Tutorial Article

Electrolyte disorders in horses with colic. Part 1: potassium and magnesium

K. E. BORER* AND K. T. T. CORLEY†

Royal Veterinary College, Equine Referral Hospital, Hawkshead Lane, North Mymms, Hatfield, Hertfordshire AL9 7TA, UK.

Keywords: horse; colic; electrolytes; potassium; magnesium

Introduction

Electrolyte disorders are common in horses with acute abdominal disease, and are a significant cause of morbidity. They are especially prevalent in horses treated surgically for colic, and may be present before, during and after surgery. In one study, 54% of horses admitted for surgical colic had preoperative serum ionised magnesium concentrations below the reference range and 86% subnormal serum ionised calcium concentrations (Garcia-Lopez et al. 2001). Electrolyte abnormalities are particularly prevalent in horses with strangulating obstructions of the intestine. These horses have lower preoperative serum concentrations of ionised magnesium and calcium than horses with nonstrangulating lesions (Dart et al. 1992; Garcia-Lopez et al. 2001). Horses with decreased preoperative ionised magnesium were also more likely to be subjected to euthanasia during surgery.

The role of electrolyte derangements in contributing to adverse clinical events in horses with colic is still being investigated. The association between cardiac electrical disorders and blood calcium, magnesium and potassium concentrations is well established in the horse (Harrington 1974; Glazier et al. 1979, 1982; Reef 1999) (Fig 1). Derangements in blood electrolyte concentration might also be associated with a longer hospital stay. Martin et al. (1994) reported that dogs with low serum magnesium concentrations were hospitalised for twice as long as those with a normal serum magnesium concentration. Conversely, Garcia-Lopez et al. (2001) did not find any correlation between serum ionised magnesium or calcium concentrations and length of hospitalisation, incidence of complications or survival in horses treated surgically for colic. The association between serum concentrations of potassium, calcium and magnesium with post operative ileus is still unknown in the horse. Dart et al. (1992) speculated that decreased serum calcium concentrations might contribute to ileus. Garcia-Lopez et al. (2001) found lower serum concentrations of ionised magnesium and calcium in horses that developed post operative ileus, than in those that did not. These decreased electrolyte concentrations may be due to decreased intake and increased loss of electrolytes in gastric reflux.

It is now possible to measure electrolytes using ‘patient-side’ portable analysers. The introduction of these cartridge-based systems has enabled results to be obtained quickly and easily when laboratory techniques are unavailable (Grosenbaugh et al. 1998). Different sensors are available to measure different combinations of electrolytes, blood biochemistry, PCV and blood gas tensions. The price of these hand-held devices is rapidly decreasing and they are now affordable for many private practices. The normal range for any electrolyte may vary to some degree between analysers. Therefore, horse specific normal ranges should be obtained from the manufacturers, if possible.

In Part 1, we discuss the importance of potassium and magnesium and Part 2 covers calcium, sodium, chloride and phosphorus.

Potassium

Potassium is primarily an intracellular ion, and uptake of potassium into cells is stimulated by increased circulating insulin concentration and β-adrenergic stimulation (Gennari 1998). The ratio of intracellular to extracellular potassium may be affected by the acid-base status. Hydrogen moves into cells during acidosis; and from the intracellular to extracellular fluid in alkalosis. In some acid-base conditions, potassium moves in the opposite direction to hydrogen to maintain electroneutrality (Corley and Marr 1998). Although erythrocyte potassium content has been used to estimate whole body potassium (Muylle et al. 1984), its accuracy has not been validated. Moreover, the extracellular potassium concentration (reflected in the plasma) is critical for neuromuscular transmission and therefore more relevant to clinical signs than whole body potassium stores (Rose and Post 2001).

*Author to whom correspondence should be addressed. †Present address: Anglesey Lodge Equine Hospital, The Curragh, Co. Kildare, Ireland.
Hypokalaemia

Fifty percent of horses are hypokalaemic after colic surgery (Protopapas 2000). Clinical signs of hypokalaemia are chiefly related to electrical conduction across the cell membrane. Hypokalaemia results in hyperpolarisation of the cell membrane (a more negative resting membrane potential) and therefore less chance of a positive increase in charge reaching threshold potential to result in an action potential (Schaer 1999). Symptoms of hypokalaemia can be mild and nonspecific, including muscle weakness and reduced gastrointestinal motility. The severity of symptoms is usually related to the speed of decrease in serum potassium. Severe hypokalaemia can result in impairment of respiratory function, but cardiac abnormalities are rare in patients with no pre-existing cardiac disease (Gennari 1998). Electrocardiogram changes that occur with hypokalaemia include increased P wave amplitude, reduced T wave amplitude, prolongation of the P-R and Q-T interval and arrhythmias such as atrial tachycardia (Fig 1: Reef 1999; Schaer 1999).

Hypokalaemia in horses with colic has been speculated to be associated with altered intake and absorption or excess loss from the gastrointestinal tract caused by diarrhoea (Nappert and Johnson 2001). Several other causes could also be involved. Hypokalaemia can result from increased losses via the kidneys (Gennari 1998). Chronic fluid therapy with lactated Ringer’s solution, which contains a low potassium concentration and may result in sodium induced diuresis, can result in hypokalaemia (Atkins 1999). Horses that are producing large volumes of gastric reflux will lose chloride ions, resulting in metabolic alkalosis and subsequent hypokalaemia (Gennari 1998). Many drugs can induce hypokalaemia, including β-adrenergic agonists, glucocorticoids, insulin, diuretics and antibiotics. Both penicillin and gentamicin, antibiotics commonly used after colic surgery, may induce hypokalaemia. In the case of gentamicin, this is an indirect action, by causing hypomagnesaemia (Gennari 1998).

Hyperkalaemia

Hyperkalaemia has been reported to occur in association with metabolic acidosis, due to potassium exchange for hydrogen ions across cell membranes. However, this is chiefly a phenomenon of mineral (inorganic) acidosis rather than lactic acidosis (Perez 1981; Graber 1993). In horses with colic, the predominant cause of acidosis is lactic acid accumulation, associated with poor tissue perfusion and hypovolaemia (Corley and Marr 1998; Nappert and Johnson 2001). We found a moderate inverse correlation between plasma potassium concentration and pH, but no correlation between potassium and lactate concentration, in jugular venous blood from horses with surgically treated colic (C. Adams and K.T.T. Corley, unpublished data). Hyperkalaemia is most commonly encountered in equine practice in foals that have a ruptured urinary bladder (Kablack et al. 2000).

ECG signs of hyperkalaemia include bradycardia, peaked T waves, small or absent P waves, widened QRS complexes and prolonged P-R intervals (Atkins 1999) as shown in Figure 1. These changes are indicative of reduced cardiac excitability, and may progress to ventricular fibrillation or cardiac arrest (Reef 1999). Muscle weakness can also be a feature of hyperkalaemia (Schaer 1999).

Magnesium

Magnesium plays a crucial role in many metabolic and cellular functions in the body, especially those involving adenosine
triphosphate (ATP) and the production of energy (Page et al. 1998). It is an important coenzyme for the sodium-potassium ATPase pump (Tso and Barish 1992). Abnormalities of the normal resting membrane potential can result from interference with the normal function of this pump, causing membrane destabilisation and hyperexcitability (Tso and Barish 1992; Martin et al. 1994). Magnesium is an essential cofactor in many enzymatic reactions in the body (Tso and Barish 1992). Magnesium also directly competes with calcium for some of its binding sites, allowing greater binding of calcium to enzymes in hypomagnesaemia. One such enzyme is phospholipase A2; increased calcium binding results in greater activity of this enzyme, which leads to the increased formation of eicosanoids, particularly thromboxane A2 (Gunter 1991), which may play a role in thrombophlebitis (Morris 1989).

Magnesium is absorbed from the intestine and filtered by the kidney. Only 5% of the magnesium filtered by the kidney is excreted, and the remainder is reabsorbed in the proximal tubule and ascending loop of Henlé (Tso and Barish 1992; Hamill-Ruth and McGory 1996). Ionised magnesium is the biologically active form of the cation (Tso and Barish 1992), and therefore is preferred to total magnesium for measurement of magnesium status. However, only 1% of total body magnesium is found in the extracellular fluid, with 50% intracellular and the rest in bone. This means that extracellular (serum) magnesium concentrations may not be representative of total body magnesium status (Fiaccadori et al. 1988; Hamill-Ruth and McGory 1996). Methods for measuring intracellular magnesium have been developed in man, using erythrocytes, mononuclear cells, bone or skeletal muscle cells (Fiaccadori et al. 1988; Tso and Barish 1992), but are expensive and not widely available. Measurement of intracellular magnesium concentrations in intensive care patients usually reveals a higher prevalence of magnesium depletion than measurement of serum concentrations alone (Olerich and Rude 1994). For example, one study found that 9.4% of humans in intensive care were hypomagnesaemic, but 47% had subnormal magnesium concentrations in quadriceps muscle cells (Fiaccadori et al. 1988).

A recent study in horses suggested that the best method of determining magnesium status was to determine the fractional excretion of magnesium in the urine. Although a 24 h urine collection provided the most accurate results, a ‘spot’ urine total magnesium fractional excretion was sufficiently accurate for clinical use (Stewart et al. 2004).

**Hypomagnesaemia**

Costa et al. (1999) surveyed 75 horses with gastrointestinal disease and found that 44% were hypomagnesaemic. Garcia-Lopez et al. (2001) found decreased ionised magnesium in 54% of colic patients on admission. Stephen et al. (2004) found 73% of horses with a small intestinal volvulus had an ionised magnesium concentration below the reference range at admission. These findings are similar to those in human intensive care patients, where studies have documented a prevalence rate varying between 20 and 65% (Olerich and Rude 1994; Hamill-Ruth and McGory 1996).

In many studies, hypomagnesaemia appears to be associated with increased morbidity and mortality rates, although the underlying disease may be responsible for both the lowered magnesium concentrations and the increased mortality (Olerich and Rude 1994). In one study, there was no relationship between ionised magnesium concentrations on admission and survival in horses with gastrointestinal disease (Garcia-Lopez et al. 2001). Relevant to the post operative colic patient, hypomagnesaemic rats had greatly increased mortality from endotoxaemia, which could be reversed by magnesium chloride administration (Salem et al. 1995).

Magnesium intake is reduced in most horses after colic surgery due to decreased food intake (Tso and Barish 1992). Renal and gastrointestinal losses constitute the most important causes of magnesium loss. Patients receiving large volumes of fluids, especially sodium chloride, without magnesium supplementation, may become hypomagnesaemic. The high renal sodium load inhibits reabsorption of magnesium in the loop of Henlé (Martin et al. 1994). Stress, and the subsequent release of glucose and adrenaline, may cause hypomagnesaemia (Tso and Barish 1992). Loop diuretics and aminoglycoside antibiotics are also associated with hypomagnesaemia (Olerich and Rude 1994). Horses with diarrhoea or large volumes of gastric reflux may lose large amounts of magnesium rich fluid (Tso and Barish 1992). Parenteral nutrition solutions are also low in magnesium and can result in a deficiency unless supplemented. Sweating also causes loss of magnesium from the body (Taylor 1996).

Hypomagnesaemia is often associated with other electrolyte abnormalities. Costa et al. (2004) found that 30% of hypomagnesaemic horses with gastrointestinal disease were also hypokalaemic, compared with 20% of normomagnesaemic horses. Hypokalaemia occurs due to inhibition of renal potassium transport, and this form of hypokalaemia may be refractory to treatment with potassium supplementation without concurrent magnesium supplementation (Fiaccadori et al. 1988; Olerich and Rude 1994).

Hypochloraeia can occur in association with hypomagnesaemia (Hamill-Ruth and McGory 1996). Admission data from horses with small intestinal volvulus demonstrated a 37% prevalence of hypochloraeia in hypomagnesaemic horses compared with 14% prevalence in horses with normal ionised magnesium concentrations (J.O. Stephen and K.T.T. Corley, unpublished data). Hypocalcaemia can occur as a result of inhibition of parathyroid hormone release by low magnesium concentrations (Olerich and Rude 1994).

In the horse, severe hypomagnesaemia can result in ventricular arrhythmias and also muscle tremors, ataxia, seizures and calcification of elastic tissue (Harrington 1974). An increased incidence of cardiac arrhythmias and myocardial ischaemia is also found in human patients with hypomagnesaemia, especially if this is associated with hypokalaemia (Olerich and Rude 1994; Hamill-Ruth and McGory 1996). These can vary from ectopic beats to atrial tachycardia and ventricular fibrillation (Fiaccadori et al. 1988;
Tso and Barish 1992). The frequency of ectopy was reduced in human patients receiving supplemental magnesium, compared with a control population (Tso and Barish 1992; Hamill-Ruth and McGory 1996). This may be due to the action of magnesium on the membrane potential and refractory period in the cardiac cells (Tso and Barish 1992). Hypertension can also occur in hypomagnesaemic patients (Fiaccadori et al. 1988).

Platelet aggregation is increased in the face of magnesium deficiency (Olerich and Rude 1994). Hypomagnesaemic patients often have poor respiratory muscle strength and bronchospasm, which can be relieved by a bolus of magnesium (Tso and Barish 1992). Seizures and central nervous system manifestations, such as restlessness and disorientation, are also reported (Tso and Barish 1992). Gastrointestinal motility is reduced, especially if hypomagnesaemia is associated with concurrent hypokalaemia (Schaer 1999).

**Hypermagnesaemia**

Hypermagnesaemia is less common than magnesium deficiency in intensive care patients. The prevalence reported in post operative colic patients (11–14%) (Costa et al. 1999; Protopapas 2000) is very similar to that reported in dogs (Barish 1992; Atkins 1999), resulting in hypotension and reflex asystole. The prevalence of ectopy was reduced in humans receiving supplemental magnesium (Tso and Barish 1992; Atkins 1999). Severe hypermagnesaemia results in respiratory paralysis and subsequent cardiac arrest (Bowen et al. 1970). Hypocalcaemia is common in horses with gastrointestinal disease (Dart et al. 1992; Garcia-Lopez et al. 2001) and this may increase the severity of signs of hypermagnesaemia.

**Prevention and treatment**

Most commonly, horses hospitalised after colic surgery will receive electrolytes intravenously together with routine post operative fluid therapy. However, these may not be in the appropriate proportions for the horse, and may not include some important electrolytes. For example, lactated Ringers' solution contains no magnesium or phosphate, and the amount of potassium is frequently inadequate to maintain plasma concentrations.

Horses that have access to oral food and water can be given electrolytes orally. Horses are often unwilling to drink electrolytes freely, when provided in a bucket of water. Nasogastric intubation is a practical way to administer electrolytes orally (McGinness et al. 1996). The horse must have a functional gastrointestinal tract and must be checked regularly for the presence of gastric reflux, indicating ileus. Up to 8 l of fluid can be given by nasogastric tube every 30 min to a 500 kg horse (McGinness et al. 1996). However, in the

**TABLE 2: Replacement guidelines**

<table>
<thead>
<tr>
<th>Measured value</th>
<th>Amount to add to 5 l bag of balanced electrolyte solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypokalaemia K &lt;2.5 mmol/l</td>
<td>Add 40 mmol/l (77 ml of 13 mEq/5 ml KCl) and 0.1–0.2 g/kg bwt per os (if appropriate)</td>
</tr>
<tr>
<td>K &lt;2.9 mmol/l</td>
<td>Add 30 mmol/l (58 ml of 13 mEq/5 ml KCl) and 0.1–0.2 g/kg bwt per os (if appropriate)</td>
</tr>
<tr>
<td>K &lt;3.2 mmol/l</td>
<td>Add 20 mmol/l (39 ml of 13 mEq/5 ml KCl)</td>
</tr>
<tr>
<td>K &lt;3.5 mmol/l</td>
<td>Add 10 mmol/l (19 ml of 13 mEq/5 ml KCl)</td>
</tr>
<tr>
<td>Hyperkalaemia K &gt;7.0 mmol/l</td>
<td>1) Ca gluconate 40% 0.5 mEq/kg bwt over 10 min 2) Dextrose 50% 2ml/kg bwt over 5 min ± regular insulin 0.1 u/kg bolus 3) Bicarbonate 1–2mEq/kg bwt over 15 min</td>
</tr>
<tr>
<td>K &lt;7.0 mmol/l</td>
<td>Diuresis with Hartmann’s (and, if clinically appropriate, 1mg/kg bwt frusemide)</td>
</tr>
<tr>
<td>Hypomagnesaemia</td>
<td>Mg sulphate 4–16mg/kg bwt per 5 l bag, and remeasure within 12 h.</td>
</tr>
<tr>
<td>Hypermagnesaemia</td>
<td>Ca gluconate 40% 125–250ml over 15 min</td>
</tr>
</tbody>
</table>

**TABLE 1: Approximate normal ranges and interference levels for electrolytes in post surgical colics**

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Normal range (mmol/l)</th>
<th>Treat if falls below (mmol/l)</th>
<th>Treat if rises above (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td>3.2–5.2</td>
<td>3.5</td>
<td>7.0</td>
</tr>
<tr>
<td>Magnesium (ionised)</td>
<td>0.45–0.65</td>
<td>0.35</td>
<td>1.3 or symptomatic</td>
</tr>
<tr>
<td>Magnesium (total)</td>
<td>0.66–0.95</td>
<td>0.4</td>
<td>1.6 or symptomatic</td>
</tr>
<tr>
<td>Magnesium (ionised)</td>
<td>0.45–0.65</td>
<td>0.35</td>
<td>1.3 or symptomatic</td>
</tr>
<tr>
<td>Magnesium (total)</td>
<td>0.66–0.95</td>
<td>0.4</td>
<td>1.6 or symptomatic</td>
</tr>
</tbody>
</table>

Tso and Barish 1992). The frequency of ectopy was reduced in human patients receiving supplemental magnesium, compared with a control population (Tso and Barish 1992; Hamill-Ruth and McGory 1996). This may be due to the action of magnesium on the membrane potential and refractory period in the cardiac cells (Tso and Barish 1992). Hypertension can also occur in hypomagnesaemic patients (Fiaccadori et al. 1988).

Platelet aggregation is increased in the face of magnesium deficiency (Olerich and Rude 1994). Hypomagnesaemic patients often have poor respiratory muscle strength and bronchospasm, which can be relieved by a bolus of magnesium (Tso and Barish 1992). Seizures and central nervous system manifestations, such as restlessness and disorientation, are also reported (Tso and Barish 1992). Gastrointestinal motility is reduced, especially if hypomagnesaemia is associated with concurrent hypokalaemia (Schaer 1999).
authors' experience, this volume can result in mild discomfort in some horses, even those with normal gastrointestinal motility. These solutions are easy to make and inexpensive. Ideally, slightly hypotonic or isotonic solutions should be provided to avoid drawing further fluid from the extracellular space into the intestinal lumen. Adding 30 ml of table salt crystals per 4 l of water results in an approximately isotonic solution.

Sosa León et al. (1998) reported the use of a concentrated paste of electrolytes to replace fluid and electrolyte deficits induced by frusemide administration. The paste was made up from sodium and potassium chloride (table salt and 'Lo-salt') and administered using feeding syringes. All horses were allowed ad libitum access to water after paste administration. The concentrated paste stimulated water consumption and improved the overall balance of sodium, potassium and chloride, compared to administration of water alone (Sosa León et al. 1998). Our subjective experience is that oral electrolyte pastes do not increase water consumption in post operative colic patients.

The approximate normal ranges for the electrolytes discussed are given in Table 1. However, it is not necessary, or indeed desirable, to maintain all the electrolytes within their normal ranges in post operative colic patients. Therefore, as a guide, we have produced electrolyte concentrations at which therapeutic intervention is likely to be necessary ('interference levels'), for use in our own hospital. These are also presented in Table 1. However, it is important to note that these may vary slightly with different analysers, and should always be used in context of the whole clinical scenario.

Guidelines for correction of these electrolyte abnormalities are given in Table 2. For all electrolyte abnormalities, it is important to realise that losses may be ongoing. Furthermore, there may be intracellular shifts, or accelerated excretion of exogenously administered electrolytes. For this reason, it is not appropriate to calculate a theoretical deficit and replace the calculated deficit. Such calculations assume that the horse is a chemistry flask, and do not acknowledge dynamic changes in absorption and excretion. It is far better to supplement the electrolyte at a standard rate, remeasure after a short interval, and adjust the rate of supplementation accordingly.

**Hypokalaemia**

Treatment of hypokalaemia involves potassium replacement, either intravenously or orally. Rapid administration of intravenous potassium can lead to hyperkalaemia, with its own attendant problems, and so potassium should be infused at a maximum rate of 0.5–1 mEq/kg bwt/h. Potassium chloride is the ideal replacement salt, especially in horses that are refluxing and concurrently hypochloraemic (Gennari 1998). High crystalloid flow rates result in increased urine production and kaliuresis, and therefore can make it harder to replace potassium intravenously. For this reason, oral supplementation is preferred for severe hypokalaemia, in horses in which the oral route is available. Suggested doses are given in Table 2. Horses that are also hypomagnesaemic may be refractory to potassium replacement therapy, unless the magnesium deficit is simultaneously corrected (Hamill-Ruth and McGorry 1996).

**Hyperkalaemia**

Treatment of hyperkalaemia can be accomplished in several ways (Table 2). Fluid therapy with sodium chloride should be instituted to promote kaliuresis. A combination of insulin and dextrose saline will promote cellular uptake of potassium. Calcium gluconate will negate the cardiac effects of hyperkalaemia (Reef 1999). Sodium bicarbonate has the least effect on potassium concentrations, but acts to alkalise the plasma and drive potassium intracellularly.

**Hypomagnesaemia**

Magnesium sulphate can be added to intravenous polyionic replacement fluids at rates up to 64 mg/kg bwt depending on the degree of hypomagnesaemia. After 4–8 h, serum concentrations should be remeasured.

**Hypermagnesaemia**

The treatment for hypermagnesaemia should include intravenous calcium, fluid therapy and loop diuretics, such as frusemide, to increase renal excretion of magnesium (Tso and Barish 1992; Henninger and Horst 1997).

**References**


ERRATUM

The article ‘Cor pulmonale in a horse with granulomatous pneumonia’ by Schwarzwald et al. was published in Equine Veterinary Education, Volume 18, pp 182-187. There was a typographic error on page 182: the final sentence of the Haematology and serum biochemistry section should have read “Serum cardiac Troponin I concentration was <0.15 ng/ml (reported reference value <0.3 ng/ml [Cornelisse et al. 2000]).” The editor wishes to apologise for any inconvenience that this may have caused.