How to Manage Severe Dehydration and the Exhausted Horse

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1. Introduction
By the middle of the 12th century, the biggest empire in history, the Mongol Empire, united land between the Arabian Sea, Indian Ocean, Pacific Ocean, and Black Sea. This legacy was the creation of the Mongol hordes led by Temujin, better known as Ghenghis Khan. Although China was disunited and the Mongols were ruthless, the biggest factor to which their meteoric success is attributed to their use of the horse. The Mongols were intrepid horsemen, giving them a fear-inspiring edge in warfare and the ability to cover long distances with speed.

Man’s ability to conquer has been immeasurably enhanced by the domestication of the horse throughout history. However, the willing and selfless nature of this incredible athlete results in vulnerability as it works beyond the point of normal fatigue and enters the dangerous zones of dehydration and exhaustion. This paper will explore the horse’s responses to exercise and why its willingness to work for us can result in life-threatening metabolic derangements.

Pathophysiology of Extreme Exercise

Muscular Work Generates Heat
Heat generated from muscular work must be dissipated for the muscles to continue their normal contraction/relaxation cycling. The principal form of heat dissipation is through evaporative losses from the skin, with a lesser contribution from the respiratory tract. Hence, the delivery of heat to the body surface is a prerequisite for heat dissipation. Two key factors necessary for this are the circulatory delivery of heat to the surface and the ability to dissipate it via the evaporation of sweat.

Sweat
Equine sweat is isotonic or mildly hypertonic relative to plasma. Substantial amounts of sodium, potassium, and chloride are lost in sweat. Smaller but nonetheless significant amounts of calcium and magnesium can also be lost. The concentration of these major ions in equine sweat has been reported to be as high as 135 mmol/L for sodium, 42 mmol/L for potassium, and 158 mmol/L for chloride.\(^1\) As such, it is easy to see how massive amounts of chloride, sodium, and potassium can be lost in the active horse. The potassium concentration is particularly striking, given it is >10-fold the concentration of plasma; however, intracellular potassium reserves can buffer this loss reasonably well. The loss of so much chloride leads to the retention of bicarbonate by the kidneys. This chloride loss and bicarbonate retention, coupled with the renal loss of hydrogen...
ions (see later), leads to hypochloremic metabolic alkalosis—the hallmark metabolic derangement of strenuous, prolonged exercise as a consequence of sweat loss. The concomitant loss of large volumes of water in sweat, up to 10 to 15 L/h, coupled with the loss of these ions, results in significant volume loss and dehydration accompanying the hypochloremic metabolic alkalosis.

Red blood cells and muscle are accessible sources of the rich body store of intracellular potassium. Plasma potassium concentrations typically increase during exercise. However, when prolonged depletion of sodium occurs, it is retained by the kidneys at the expense of hydrogen and potassium ions, and hypokalemia can occur. Hypokalemia can lead to a decrease in nerve depolarization thresholds and neuronal hyperirritability. This manifests as flaccid paralysis of muscles, gastrointestinal (GI) motility dysfunction, and/or rhabdomyolysis. Calcium loss can be actual and relative. Actual losses occur via sweat, and relative losses are due to decreased availability as a function of increased protein binding under alkalotic conditions. Hypocalcemia decreases the electrical depolarization threshold in nerves, resulting in their irritability. Another clinical manifestation of calcium depletion is synchronous diaphragmatic flutter (SDF, “thumps”). During “thumps,” the phrenic nerve depolarizes as a result of electrical activity in the atria. The diaphragm then contracts in rhythm with the heart rate, leading to inefficient pulmonary ventilation. SDF is exacerbated by hypokalemia. Hypomagnesemia is a possible but a less well-diagnosed sequela to prolonged sweating and dehydration. Hypomagnesemia causes an increase in the release of acetylcholine at the neuromuscular junction, leading to muscular spasms and tetany. Equine sweat also contains a surfactant protein, latherin, to promote its spread over a larger surface area and facilitate evaporation. This is what gives equine sweat its unique foamy quality. The sweat glands are largely under the control of circulating catecholamines, but treadmill evidence supports that their control is also related to internal carotid blood temperature (a reflection of brain-afferent blood temperature). In the same set of experiments, it was found that fatiguing exercise increased the volume of sweat produced.

The efficiency of sweat evaporation is dictated by ambient climate conditions. Hot and humid conditions are the least favorable for sweat evaporation. Inefficient sweat evaporation causes the horse to sweat more. This does not have the desired effect; instead, it increases fluid losses up to 10 to 15 L/h with ineffectual cooling. Acclimatization to exercise can alter the content of sodium in the vascular compartment and increase plasma volume. Research is ongoing in determining the effect of conditioning training in more favorable cool, dry conditions. It appears that acclimatization to exercise under cooler conditions can reduce total sweat loss and decrease sweating threshold. However, it is recognized that a thorough exercise training program together with a period of acclimatization to climate can invoke greater thermoregulatory adaptation to exercise in hot, humid conditions. This suggests that horses may be better off being conditioned under conditions favorable for sweat evaporation even if they are expected to perform in hot and humid conditions.

**Hyperthermia**

Inefficient dissipation of metabolic heat energy results in hyperthermia and a dangerous rise in body core temperature. Horses with anhydrosis—even partial anhydrosis—are particularly susceptible to this. Dehydration and electrolyte imbalances increase the risk of thermoregulatory failure, which may result in vascular collapse, circulatory shock, disseminated intravascular hemolysis, flaccid muscle paralysis, multiple organ failure, and possibly death. It is worth noting that certain transportation conditions or poorly ventilated barns can increase the risk of hyperthermia in horses suffering from anhydrosis.

**Perfusion**

The initial bodily response in the effort to maintain appropriate perfusion to the working musculature is to shunt circulation away from nonobligate tissues, such as the splanchnic and renal capillary beds. This is rapidly initiated at 50% to 60% of maximal oxygen consumption (VO2max), before any significant sweating has commenced. This initial shift is not a result of fluid and electrolyte balances but rather the “stress of exercise,” mediated through the endocrine system and nervous system. Catecholamines play a significant role in this response, not surprisingly in the “fight, fright, and flight” responsive horse. This level of oxygen consumption coincides with the point at which increases in renal nerve activity, plasma renin activity, and catecholamines are detectable, and renal vasoconstriction occurs. With exercise, there is an absolute and a relative decrease in renal blood flow. The kidneys receive a reduced percentage of cardiac output, going from a reported 23% to 6% of the cardiac output. If fluid losses are not replaced, then circulating renal perfusion suffers further with intense or prolonged submaximal exercise.

Atrial natriuretic peptide (ANP) is a hormone secreted from the atria in response to atrial wall stretch. Exercise increases cardiac filling pressure, which increases the secretion of ANP in a linear fashion with the increasing exercise intensity. Along with a host of other neuroendocrine factors, ANP plays a major role in maintaining blood pressure and regulating blood flow during exercise. It also inhibits the excretion of vasopressin, renin, and aldosterone, further influencing vascular homeostasis. It is a potent vasodilator that is thought to decrease vascular resistance in the working mus-
cle to accommodate the increased arterial pressure. Sodium excretion is promoted during the initial phases of exercise, which is thought to be responsible for the initial increase in urine production at the beginning of exercise and post-exercise diuresis. Diuresis would normally lead to an increased plasma osmolality, leading to increased thirst and therefore water consumption. However, in strenuous or prolonged exercise conditions, the loss of hypertonic sweat leads to plasma hypotonicity—hypotonic dehydration. This actually reduces the physiologic drive to drink water even in the face of dehydration. This is an important point to remember—exercise can suppress the desire to drink.

The disproportionately high loss of chloride in sweat leads to the kidney conserving bicarbonate ions to maintain acid-base homeostasis. The kidney also tries to preserve sodium, and as such, it exchanges the excretion of sodium ions for hydrogen ions in the tubules. This results in the production of acidic urine. It is termed *paradoxic aciduria* because it flies in the face of the ongoing metabolic alkalosis.

Colic can result from ileus caused by electrolyte and blood perfusion disturbances and from dehydrated ingesta forming impactions. Ileus can develop as the horse becomes sequentially more dehydrated and electrolyte-deranged, but it can also occur during the rehydration/reperfusion phase. As such, care must be taken to consider this when using the oral route for rehydration therapy. If this route is chosen, frequent checking for the accumulation of enterogastric reflux is indicated.

**Exhaustion**

In the exhausted horse, the effect of excessive fluid and electrolyte loss through sweating and inefficient heat dissipation not only leads to hypochloremic metabolic alkalosis, hyperthermia, and glycogen depletion but can lead to hypoglycemia, circulatory collapse and shock, pulmonary edema, laminitis, activation of the clotting cascade, rhabdomyolysis, and renal damage. In addition to hypoperfusion, renal damage can be exacerbated by oxidative damage from myoglobinuria and the formation of intratubular crystals.

Clinical Signs in the Exhausted Horse

Every case is judged individually and will not necessarily demonstrate all of the following signs. Horses frequently show depression and disinterest in their surroundings and in drinking or eating. They may still be dripping with sweat or have dried sweat on them. If the conditions are still hot, they may have stopped sweating, which is a sign of exhaustion. This can often be judged after they have been washed down, the weather is still hot, but they are not sweating. They have an elevated heart rate and respiratory rate even after the cessation of exercise. Their rectal temperature may be increased or decreased. A decreased rectal temperature may be due to hypoperfusion or air in the distal rectum from a decreased anal tone, which is a sign of exhaustion. Pulse pressure will be decreased whenever the pulse is palpated. It will feel “hollow” to the touch and will require the most delicate of touches to consistently feel. The author usually compares the pulse quality in the palmar digital arteries, transverse facial artery, and facial artery. Repeated palpation is a good albeit subjective way to assess the improvement of perfusion. Mucus membranes are tacky or dry, with a prolonged capillary refill time (>3 seconds). If the membranes are red and capillary refill time is very short (<1 to 2 seconds), this can mean that capillary sludging is occurring as a result of endotoxic shock. With the head in a normal position, jugular distention may be decreased in severely dehydrated horses. In euvoletic horses, digitally occluding the jugular groove will result in distention of the vein proximal to the occlusion (i.e., within 2 to 3 seconds). If it fills more slowly than that, the horse is dehydrated. Skin tent is an insensitive technique to assess mild dehydration, but in cases in which a skin pinch on the neck leads to a skin tent, the horse is most certainly dehydrated, and, in the opinion of the author, this would correlate with a 7% to 8% or more dehydration. Borborygmi can be decreased as a result of not having eaten recently but also can be due to dehydration/electrolyte derangement.

Horses with synchronous diaphragmatic flutters (“thumps”) have synchronous heart and respiratory rates but have the outward appearance of “hicupping,” for want of a better description.

Any horse with severe dehydration and exhaustion may be unwilling to move and may walk with a stiff gait. Before encouraging the horse to move, palpate his musculature, because it may be very hard and painful, which indicates the onset of myositis and rhabdomyolysis. As the horse becomes better perfused with treatment, this pain may not abate as muscle damage manifests itself, but horses will usually walk steadily, albeit stiffly, if encouraged to do so.

**Therapy**

The following therapy suggestions are not exhaustive but are designed as a guide for initial treatment.

**Exercise**

Immediately cease all exercise. If at a competition, request that the rider dismount and walk the horse to the treatment area if possible.

**Physical Examination**

Routine physical examinations are essential. Assessments of hydration will be helpful but remember to piece them together because isotonic or hypotonic dehydration may result in normal electrolyte values, or normal urine specific gravity in mild cases, in which assessment is possible. The signs listed above will be demonstrated, and comparing initial
and sequential skin turgor, capillary refill time, and pulse quality will be invaluable to monitor the progress of treatment. Be sure to check skin turgor in several places; the upper eyelid can be a particularly useful place to evaluate it accurately. The soft skin of the upper palpebra is more supple and in eu-

venomous horses will tent just for a few seconds after being pinched gently between the thumb and forefinger.

With the intervention of fluid therapy, horses begin to respond to their owners and surroundings, often searching for something to eat or being anxious to find bedding so they may urinate comfortably. Providing an area with some kind of grass or bedding can be important to encourage horses to urinate once fluid therapy has commenced.

**Hyperthermia**

Remove all wet blankets, saddle pads, and leg wraps. Aim to get the horse’s temperature down to just above normal. Monitor temperature every 15 minutes to be sure the horse does not become hypothermic, and keep checking it for the next 60 to 120 minutes. Use copious volumes of cold water all over the body; then scrape it off to prevent it from becoming an insulating layer as it warms up to body temperature.\(^{12}\) Repeat the process continuously to optimize heat removal. Use a fan to increase airflow and aid in evaporation, in addition to keeping the horse in the shade. The use of ice and ice water was once thought to cause peripheral vasoconstric-

the horse in the shade. The use of ice and ice water flow and aid in evaporation, in addition to keeping

optimize heat removal. Use a fan to increase air-

tion and hamper cooling efforts. This has been found not to be the case.\(^{12}\) Iced water is useful, but the author prefers to add isopropyl alcohol to a bucket of ice and water as the cooling fluid (approximately one part alcohol to three parts ice/water).

**Hematology**

If possible, serum electrolytes, serum chemistries, and a complete blood count (CBC) should be obtained. Serial monitoring of electrolytes and creat-

inine/blood urea nitrogen (BUN) will provide useful information regarding prognosis if renal damage should be an issue. Typically, this manifests over the first few days. However, assume a hypochloremic metabolic alkalosis if you are called to a dehy-

drated horse that has been exercising hard, or for a long time, in the absence of clinical chemistry results.

Serial complete blood counts may reveal leucopenia as a result of GI hypoperfusion. In cases of leucopenia, the author uses a broad-spectrum antimicrobial drug until the white blood cell count has returned to normal levels. This author considers a total white blood cell count of <2500 cells per micro-

liter as immunocompromised, particularly if the neutrophil count is <1200 cells per microliter. Aminoglycosides should be avoided in dehydrated horses or those with renal compromise.

Cardiac troponin I concentrations can be helpful to determine cardiac muscle compromise. Hepa-

rinized samples should be stored on ice for analysis, which can sometimes be done at the local human hospital. Normal ranges vary per machine, but normal ranges are typically 0 to 0.06 ng/mL.\(^{13}\) Typical recommendations for abnormally high values include rest and possibly the use of modest doses of corticosteroids. However, before using cortico-

steroids in such cases, consultation with a cardiolo-

gist is prudent in addition to performing and jointly analyzing an electrocardiogram (ECG).

**Fluid Replacement**

The most immediate need is gaining venous access by placing an intravenous catheter in one or both jugular veins. A 12-g catheter in one jugular vein and a 14-g catheter in the other jugular vein is the author’s preference (once immediate fluid replacement therapy has been successfully performed, the 12-g catheter is removed).

Severely dehydrated horses may need 60 to 80 L of intravenous isotonic fluid therapy over a 6- to 12-hour period (~500-kg horse). Fluid rates of 10 to 20 mL/kg are easily tolerated by horses (5 to 10 L/h, ~500-kg horse). The use of hypertonic saline is contraindicated because fluid losses have occurred in all body compartments, and so this would simply be adding large amounts of sodium and chloride without helping perfusion. If possible, avoid alkalizing fluids such as lactated Ringers solution. However, do not forego giving such fluids if the alternative is no fluids or an insufficient volume of fluids. Remember: the dumbest kidney is smarter than the smartest internist. The author and others\(^{14}\) have successfully used lactated Ringers solution to rehydrate severely dehydrated hypochloremic alkalo-

tic horses with no ill effects. Perfusion is much more important than the type of fluid.

The first choice for fluid replacement therapy would be non-lactated polyionic isotonic intravenous fluid\(^{6}\) or 0.9% normal saline with the addition of a modest amount of potassium (10 to 15 mEq/L). This level of potassium supplementation to 0.9% saline will not overload the body if one considers that potassium should not be added at >0.5 mEq/kg per hour. Lactated Ringers solution is the next choice but it has, as the name implies, lactate, the addition of which is not desired, and it is more alkaline. Lactated Ringers solution also has a lower sodium concentration at 130 mEq/mL versus 140 mEq/mL in non-lactated polyionic isotonic intravenous fluid\(^{6}\) and 154 mEq/mL in 0.9% sodium chloride. As already pointed out, perfusion is more critical than the exact composition of the fluid, provided it is isotonic.

To treat SDF, the slow administration of 100 to 300 ml of 23% calcium borogluconate solution diluted in intravenous fluids should be administered. Gastrointestinal (GI) motility can be affected even if the horse is not showing clinical signs of SDF. The author supplements the horse with calcium if the ionized calcium value is <1.5 mmol/L. If labo-
ratory results are not available, supplementation of each liter of saline with 10 to 20 mL 23% calcium gluconate is recommended.

Oral fluid replacement can be aggressive, provided the GI tract is functional and before every dose the horse is checked for enterogastric reflux (see below). A handy formula for an oral therapy electrolyte solution for the severely dehydrated horse is provided by Whiting as follows: 1 L tepid water, 2 tbsp (37g) NaCl, 1 tbsp (18 g) KCl or a mixture of NaCl/KCL (Lite salt). Oral rehydration per nasogastric tube can be safely delivered as 5 to 7 L of solution as a bolus every hour, provided reflux is not present.

It is worth noting that excessive diuresis is metabolically demanding on the kidney, and rehydration should be effective but not excessive. Urine specific gravity measurements in combination with clinical assessment can be very helpful in the normally functional kidney to avoid excessive diuresis. Equine urine is isothenuric at approximately 1.014 to 1.015. The author uses the range 1.018 to 1.24 as a goal in the rehydrated horse. When this urine specific gravity is achieved, aggressive hydration efforts can be reduced. Excessive diuresis can cause renal medullary washout. However, in a horse with obvious renal involvement (hematuria, pigmenturia, elevated creatinine), fluid administration must provide ongoing diuresis but avoid the development of secondary sequelae such as pulmonary edema. Referral of exhausted horses to a hospital setting is appropriate when horses fail to urinate after 40 or so liters of isotonic intravenous fluids; maintain or develop respiratory distress; develop cardiac arrhythmias; develop signs of colic; or do not become more responsive to their environment after a couple of hours of therapy.

**Gastrointestinal Tract**

It is important to check for enterogastric reflux in any horse that has suffered severe dehydration, and it is essential should enteral fluid therapy be used. Enteral fluid therapy can be economical and efficient. Up to 8 L per hour can be administered by nasogastric tube, provided that the GI tract is functional. Exertional ileus is manifest within hours of the cessation of exercise or during strenuous exercise when signs of colic are demonstrated. With fluid therapy and electrolyte correction, it usually abates, but frequent assessment for enterogastric reflux or the escalation of abdominal discomfort is critical.

Reperfusion injury causing GI dysfunction may occur 1 to 2 days after rehydration. It frequently manifests as GI stasis, causing small intestinal ileus and/or large intestine impaction colic. GI damage may cause endotoxemia, which will manifest as an elevated heart rate, poor peripheral perfusion, cool extremities, increased serum lactate levels, GI stasis, and colic. Anti-endotoxins drugs such as intravenous flunixin meglumine (0.5 to 1 mg/kg IV q 12 to 24 hours), fresh frozen plasma and/or polymyxin B (3000 IU/kg IV q 8 to 12 hours), and pentoxyfilline (8.5 mg/kg PO q 12 hours) may be used. However, caution should be used if using nephrotoxic drugs such as flunixin and polymyxin B in the dehydrated horse.

Exertional ileus is typically readily resolved with correction of fluid and electrolyte balances over 24 to 36 hours. Management of colic pain as a result of ileus, and after appropriate colic workup and gastric decompression with a stomach tube, is best managed with butorphanol (0.01 to 0.02 mg/kg IV PRN: 5 to 10 mg for a 500-kg horse) and xylazine (xylazine 0.3 to 0.5 mg/kg IV: 150 to 250 mg per 500-kg horse). The use of nonsteroidal anti-inflammatory drugs such as flunixin meglumine should be kept to a minimum where possible.

**Laminitis**

Laminitis may occur in the exhausted horse. The cause of laminitis is as yet undetermined and probably multifactorial. In exhausted or endurance horses, it is likely that there is a contribution from dehydration and direct exercise-related laminar injury. The author and other practitioners do not hesitate to use ice therapy on feet if the horse is at risk to develop laminitis. Unfortunately, determining those at risk remains challenging. Whiting noted that in 25 horses presented to the Dubai Equine Hospital there was no correlation between those who developed laminitis and the severity of exhaustion or leukopenia. More often, an abnormal hoof growth ring will be seen that corresponds to the timing of the stressful event.

**Rhabdomyolysis**

Rhabdomyolysis may manifest clinically as a horse being stiff or reluctant to walk, with firm, painful musculature on palpation. In the exhausted horse, phenothiazine derivatives, such as acepromazine, should be avoided due to their vasodilatory effects with a subsequent decrease in blood pressure. Pain can be managed with drugs such as butorphanol (0.01 to 0.02 mg/kg IV PRN: 5 to 10 mg for a 500-kg horse) and modest doses of the α-2 agonist xylazine, whose deleterious effects on blood pressure are less pronounced (xylazine, 0.3 to 0.5 mg/kg IV: 150 to 250 mg per 500 kg horse). Nonsteroidal anti-inflammatory agents should be avoided or used very sparingly until satisfactory rehydration and renal perfusion has been achieved (for example, the horse is starting to urinate normally and produce a relatively normal urine specific gravity of 1.018 to 1.025). The use of dimethyl sulfoxide (DMSO) is typically avoided by this author because its efficacy is not truly proven and it has a diuretic effect even when given at the appropriate dilution of <10% vol/vol.

Pigmenturia can be easily diagnosed by urine dipstick, but not by the naked eye in all but the worst cases. The determination of whether the pigment
is myoglobin or hemoglobin requires a laboratory analysis, but a handy way to triage can be done by the practitioner. Although not an absolute, the author assumes that in the absence of pink, hemolytic serum, the pigment seen on a dipstick is myoglobin. This simply helps you to think about the two pigments and be aware of their potential to cause damage. Myoglobin can directly damage the kidney by membrane oxidation as a function of hydroxyl radical production. It can also polymerize and form intraluminal crystals in the renal tubules. If pigmentation is seen and the horse is well rehydrated, then administration of mannitol can help to draw fluid into the renal tubules to effect dissolution of the crystals (0.5 to 1 g/kg IV over 5 minutes).

Summary

Hypochloremic metabolic alkalosis results from severe dehydration and excessive sweating. Recognition of the metabolic and vascular changes in the severely dehydrated horse is critical to institute prompt and appropriate therapy. The mainstay of treatment is aggressive fluid therapy and mitigation of heat stress. Successful fluid resuscitation is usually very rewarding, with horses usually making remarkable and complete recoveries. Treating short-term complications such as SDF, or longer-term complications such as ileus, also usually results in successful outcomes, and only in severe cases do life-threatening conditions such as multiorgan failure or laminitis occur.

References and Footnotes


**Additional Reading**


