutions may also be delivered by nasogastric tube. In foals, sodium chloride and sodium bicarbonate (1 tsp up to four times daily) may be supplemented orally by syringe. Mixing the sodium salt with yogurt makes a paste, which foals tend to retain better when given orally by syringe.

**CONCLUSION**

The potential clinical benefit of treating mild to moderate electrolyte disturbances in horses has not been examined. Electrolyte physiology, evidence from other species, and limited data from horses suggest that treatment of potassium and magnesium disturbances are most likely to be beneficial without risk of causing harm. Therefore, potassium and magnesium are the electrolytes that I supplement most frequently.

**REFERENCES**