The Practical Diagnosis and Treatment of Metabolic Conditions in Endurance Horses

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INTRODUCTION

Perhaps the greatest tests of a horse's athletic ability and "heart" are the long distance trail ride competitions and combined training events. In no other equestrian competition is a horse's endurance and stamina so challenged. Long distance trail rides are generally grouped into two classifications: competitive and endurance rides. In competitive trail rides, horse and rider cover from 25 to 100 miles in one to three days, depending on the specifications of each ride. Generally, 30 to 40 miles are covered in six to seven hours. Horses are judged on three criteria: soundness, condition, and attitude. The real test of the ride is the rider's ability to pace his horse during a long distance ride over varied terrain in an allotted time.[1] Endurance horses, on the other hand, win the competition simply by being first across the finish line in good condition. Endurance rides are 50 to 200 miles in length over a time of one to two days. No minimum time is set, but a maximum ride-time to prevent over-exertion of a horse is required.[1] Environmental parameters on these rides can range from 100–120°F in the shade, with high relative humidity, to significantly milder conditions.[2] At the Tevis Cup, an endurance ride, horses climb 500 meters over a distance of 2.5 kilometers at high environmental temperatures.[2]

Combined training, otherwise known as three-day eventing, is a test of a horse's obedience, training, athletic ability, and intelligence. This three day test starts with a dressage test on day one. This is a series of schooling movements performed in an arena which tests the horse's training, obedience, and flexibility. Day two is the Speed and Endurance test made up of four phases covering a total of fifteen to twenty miles in a maximum time limit. In advanced competitive levels, Phase A is Road and Tracks, consisting of a warm-up of three to four miles at a trot and canter over easily-covered ground. Phase B is the Steeplechase, involving a course with two miles of large, imposing fences cleared at a gallop. Phase C is again six to eight miles of Road and Tracks covered at a trot. This phase allows the horse to recover before the final phase. Phase D is Cross-Country, the most grueling phase of the three-day event. The horse and rider cover three to five miles at a slow gallop and jump twenty-two to thirty-five large, complicated fences. After completing this, the horse must trot out sound on day three and complete the Stadium Jumping test. This set of large fences tests the horse's ability to compete successfully after the Cross-Country.[1]

In these endurance competitions, horses undergo periodic veterinary checks to assess the horse's ability to continue. The veterinarian's responsibility in these competitions is to ensure the health of the horse, to provide emergency care, and to advise riders on the judicial use of their horse to improve chances of completing the competition.[3]

The veterinarian must perform these duties under less than ideal conditions. Especially at competitive/endurance rides, horses may need to be treated miles away from a veterinary clinic, sometimes even in places where only riders on horseback can reach them. The routine tests many practitioners rely on for diagnosis and treatment, CPK levels for azoturia, lactate levels for colic, total protein and PCV for dehydration estimation, and electrolyte or acid/base levels for metabolic evaluation, are simply not available. At these com-

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petitions a veterinarian must rely totally on his/her ability to recognize a disease condition and treat it rapidly and effectively without the help of modern technology.

A discussion of the practical field diagnosis and treatment of dehydration, synchronous diaphragmatic flutter, exhausted horse syndrome, and exertional rhabdomyolysis, four commonly occurring conditions in endurance athletes, will be detailed. For simplicity's sake, the endurance trail horse shall serve as the representative of the three types of endurance athletes since this horse covers the longest distance and is therefore more prone to these conditions.

DEHYDRATION

Predisposing factors

When a horse is ridden over 50 miles per day under various environmental conditions, it suffers some degree of dehydration. The amount of dehydration depends upon temperature, humidity, and the rate and amount of work done by the horse. The health status of the animal also plays a role in the amount of dehydration it suffers: any fluid or electrolyte imbalances already present will influence the onset of significant dehydration.[2] Weight losses of 10–15 kg/hr have been recorded in endurance horses competing at temperaures of 32°C (90°F).[2] Animals severely stressed during rides can have fluid losses of 40–50 liters.[2] A horse showing severe dehydration is usually excused from the ride since, if exercise continues, it will likely develop more severe metabolic problems.[2]

Dehydration can be evaluated as a percent of body-weight loss by observing the following criteria:

1. Mild dehydration, approximately 3% b.w. loss: decreased urine output, dry mouth and mucous membranes, slight decrease in skin elasticity.
2. Moderate dehydration, approximately 5% b.w. loss: marked loss of skin elasticity, shrunken eyes, weakness, fever, weak pulse, low blood pressure from decreased plasma volume.
3. Marked dehydration, approximately 10% b.w. loss: circulatory failure from severe plasma volume depletion.

Scant, dry feces and serious electrolyte imbalances also accompany severe dehydration. A 7–10% loss of body weight is equivalent to a loss of 30–40 liters of fluid.[2,4,5,6]

Pathophysiology

In order to effectively treat severe dehydration and the concurrent electrolyte deficits, it is important to understand what changes occur in the body causing this condition.

Exercise for extended periods of time, such as endurance rides, may require energy usage of 10–20 times the basic metabolic rate, equivalent to 3000–6000 kcal/hr. This energy required for muscular activity is supplied by the metabolic oxidation of lipids. The expenditure of this energy results in the generation of massive quantities of heat. If this heat is not dissipated, body temperature can exceed 106°F. Methods of heat loss include radiation, convection, conduction and evaporation. With the high temperatures usually encountered on many endurance rides, evaporation of sweat is the primary means of thermoregulation. Body surface, thickness of hair coat, wind, barometric pressure, humidity, and temperature all affect the evaporation of sweat.[3,5] If the environmental temperature (°F) + relative humidity (%) value is less than 130, the horse's thermoregulation system will adapt and dissipate heat effectively. Usually, only excessively muscular or obese horses will not be able to adapt to this level of temperature and humidity. With a temperature + humidity value greater than 140, horses rely more on sweating to dissipate heat. Heat loss by radiation is less effective. At levels of temperature + humidity greater than 150, especially with humidity greater than 50%, the evaporative cooling mechanism is compromised. At temperature + humidity values greater than 180, the horse's cooling mechanism is no longer effective and any exercise can only be maintained for short periods of time before body temperature exceeds 105°F. Sweating under this amount of environmental heat stress does little to cool the body.[2,3]

The fluid component of sweat comes directly from the extracellular fluid compartment (ECF). A 1000 lb. horse has a total body water volume of approximately 300 liters comprised of 100 liters as ECF and 200 liters in the intracellular fluid compartment (ICF). The ECF and ICF differ in electrolyte composition, but are equal in osmotic concentration. Water is freely diffusible between the two compartments. The principle ion in the ECF is Na^+; the principle ion in ICF is K^+.[5] Sweat is primarily composed of Cl^- with significant amounts of Na^+, K^+, Ca^{++}, and possibly some

Mg^{++}.[7] However, it is the Na^+ depletion which is the most serious of the electrolyte imbalances and poses the most life-threatening problem (Fig. 1).[5]

The Na^+ content in the body, approximately 14,000 mEq, is the primary determinant of ECF volume. With significant amounts of Na^+ being lost in sweat, the ECF volume is decreased, thereby decreasing plasma levels and circulating blood volume. With severe dehydration, cardiovascular compromise occurs because of this decrease in blood volume. A decrease in plasma volume contributes to decreased tissue perfusion and impaired renal function progressing to partial shut down.[5]

The decreased Na^+ in the plasma signals the kidneys to conserve Na^+ and excrete H^+ and K^+ in exchange.[2,7] It also will cause the horse to become thirsty. In severely dehydrated horses, the amount of Na^+ and electrolytes lost causes the plasma to actually be hypotonic, which is why these horses do not appear to be thirsty.[2]

The H^+ lost in urine due to exchange for Na^+ is one cause of metabolic alkalosis seen in severely dehydrated horses. The high amount of Cl^- lost through sweating also contributes significantly to this alkalotic state. Cl^- levels may be as low as 80 mEq/l or less.[5] The kidney creates a decreased serum [K^+] due to its exchange for Na^+. K^+ in the ICF exchanges with H^+ in the ECF to correct the deficiency. This further loss of H^+ also complicates the alkalosis.[3,5,7]

In summary, significant losses of Na^+ from ECF causes decreased plasma volume increased excretion of H^+ and K^+ from the kidney, which further leads to alkalosis. A decrease in Cl^- due to sweating is also a primary cause of the alkalosis.

Treatment

The degree of dehydration needs to be evaluated using the previously discussed parameters. Horses only moderately dehydrated may do well if allowed to drink small amounts of cool water. However, for horses dehydrated to a significant degree, IV fluids are indicated. Severely dehydrated horses may have deficits of more than 40 liters.[2,5] Lactated Ringer's (LR), isotonic saline or a balanced electrolyte solution should be given IV at a rate of 30–40 ml/kg/hr (12–16 l/hr) initially. Potassium given IV should not exceed 20 mEq/l or 1.5 mEq/kg/hr.[3] When standard electrolyte solutions are not available, 2 tablespoons of NaCl in one gallon of distilled water approximates physiologic saline.[3] Often using IV catheters in both jugular veins is necessary to deliver the large fluid volumes. The horse should be allowed to drink small amounts of water if interested; otherwise, passing a stomach tube and pumping 5–10 l of fluids every 30–60 minutes is helpful. The oral solution should have approximately 40 g NaCl and 30 g KCl for every 10 l of water. Hypertonic solutions should not be used since they will draw fluid into the GI tract, complicating the dehydration.[3] As for any gastric intubation, with any evidence of GI discomfort, fluid pumping should cease. Fluids are also absorbed from the colon, so cool water enemas may also be used.[2,3]

The principal concepts the veterinarian should realize with a severely dehydrated horse are that it is alkalotic and not acidotic and that *large* volumes of fluids are required to restore its fluid and electrolyte balance.

SYNCHRONOUS DIAPHRAGMATIC FLUTTER

SDF, commonly known as "thumps", is the synchronous contraction of the diaphragm with the heartbeat. In the absence of other concurrent disease, SDF is not a life-threatening condition, but does indicate a metabolic imbalance, which if allowed to progress, may become dangerous. A horse which exhibits the symptoms of SDF should be withheld from competition.[2]

SDF may develop as soon as 20–30 minutes into the ride. There appears to be no sex, age or breed predilection. Horses that have a history of "thumps" may or may not ever have another episode.[2]

Clinical Signs

Observation of the horse reveals spasmodic contraction in the flank area either unilaterally or bilaterally. Auscultating the heart while lightly palpating the flank area shows the synchronicity of the two. The diaphragmatic contractions may range from barely palpable to spasms that move the whole body. These contractions may be continuous or intermittent.[2,8]

Pathophysiology

SDF has been noted to occur in man,

canines and horses. It is associated with phrenic nerve conditions: trauma causing pressure on the nerve and congenital malpositioning of the nerve. Alkalosis and decreased serum Ca^{++} are also associated with SDF.

The phrenic nerve passes over the pericardium next to the atria. It is thought that SDF is caused by a hypersensitivity of the phrenic nerve to electrical stimuli in the heart. The cause of the hypersensitivity is not known, but is hypothesized to be due to an electrolyte and acid/base imbalance.[2,3]

The electrolyte and acid/base imbalances develop from the progressive alkalosis and stress encountered by endurance horses. Alkalosis causes a decrease in ionized and total serum calcium. This causes a decreased threshold of the nerve to electrical stimuli, thereby allowing the phrenic nerve to be depolarized by heart impulses. Stress of extensive exercise causes an increase in cortisol levels which further exacerbates the decreased Ca^{++}. Increased cortisol levels also increase urine excretion of K^+, which exacerbates the K^+ lost during sweating. This produces a significant hypokalemia and alkalosis. Hypokalemia has been shown experimentally to cause hyperexcitability in long nerves.[2,3,5]

SDF often occurs following a rest stop because, if the horse is rehydrated without electrolyte intake, the fluid replenishment further dilutes Ca^{++} and K^+ and exacerbates the present alkalosis. Therefore, hypocalcemia, hypokalemia and alkalosis present in endurance horses are likely causes of SDF.[2]

Treatment

SDF usually resolves with rest, but recovery is hastened by rehydrating the horse with a balanced electrolyte solution, either orally or IV. Twenty percent calcium borogluconate can be given IV *slowly* to effect, with careful attention to cardiac monitoring. A standard milk fever preparation with Ca^{++}, Mg^+ and glucose may also be given. Discontinue the IV calcium solution if cardiac irregularities appear. Metabolic alkalosis can be helped by having the horse breath into a paper bag, if tolerated. It should be stressed that endurance horses are alkalotic and not acidotic and therefore *no* sodium bicarbonate should be given to these hores. Horses with SDF should be removed from competition since further exercise may lead to development of exhaustion.[2,3,5,8]

EXHAUSTED HORSE SYNDROME

EHS is a complex metabolic condition which occurs when horses are pushed beyond their physical endurance limits. Three-day event horses usually do not develop the metabolic and acid/base problems to the extent that endurance horses do.[4]

Predisposing Factors

The development of EHS relates to several factors: conditioning, pace of the ride, trail terrain and conditions, temperatures, wind, humidity, and acclimatization of the horse.[4,6] Fluid and electrolyte losses and decreased energy stores also contribute to the condition.[3] Any horse, even in peak condition, in a certain set of circumstances can develop EHS.[4]

Clinical Signs

Each individual horse will show different signs of suffering from EHS. Horses may show a combination of the following symptoms:[2,3,4,6,8,9]

1. General signs: severe dehydration, hyperthermia (may reach 106°F), flaccid anal tone, non-responsive to pinch reflex, myoglobinuria, depression, lethargy, head hung low, expressionless ears, anorexia, no apparent thirst, glazed eyes, anxious expression (especially if experiencing muscle spasms), colic.
2. Cardiovascular signs: tachycardia, tachypnea (shallow), SDF, arrhythmias, moist rales in lungs progressing to pulmonary edema, increased CRT, cyanotic mucous membranes, decreased pulse pressure, jugular pulse.
3. Musculoskeletal signs: fatigue, trembling, muscle spasms, stiffness, muscle pain on palpation possibly progressing to azoturia.
4. GI signs: decreased or absent GI sounds, proneness to colic, usually of a spasmodic type.

All endurance horses will have increased heart rates and respiratory rates with some degree of dehydration during a competition. The respiratory rate may even be greater than the heart rate (known as "inversion") and is seen in humid conditions. This condition is usually transient. The best guide to evaluating impending exhaustion is the recovery from the increased heart and respiratory rates. At veterinary checks during a ride, horses must recover to a heart rate of 70

beats/min and a respiratory rate of 40 breaths/min within 30 minutes in order to continue. Horses not in danger of impending exhaustion will recover to the 70/40 requirement with cooling off, rest, feed, and water.[8]

Pathophysiology

The pathophysiology of the electrolyte and acid/base imbalances is identical to that already discussed under dehydration, only much more extreme (Table 1).[2,3,4,5,6,7]

Alkalosis with decreased serum [Cl^-], [K^+], [Ca^{++}], and [Mg^{++}] affects neuromuscular transmission, and contributes to GI stasis, cardiac arrhythmias, muscle cramps and spasms.

At body temperatures greater than 105.8°F, oxygen utilization exceeds the amount of oxygen supplied by respiration and will lead to tissue hypoxia. Hypoxia will primarily damage the brain, kidneys, and liver.[2]

Treatment

Prompt, intensive treatment is vital to saving the lives of horses with severe exhaustion. The primary concerns are rest, rehydration, and replenishment of electrolytes.

Rapid IV fluids are needed to restore vol-

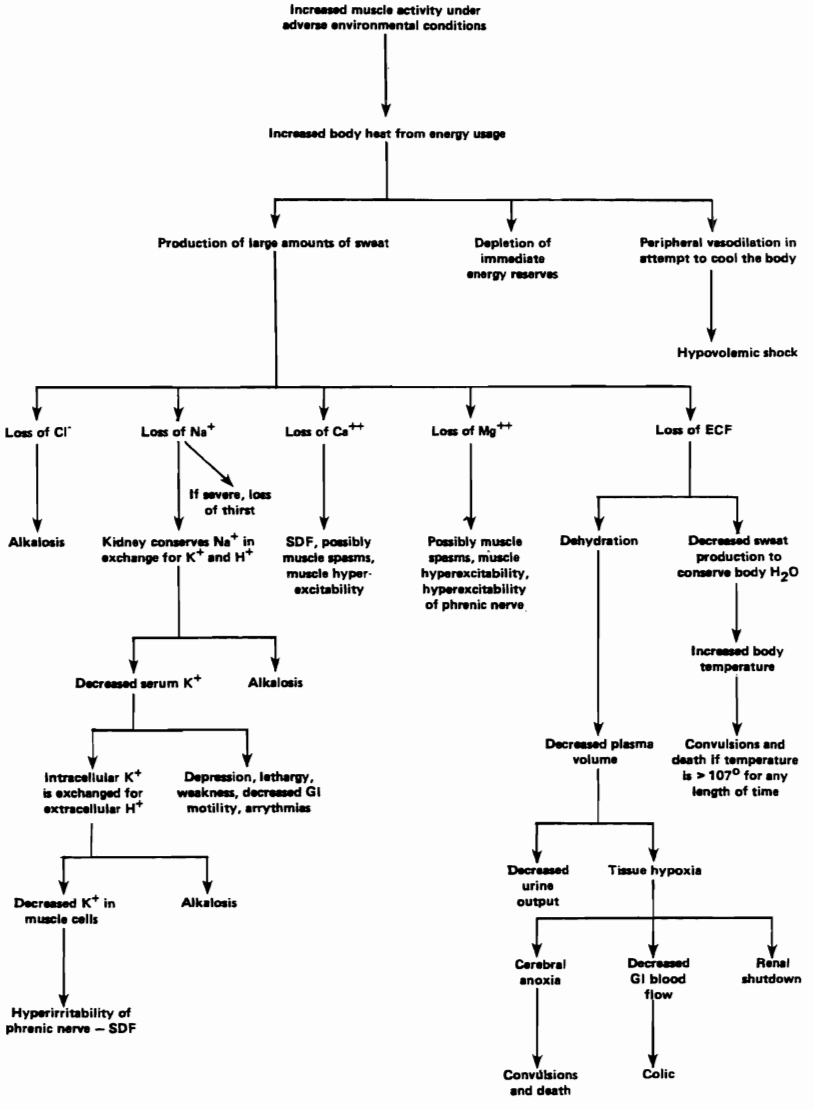

Figure 1. Cascade of events occurring during a stress crisis in endurance horses.

ume deficit, correct electrolyte imbalances, and provide glucose for energy. Since Na^+ is the primary electrolyte of concern, fluids rich in Na^+ should be used. Lactated Ringer's is a good choice. Isotonic saline is not balanced physiologically, but does provide Na^+ and Cl^-. A severely dehydrated horse can have a Na^+ deficit of 4000 mEq. To supply 2000 mEq of Na^+, 15 l of LR or 13 l of isotonic saline are required; therefore, 30 l would be needed. Often horses require 30–40 l of fluids. To supply glucose-dependent organs, 5% glucose may be given at 2 l/hr. Fluids should be administered at rates previously indicated for a severely dehydrated horse.

Oral fluids may be used alone in mild cases, or in addition to IV fluids. Total fluids given by all routes should be monitored. Oral fluids are usually rapidly absorbed and well tolerated. They are contraindicated in collapsed horses or those with severe colic. Approximately 5–10 l can be given at once every 30–60 minutes, as tolerated.[2,3,4,6,8]

To combat hyperthermia, running cool water over areas traversed by the major veins is most effective. Water directed over large muscle groups can delay internal cooling and may cause muscle cramps.[3] Ice water poured over the head and neck is also effective. Cold water enemas will help decrease body temperature and also supply fluids to the horse. Once the temperature is less than 105°F, lowering the temperature by one degree every 30–40 minutes is optimum, since too rapid cooling can cause chilling and lead to shock.[2,3,8] Phenylbutazone at 4 mg/lb will help decrease body temperature and prevent laminitis and DIC.[3] Dipyrone may also be used.[8]

With successful treatment, there should be a decrease in capillary refill time, improved pulse pressure, improved attitude and a return of urine flow.[8] The primary objective of treatment is to restore fluid and electrolyte deficits so the horse can begin eating and drinking to restore the remainder of the deficits.[8]

No sodium bicarbonate should be given to endurance horses because they are already alkalotic. Furosemide (Lasix®) should not be used to restore urine flow until fluid deficits are replaced. Phenothiazine tranquilizers should not be used to treat muscle cramps since there is danger of hypovolemic shock. Muscle cramps are usually due to fluid and electrolyte imbalances and generally respond to fluid thereapy. Azoturia should be considered a possibility.[8] Use of corticosteroids is questionable since cortisol levels are already high from stress.

Recovered horses should have complete rest and should be closely monitored for several weeks for recurrance or the development of laminitis.[3]

EXERTIONAL RHABDOMYOLYSIS

Exertional rhabdomyolysis (ER) is also known as exertional myopathy, azoturia, Monday morning disease, paralytic myoglobinuria, tying-up syndrome, and myositis.[10] ER is a muscle disease which begins with gait alterations progressing to a state in which the horse is too painful to move.[2]

Predisposing factors

Horses most prone to developing ER are those heavily exercised and on high feed rations, obese unconditioned horses that are suddenly put to heavy work, and young horses, especially fillies. Horses with heavy muscling in the loin, croup and thigh, especially Quarterhorses and draft-type horses, are more at risk. Exercise over difficult terrain, such as mud, and under cool, damp conditions also predispose the condition. Thin, nervous horses are also prone to ER. It has also been associated with horses receiving corticosteroid injections.[8,10,11]

Clinical signs

In mild cases of ER, the horse will have some gait abnormalities, muscle stiffness, and reluctance to move. In severe cases, the horse will be in obvious pain. The iliopsoas, quadriceps, gluteals, biceps femoris and triceps muscles are most commonly affected. Horses usually sweat, especially over affected muscles, which are hot, swollen, firm, and painful to palpation. Muscle spasms may be evident. Increased heart rate, respiratory rate, and body temperature are present; myoglobinuria is present in mild to severe cases. The horse's legs appear to be nailed to the ground. If the horse goes down, struggling to rise makes the condition much worse. These severely affected horses may go into shock due to impaired renal function.[2,3,8,10,11,13]

Pathophysiology

The cause of ER is not fully understood. Defects in carbohydrate metabolism and vita-

men E/selenium deficiency are possibilities. The lesion encountered is ischemic necrosis of muscle fibers. The current hypothesis for the development of ER is that with increased muscle activity, blood is shunted to the muscles to supply oxygen and energy. The stimulus for muscle vasodilation may be the release of K^+ from muscle cells. However, with the other metabolic demands causing a hypokalemia, there is a failure for K^+ to be released from muscle cells. Therefore, no vasodilation occurs, resulting in inadequate tissue perfusion for tissue demands. Necrosis of muscle then occurs. Since not all endurance horses get ER, even though all are usually hypokalemic, there must be other factors involved.[2,3,10] Since endurance horses rarely exercise anaerobically, lactic acid accumulation is not thought to be the cause.[2]

With significant muscle damage, myoglobin and muscle enzymes leak into circulation. Myoglobin can pass into glomerular filtrate and causes a distinct coffee-colored urine. Myoglobin causes lower nephron degeneration when it passes through the kidney.[10,13]

Treatment

For mild cases showing only muscle stiffness and no myoglobinuria, the horse should be kept warm and slowly walked. In severe cases the horse should not be moved at all, but should be kept absolutely quiet and blanketed if it is a cool day. It may be necessary to treat the horse out on the trail. It is imperative that the horse should not be moved at all until it is cooled off, rehydrated, and adequately recovered. Intravenous treatment with a balanced IV electrolyte solution is necessary to resolve present dehydration and to flush kidneys of myoglobin. Oral fluids may also be used as discussed in EHS. Pain relief is most important to allow relaxation of muscles and to break the "pain, muscle-spasm, pain" cycle. Non-steroidal anti-inflammatory agents such as phenylbutazone at 4–8 mg/kg are best to use to control pain. If the horse is anxious and in danger of going down, xylazine (Rompun®) 0.5–1 mg/kg may be helpful. Phenothiazine tranquilizers are dangerous to use because of the threat of vasodilation leading to shock.[2,3,8,10,13]

Other treatments have various degrees of success. Twenty percent calcium gluconate, 250–500 cc IV, may be helpful. Antihistamines and IV thiamine have also been used. Prednisolone sodium succinate 1–2 mg/kg IV, or dexamethasone, 0.1–0.2 mg/kg IV, may be helpful in severe cases.[8,13] Vitamin E/selenium is reported to be counter-productive.[2]

Horses which go down and struggle to rise cause much muscle damage. In these cases, a muscle relaxant such as methocarbamol, 15–25 mg/kg slow IV every 6 hours as needed, may help as well as act as a mild tranquilizer.[8]

Feed should be restricted. The horse may be given hay and water *ad lib* until all signs disappear. Rest and restricted feed should be continued for several days after which the horse can be slowly brought onto grain.[8]

Horses which have had ER may recover without any further muscle problems. Others may have muscle fibrosis and loss of muscle mass leading to gait abnormalities and disfigurement. Young horses are predisposed to future attacks, but this usually decreases with age.[8,10]

CONCLUSION

Providing care for endurance athletes poses a unique challenge for the veterinarian. Diagnosis and treatment of an acutely ill animal under varying circumstances are the true tests of a veterinarian's ability to apply principles of equine medicine. But the real reward in working with these horses is to see firsthand the athletic endurance, stamina and "heart" of the equine.

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