Diagnostic Approach To Equine Muscle Disorders

Stephanie Valberg, DVM, PhD, professor of large animal medicine and director of the University of Minnesota's Equine Center, began the in-depth seminar on muscle disorders by discussing diagnosis of muscle disorders, beginning with a careful physical exam and concise history of the clinical problem. She said in most cases, a muscle disorder arises from one of the following situations: muscle strain, exertional rhabdomyolysis (tying-up), weakness and exercise intolerance, abnormal muscle contraction and nerve conduction, or muscle atrophy. Identifying the exact nature of a horse's muscle problem is dependent on characterizing the clinical signs to narrow down the problem.

Careful inspection of a horse's muscle mass and symmetry and hands-on palpation educates the examiner about the horse's muscle tone and comfort level. The examiner also evaluates the horse in motion and conducts a full lameness exam. A serum chemistry panel is helpful for biochemical analysis of muscle enzymes that provide telling information about muscle health and the duration of muscle injury.

There are some enzymes that can yield information about muscle, although not all of these enzymes are found exclusively in muscle. Serum creatine kinase (CK) is released within just a few hours of muscle damage, peaking within six hours following injury. Elevations in CK are usually consistent with training, transport, or taxing endurance or eventing competition. Higher levels from these causes generally are within an expected range and should return to normal quickly, whereas a horse experiencing rhabdomyolysis will have extreme increases in CK.

Serum aspartate transaminase (AST) is an enzyme that rises more slowly after muscle insult, peaking in 12-24 hours. Its clearance rate to return to baseline might take as long as two to three weeks after a bout of rhabdomyolysis. Serial chemistries (several taken in a row) can provide a veterinarian with knowledge about healing response.

Lactate dehydrogenase (LDH), an enzyme important to energy function in cells, is useful when evaluated in conjunction with CK.

Myoglobin (the oxygen-transporting pigment of muscle) increase in plasma or serum can alert the veterinarian to acute muscle damage even before it is visible in the urine. Urinalysis for myoglobin and fractional excretion of electrolytes (a technique used to measure electrolytes or other components of urine in comparison, or fraction, with serum values of those same components) are invaluable tools to ascertain muscle damage and electrolyte abnormalities leading up to rhabdomyolysis. Exercise testing at a trot for 15 minutes, coupled with serial muscle enzyme (CK) evaluation, help quantify the degree of exertional rhabdomyolysis in chronic cases with mild signs.

Valberg discussed using thermography to detect heat from inflammation, reminding veterinarians to compare both sides of the horse. Nuclear scintigraphy is another useful tool to zero in on a complaint of...
subtle poor performance possibly related to rhabdomyolysis. Release of large amounts of calcium from damaged muscle seems to be an attractant for the radioactive material injected into the horse for the nuclear scan. Therefore, areas of damage are visible when using this imaging modality.

Diagnostic ultrasound is another useful tool to look for traumatic fiber disruption and/or scar tissue. Valberg stressed that artifact (something artificial or a distortion that does not represent normal anatomy or pathology) is easily confused with reality in these scans, so it is important that a horse stands squarely with his full weight equally dispersed among his limbs, and that the veterinarian compares the same muscles on the left and right sides.

Another diagnostic technique uses electromyography (a test that measures electrical signals in the muscles) when there are signs of muscle atrophy, tremors, or dimpling. This evaluates conduction within the electrical system of muscle and nerve units. Abnormalities are seen on electromyography with hyperkalemic periodic paralysis (HYPP, a genetic disease of Quarter Horses and derived breeds, characterized by sporadic episodes of muscle tremors and stiffness, along with elevated serum potassium levels), myotonia (increased muscular irritability and contraction with decreased power of relaxation), or atrophy created by nerve injury.

One extremely helpful diagnostic tool is a muscle biopsy, particularly in the diagnosis of polysaccharide storage myopathy (PSSM) or immune-mediated myopathies.

Finally, Valberg reported on the value of genetic testing using DNA markers in hair root samples to identify genetic mutations that are characteristic of muscle diseases. Genetic tests can be used to detect HYPP, glycogen-branching enzyme deficiency (GBED), and other muscle diseases.

**Equine Polysaccharide Storage Myopathy**

Valberg also discussed EPSM. The disorder causes muscle pain, stiffness, and cramping, and some horses demonstrate exercise intolerance, poor performance, muscle weakness, atrophy, or related behavioral changes. This syndrome is caused by an inherited enzyme defect that allows accumulations of glycogen in the muscle.

Gene expression is markedly influenced by the horse's environment, particularly as related to diet and exercise. The most prevalent breeds affected include Quarter Horse-related breeds, drafts, and Warmbloods. Individuals within other breeds have been recognized with this syndrome.

The typical horse with EPSM is calm--even sedate--and well-muscled, but not very fit. Signs usually show up at the beginning of a training session. An affected horse is not necessarily being fed a high-grain diet, although high-carbohydrate and high-starch diets amplify signs in many instances. Many affected horses stop to posture as if to urinate; it is thought that this relieves cramping in the abdominal muscles. In draft breeds, it is typical to see muscle atrophy, abnormal gait, and high-stepping action in the rear limbs. Eventually there is progressive weakness and muscle atrophy. Warmblood horses often demonstrate signs of a sore back, sore muscles, reluctance to collect, and overall poor performance issues.
Although there is a genetic marker test potentially available in the near future, the most definitive way to diagnose this problem is with a muscle biopsy, which is sent to an appropriate lab where it is frozen for analysis. The results are graded as mild, moderate, or severe.

Not all horses with PSSM demonstrate clinical signs, nor do they all have elevations in muscle enzymes. It tends to be first recognized in Quarter Horses at four to five years of age and Warmbloods and draft horses aged eight to 11 years. After exercise, a normal horse will slowly absorb glucose from the bowel and slowly replenish metabolized glycogen to normal resting levels. In a PSSM horse following exercise, there is overcompensation, so glycogen is packed into the muscles, reaching twice-normal levels.

A PSSM horse that eats grain seems to produce less of an insulin response compared to a normal horse, yet that insulin has a greater impact to transport more glucose into the cells as free, non-phosphorylated (not yet activated into a useful high-energy compound) glucose. If the glucose is not immediately used, it is synthesized and stored as excess glycogen. These glycogen packets are visible under the microscope through staining of the biopsy sample. There seems to be some limitation on how well PSSM horses can use this excess glycogen for energy generation.

Once a diagnosis is obtained, treatment relies on management with strict diet and exercise protocols. These horses will always be at risk of some underlying muscle soreness, but at least 80% can improve with diet and exercise.

It is best to turn a PSSM horse out on pasture and resist the urge to confine the horse to a stall or paddock. Those PSSM horses confined to stalls or paddocks will experience CK elevations, particularly within the first five minutes of exercise. Those horses turned out to exercise at will and can walk around to forage do not experience CK elevations. Free exercise is key to keeping a PSSM horse comfortable over the long term. Exercise should be applied through careful conditioning programs that steadily increase demand and allow the horse to adapt to the demand.

Dietary modifications essential to managing PSSM include feeding a low-sugar, low-starch diet with fat to serve as an alternative energy source. This helps the muscle cells take up less glucose while providing free fatty acids for muscle use during aerobic exercise. Figure out how much energy each individual horse really needs. Small, frequent meals work best to eliminate generation of high blood glucose surges from a meal. Caution should be taken in feeding a high-fat diet to an already fat horse. These horses might need hay restricted to no more than 1% of body weight and also to be fed low-starch, fat-supplemented feed that is high in fiber, combined with daily exercise to get weight under control.

The worse the clinical signs experienced by the horse, the lower in starch (non-structural carbohydrates or NSC) the diet should be. Less than 10% of a horse's daily energy should be fed as sugar or starch, and fat should be added at 10-15%. Commercial feeds are available to provide low starch and high fat. The horse's diet might need to be tweaked to find one that is most palatable and low enough in starch, yet one that provides adequate fat.

One cannot solely change the diet to achieve results. Increased daily exercise is key, especially in Quarter Horses that have low oxidative enzymes, so they must exercise and train to efficiently burn fat. Regardless of breed, a horse must be in regular exercise--working gradually up to 30 minutes daily for five days a week. To achieve the best results possible, it is essential to follow dietary recommendations along with
providing routine exercise and regular turnout. Results from diet and exercise modifications should be evident in as few as three weeks, or it might take as long as four months.

**Hyperkalemic Periodic Paralysis: 14 Years Later**

Sharon Spier, DVM, Dipl. ACVIM, PhD, associate professor at the University of California, Davis, has pioneered much of the research on HYPP. She presented updated information gleaned over the past 14 years on this disease.

The syndrome was identified in the 1980s in descendants of the Quarter Horse stallion Impressive. It was noted that HYPP particularly seemed to affect young, well-muscled Quarter Horses, Appaloosas, Paint horses, or crosses with this lineage, leading researchers to look for a genetic link. They found a genetic defect that affects conductivity of muscle membranes, resulting in persistent muscle contraction.

Diagnostic testing for HYPP (on tail and mane hairs) has been available since 1992, and it can distinguish among homozygous-affected (H/H), heterozygous-affected (H/N), and normal (N/N) horses. (Homozygous horses, which have a double copy of the defective gene, are far more severely affected than heterozygous ones.)

In 1996 the American Quarter Horse Association (AQHA) officially declared HYPP as an undesirable genetic defect, and it established mandatory testing for all foals born after Jan. 1, 1988, descending from Impressive. Just a couple of years ago, it was mandated that any foal born in or after 2007 that tests H/H cannot be registered.

The clinical signs and onset are variable; most affected horses do have clinical signs, while a few horses with the mutation will appear normal. An affected horse has intermittent attacks of weakness, muscle tremors, and/or paralysis and recumbency that can last from minutes to hours. In some cases a paralytic episode might persist longer than 24 hours and the horse might require slinging with a large animal lift. Many horses remain standing during an episode; some might sway or stagger, while others dog sit due to hind limb weakness.

Other signs include elevated heart rate, prolapse of the third eyelid, abnormal respiratory noises, and sweating; yet the horse remains bright and alert, albeit anxious. A horse can die from respiratory or cardiac paralysis.

An attack is not necessarily precipitated by anything definable such as exercise, although high-potassium feeds and/or stress situations readily precipitate an episode. A foal might demonstrate clinical signs early on, including an inability to nurse or swallow due to partial or complete laryngeal paralysis. Such a foal will make a high-pitched wheezing noise that accompanies respiratory distress.

An HYPP-affected horse appears normal between episodes, with normal laboratory findings. Veterinarians have found if affected adult horses are managed carefully through diet and exercise, many can live relatively normal lives.

During an HYPP episode, laboratory tests show blood concentration and high circulating serum levels of potassium, with muscle enzymes ranging from normal to slightly increased. Muscles remain abnormal
even between bouts of symptoms, maintaining some degree of excessive tone. Electromyography demonstrates that all muscles are affected.

The actual genetic mutation that causes HYPP involves a defect affecting a protein that is called the voltage-gated sodium channel. This channel is a tiny gateway in the membrane of a muscle cell that controls the movement of sodium particles in and out of the cell. These particles have a charge that changes the voltage current of the muscle cell. The current allows the cell to contract or relax.

In HYPP horses, the channel's regulation of the particles sometimes fails, and this disrupts the normal flow of sodium in and out of the cell. During episodes, uncontrollable twitching or complete muscle failure can result, as can potassium leakage into the bloodstream.

At UC Davis Spier tested samples submitted by Quarter Horse owners for HYPP testing in 1992-1996 and found that 1.1% had the H/H homozygous genotype, indicative of disease, while 35% were heterozygous affected (N/H). Results of testing in 2005 indicated that 2.2% of horses tested had the H/H genotype, and 37% were N/H. (N/H horses are affected with the disease and have a 50% chance of producing a foal that is HYPP positive.)

What is particularly striking is that in the past 14 years of genetic testing, not only has the HYPP gene frequency not decreased, but there has been a two-fold increase in the number of horses with this homozygous gene frequency.

Breeders continue to be "rewarded" for this defect since HYPP horses of Impressive descent tend to be heavily muscled, a trait that is considered desirable in the halter horse show ring. It is possible that constant muscle stimulation from the disease itself leads to muscle definition and tone that is visually favored. Thus far, 4% of Quarter Horses are affected; meaning that approximately 160,000 individuals have this disease.

HYPP can be controlled and managed to some degree with diet by eliminating or limiting high-potassium feeds or supplements containing alfalfa hay, soybean products, molasses, electrolytes, or kelp. Dietary potassium should be kept below 1½% of the horse's daily nutritional intake. Fasting periods should be minimized, as should stressful events such as trailering.

Anytime a horse will be sedated or undergo general anesthesia, the owner must warn the veterinarian of a horse's HYPP condition. An owner might avoid an acute, mild attack by administering corn syrup or grain to provide sugar that drives potassium back into the muscle cells, and by lightly exercising the horse. A veterinarian might also implement intravenous medical treatment with calcium and dextrose to elevate calcium levels and decrease membrane hyperexcitability. He or she also might give other medications to increase potassium excretion in urine and stimulate insulin secretion to drive potassium into the cells.

**Glycogen-Branching Enzyme Deficiency**

Valberg presented information on GBED. The disorder involves glycogen storage in Quarter Horse or Paint horse foals, and it is associated with abortion or high neonatal mortality.
Glycogen-branching enzyme deficiency, a genetic mutation affecting a particular glycogen-storage enzyme, is traced back to Quarter Horse sire King or his sire, Zantanor. Up to 8% of Quarter Horses and Paint horses carry the GBED defect. Pedigree analysis is not particularly helpful in identifying if a horse has this gene because both of these horses are foundation stallions for the breed.

Researchers believe GBED has caused significant fetal and neonatal mortality in Quarter Horses and Paints for decades, although sophisticated histopathology of muscle tissue biopsy is required to identify it. Currently, the recommendation for diagnosis is genetic testing through the Veterinary Genetics Lab at UC Davis (www.vgl.ucdavis.edu) using mane or tail hairs with intact roots or samples of fetal liver tissue.

Many affected foals are aborted in late term or are stillborn. If the mare delivers a live GBED-affected neonatal foal, it is born weak with a low body temperature, is slow to stand to nurse, and might have slightly contracted tendons. With good clinical care, a GBED foal might respond for a short while and seem to improve, then deteriorate. Others might initially experience respiratory distress, collapse from hypoglycemia, seizures, then sudden death. GBED-affected foals have poor survivability, with most succumbing by eight weeks of age. One foal survived until 18 weeks old with aggressive nursing care.

There is no treatment for GBED foals. The best prevention is through early recognition that aims to limit an owner's heartache and financial investment in attempts to save a dying foal. It is important to know that rebreeding a mare to the same stallion will give an owner a 25% chance of repeating the same fatal outcome.

Four percent of Quarter Horses have GBED and do not survive, and 8% are carriers. In Paint horses, 7% are carriers. No GBED defect has been found in Thoroughbreds.

**Immune-Mediated Myopathies**

Valberg discussed muscle disease created by immune-mediated situations, describing three possible different manifestations. One type of muscle damage develops subsequent to an outbreak of *Streptococcus equi* (strangles).

If a horse has strangles, then about three to four weeks later the horse might experience a severe immune-mediated case of muscle damage, also known as *S. equi* rhabdomyolysis (a form of tying-up). Such a horse might seem fine, then suddenly he develops severe muscle damage and his urine might be a dark coffee color. Most reported cases of this have been in Quarter Horses less than seven years old.

An affected horse has typical signs of a strangles infection: swollen submandibular lymph nodes and/or pus in the guttural pouch caused by *S. equi*. Then, suddenly the horse demonstrates a stiff gait, firmness of the muscles with severe muscle pain, and eventually he can become recumbent in spite of anti-inflammatory medication and antibiotics. Imminent death in cases of *Strep* myositis is caused by severe muscle necrosis, subsequent kidney damage, and multiple areas of venous thrombosis (blood clotting in the veins). This is not a result of any specific strain of *S. equi*, but the inflammatory cascade can produce a toxic shock-like syndrome.
A second immune-mediated condition is severe muscle swelling from infarction (localized tissue death resulting from obstruction of the blood supply to the affected site), called infarctive purpura hemorrhagica (PH), that also develops two to four weeks following a respiratory infection associated with *S. equi* spp.

Deposits of immune complexes of antigen and antibody elicit inflammation in blood vessels (vasculitis) and focal hemorrhages in the muscles. An affected horse becomes stiff and lame, depressed, and he might appear colicky. Other signs of classic PH also appear, such as depression, blood spots on mucous membranes (petechia, or pinpoint purplish red spots, and ecchymoses, which are similar, but larger, spots), and pitting edema of the lower legs, belly, chest, and head.

With the infarctive form of PH, the pectoral, abdominal, and/or hind limb adductor muscles become hard and painful. Infarctions in the bowel will elicit colic and death. In most cases, infarctive PH is rapidly progressive with a high mortality rate; early recognition and aggressive treatment with high doses of corticosteroids and antibiotics might save a horse's life.

During her presentation, Valberg also discussed a third immune-mediated muscle disease--immune-mediated polymyositis--is characterized by sudden muscle atrophy. In about 40% of these cases, exposure to some respiratory disease or *S. equi* has been a trigger. This syndrome, so far identified predominantly in Quarter Horses, some Paint horses, an Icelandic horse, a Thoroughbred, and a couple of ponies, is more common than the two previously discussed, and it is very dramatic.

Within three to four days, an affected horse loses all muscle mass, starting on the topline and haunches. Usually the atrophy is symmetrical, but not always. Within a week, more than half of a horse's muscle mass might waste away, causing dramatic weakness, stiffness, and general malaise. Muscle biopsy of the back or gluteal muscles is important to establish a definitive diagnosis and to rule out PSSM.

Antibiotic treatment is recommended to resolve bacterial respiratory disease, and tapering doses of corticosteroids can limit progression of muscle atrophy. Clinical signs can recur in 40% of cases and might require corticosteroid treatment (although not intramuscularly, since muscle damage has already occurred).

Once the immune-mediated syndrome is interrupted, the muscles should respond and the horse can recover, although in some cases, the full extent of muscle mass might not entirely return to normal.

**Shivers in the Horse: A Review**

John Baird, BVSc, PhD, of the University of Guelph's Ontario Veterinary College, presented information on "shivers," which has been recognized by horse owners for more than a century. Shivers refers to a chronic nervous or neuromuscular condition that in a 1962 text was stated to be "as common as dirt." This was referring to the period when draft horse populations were much larger than today.

Besides drafts and draft crosses, shivers is recognized also in Warmbloods and Warmblood crosses, as well as occasional light horse breeds such as Quarter Horses, Standardbreds, and Thoroughbreds. It is rare to find it in pony breeds.
Typically, shivers starts out as difficult to detect, and it progressively worsens with age. The horse experiences involuntary muscle spasms or jerky movements in the pelvis, one or both hind limbs, and tail. Initially, it seems a horse is snatching its leg away when asked to pick up a foot for cleaning.

Signs are most evident when an affected horse is asked to back up, move quickly, or when lifting a hind leg for hoof cleaning or while being shod. The horse abruptly raises the hind leg in a partially flexed position held slightly out from the body, and he is unable to put it down. This posture lasts from seconds to minutes. As the limb is slowly replaced to the ground, the horse might experience spasms again, especially if asked to move backward. The degree of associated tail elevation is variable. Usually, signs are not elicited by asking a shivers horse to move forward.

The problem seems worse on slippery surfaces; stress or excitement can exacerbate clinical signs. This condition might rarely affect the forelimbs, neck, or trunk. Occasionally, the muscles of the ears, eyelids, lips, and cheeks can be affected with spasmodic contractions, rapid blinking of the eyelids, ear quivering, and twitching of the lips.

As the disease advances, initially there is noticeable atrophy of the thigh, and this might progress to generalized muscle atrophy with hind limb muscle weakness in 58% of horses with shivers. The limbs become stiff or rigid, and the horse prefers to stand with his hocks wider apart than normal. Because the horse is reluctant to lie down, there might be bumps and bruises from partial falls due to fatigue, and sometimes the horse will make crouching movements. Some owners have noted a disproportionate amount of sweating in shivers horses.

Stringhalt is often confused with shivers, and although stringhalt is a jerky, rapid flexion of a hind limb, with stringhalt, the limb is violently thrust back to the ground after flexing hard into the abdomen. Stringhalt can occur in all breeds; the abnormal gait typically is elicited when a horse is asked to move, turn, or back up. In contrast, a horse with shivers holds the limb up and away from the body for a period of time.

Another differential diagnosis in a possible shivers case would be upward fixation of the patella that is caused by a transient sticking of the medial patellar ligament above the medial trochlear ridge of the femur. This holds the limb in a hyper-extended position that can release suddenly, making it look like stringhalt.

Fibrotic myopathy is caused by scar tissue restriction along the thigh muscles subsequent to an injury. A horse with this problem slaps the hind limb down abruptly, and he doesn't raise it very high to the abdomen due to mechanical restriction by the scar tissue.

"Stiff-horse syndrome" has been reported in Belgian horses; these horses show intermittent stiffness and spasms in the muscles of the lower back and hind limbs, with contractions triggered by voluntary movement, fright, or noise. These horses experience muscle hypertrophy (enlargement) rather than atrophy.

Another differential diagnosis is equine motor neuron disease (EMND) caused by vitamin E deficiency. It is characterized by progressive weight loss, symmetrical muscle wasting, muscle tremors, excessive sweating, a tucked-up abdomen, abnormal gait, excessive recumbency, a low head carriage, and abnormal
tail elevation. On occasion, an EMND horse or a horse with equine protozoal myeloencephalitis (EPM) will demonstrate stringhalt-like movements of front or rear legs if certain areas of the spinal cord are affected.

No specific lesions have been documented in horses with shivers in any portion of the central nervous system. While no one knows what causes shivers, it possibly has a neurologic pathology. Another suggestion has been some depletion of glycogen leading to muscle cramping, but there has been little correlation between the severity of clinical signs and findings on histopathology to support this view.

There is also a question whether shivers is genetic or is genetically predisposed. Although not proven, it is suspected that there is some hereditary component.

There is no effective treatment for shivers. A horse might improve with rest, but shivering will return when exercise resumes. Some owners feed a high-fat, low-carbohydrate diet similar to that used to manage PSSM, and this might help early in the disease. In another study, clinical signs did not resolve when fed a high-fat diet.

Some days are better than others for some shivers-affected horses. In general, this is considered a progressive, debilitating disease with a poor prognosis. Clinical signs eventually increase in frequency and severity, and muscle wasting and weakness worsen. The course of the disease can progress rapidly or take as long as 24 years to render a horse incapacitated.

**Exertional Rhabdomyolysis**

Valberg then tackled the broad subject of skeletal muscle disease related to exercise. This has been a problem recognized for centuries with names such as azoturia, Monday morning disease, tying-up syndrome, myositis, and set fast, all of which are accompanied by muscle pain. But each syndrome is a little different. These are more descriptively related under the term exertional rhabdomyolysis or ER.

Valberg noted that as many as 3% of horses in exercise might have experienced an episode of ER within the past year. Certain exercise, such as polo and racing, might elicit a greater incidence. Typical clinical signs include muscle stiffness, muscle cramping, acute onset of hind limb lameness, refusal to move, and other signs of distress, such as increased heart and/or respiratory rates, sweating, and colic.

Diagnosis is made based on clinical signs and muscle enzyme levels. Creatine kinase values greater than 300,000 u/L might be associated with muscle atrophy following several days of muscle edema and swelling. Usually muscle tissue will regenerate in about three months, and prognosis is good for performance if CK never exceeds 250,000 u/L. For horses with CK greater than 500,000 u/L, there is a guarded prognosis for healing or future athletics.

For a few days following an episode of ER, horses should be confined with minimal hand-walking. When stiffness dissipates, turnout in a small paddock is advised and muscle enzyme values should be checked continually until they return to normal.
Sporadic cases of ER develop related to overexertion in a horse not fit for the task, or from heat exhaustion with dehydration and electrolyte imbalances. Vitamin E deficiency is also becoming more common as horses get less access to pasture.

One question often asked is if this syndrome is heritable. There is a form of recurrent exertional rhabdomyolysis (RER) in Thoroughbreds, Standardbreds, and Arabian horses, and PSSM is found in Quarter Horses and draft breeds. Both are associated with genetic susceptibility.

Recurrent exertional rhabdomyolysis is thought to be caused by an abnormality in regulation of calcium within muscle cells. It tends to be more prevalent in mares, with as many as 80% of 2-year-old Thoroughbred fillies affected. Nervous temperament increases prevalence by fivefold. The presence of concurrent lameness increases likelihood of developing RER by fourfold.

Recurrent exertional rhabdomyolysis seems to be a stress-related disorder with intermittent clinical signs, and it is especially prevalent in fit horses. Everything proceeds along okay for the first 15-30 minutes of exercise, then something triggers the muscles to expel excess calcium, leading to sustained muscle contractions and spasms. During this time, CK enzyme levels increase dramatically.

Muscle lactate concentrations in RER horses are 5-10 times lower than in healthy horses after racing, so there seems to be no relationship between an episode of RER and lactic acidosis (accumulation of lactic acid more rapidly than it can be metabolized), an important point when devising management strategies.

Treatment recommendations for RER include minimizing stress and continuing with light training, as rest makes this syndrome worse.

A horse should be warmed up well before being asked for more exertion. Any lameness condition should be accurately diagnosed and treated. In some cases, administration of low-dose (1-2 mL) acepromazine given 30 minutes prior to exercise is helpful. In Standardbreds, interval training using sprinting episodes seems to help diminish occurrence, and trot time should be limited to 15-20 minutes.

In some cases, treatment with daily progesterone might improve a nervous mare by controlling her heat cycles. There are reports that dantrolene (trade name Dantrium, a direct-acting skeletal muscle relaxant) given 90 minutes before exercise (and on a fasted stomach) has been shown to deter episodes.

In all cases, dietary management is important. Limiting starch not only modifies fuel energy use in the muscle, but it also decreases excitability and nervousness. Because it's difficult to keep weight on a racehorse or a horse engaged in rigorous training and athletics, a commercially prepared high-fiber, high-fat, low-starch diet should be offered, as these diets are palatable, nutritionally balanced, and designed to maintain a horse's weight. There are presently some commercially available feeds that specifically address ER horses.