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EQUINE ROUNDS

Student Chapter ● American Association of Equine Practitioners
New York State College of Veterinary Medicine ● Cornell University

A NEW AND IMPROVED HAY

by Andrew Pepper, '87
with special thanks to Dr. Hintz and Dr. Lowe

Good quality hay is an important component of any horse's diet, but it is often expensive or unavailable. As a result, poorer quality hay which is moldy or dusty, but cheaper, may often be used. Poor quality hay can result from baling when the hay is too wet. Poor quality hay is often wasted feed, and can also aggravate respiratory problems such as heaves. Better methods of curing hay could provide less expensive hay and improve respiratory conditions.

A feeding study with acid treated hay was conducted recently at Cornell's Equine Research Park by H.F. Hintz, J.E. Lowe, and W.F. Miller. This study investigated a method of hay treatment which has been used for cattle, but has not been studied in horses. The hay was treated with a combination of 80% propionic acid and 20% acetic acid at a rate of 15 pounds of acid for each ton of hay. These acids are normal products of digestion in the horse. The acid was added to the hay at baling time from equipment mounted on the baler. Acid treatment allowed the hay to be baled at a moisture content of 30%. (Hay is usually baled at 15-20% moisture.)

The study looked at palatability and acceptance, composition, and general condition after feeding for 2 1/2 to 4 months. Although the acid treated hay had a higher moisture content, the nutritional content, on a dry matter basis, was not altered. The treated hay (second cutting alfalfa) appeared much less dusty than the untreated hay, had a slightly different odor, and felt slightly more "slippery".

Two horses with heaves were fed treated hay for 3 or 4 months. At the end of this time both horses showed definite clinical improvement with ease of breathing and decreased coughing. A mare and her 10 week old foal were fed the treated hay for 2 1/2 months. The foal grew well, and both had ample body fat and sleek coats.

In a study with 8 horses given a choice of untreated or treated hay, the horses preferred the untreated hay. When, however, the horses were offered treated hay alone they ate normal amounts.

Acid treated hay seems to be a very promising solution for horseowners who have trouble finding good quality hay or who have horses with respiratory problems. The positive side of the study is that hay can be baled at a higher moisture content while less hay is lost due to mold and poor curing. Less dusty hay could improve the health of some horses with respiratory problems such as heaves. Possible disadvantages are mainly economic. Acid to treat the hay and the equipment to apply it would increase costs. Also the bales would be wetter, and therefore, heavier and harder to handle. More studies are needed but the present results indicate acid treated hay has great potential as a feed for horses.

IVERMECTIN

By Carolyn Prouty, '87
with thanks to Dr. William Rebuhn and Dr. Edward Boraski

The discovery of a new drug often creates great excitement and controversy; such is the case with the new antiparasitic drug, ivermectin. Ivermectin, one of a family of pharmaceuticals called "avermectins", is found to be effective against a tremendously broad spectrum of arthropod and nematode parasites that are seen in cattle, sheep, swine, dogs and horses. The commercial product, Eqvalan , was first marketed in April, 1983, and quickly gained widespread use in equine veterinary practice both because of its efficacy against a wide variety of parasites, and because of its potency at relatively low doses. Eqvalan was first distributed in an injectable form, and later as an oral paste. Recently, however, Merck & Co. withdrew the injectable form from further sale due to complaints associated with its administration. There are still questions as to why the injectable form is associated with adverse reactions in some horses, and the situation was discussed with clinicians at the Large Animal Clinic of the New York State College of Veterinary Medicine, and with a representative from Merck & Company, the manufacturer.

The avermectins were discovered in Japan as the natural products of a new species of bacteria, Streptomyces avermitilis. They derive their name from their efficacy against two major groups of parasites: internal roundworms, or nematodes; and ectoparasites, or arthropods. That is, they are anti-vermes and anti-ectoparasites, thus avermectins.

Ivermectin is effective against nearly all genera of equine nematodes, and most importantly, is highly active against both mature and immature worms. This includes the migrating larvae of Strongylus vulgaris (strongyles), which may cause vascular damage and thromboembolic colic. Ivermectin is also

Bot fly larva (Gasterophilus) in horse's stomach.

effective against the Gasterophilus species (bot flies) and the flies implicated in summer sores, Habronema species and Draschia megastoma. It has no effect, however, on tapeworms.

Extension of the use of ivermectin to treatment of human parasites is being considered because of its success in the horse against the microfilaria Onchocerca cervicalis, O. volvulus infections, including river blindness, are a significant problem in some African nations, and preliminary tests are promising.

The effectiveness of ivermectin is enhanced by its potency: microgram quantities provide adequate levels for anthelminthic activity where most conventional drugs require milligrams. Toxicity of the drug is very low in part because it does not readily cross the blood-brain barrier.

Ivermectin works in the parasite at the junction between neurons and muscle cells or muscle neurons, where it enhances inhibition of muscle movement. This paralyzes the parasite and eventually leads to its death.

Two different clinical problems are associated with the use of ivermectin in horses. The first is a minor problem of ventral edema which may develop in animals that have a heavy

infestation of Onchocerca cervicalis. The reaction is an immune response to the killed microfilaria in the skin, and usually resolves without treatment in two to three days.

A more serious problem is the incidence of clostridial infections following intramuscular administration of the drug. The infections that result produce a local clostridial myositis and in some cases progress to a systemic toxemia and a condition called malignant edema which can cause death. This range in the degree of disease is typical of clostridial infections of equine muscle and will vary depending on local tissue factors, the virulence, toxin-producing capability, and potency of the toxins produced. Clostridial infections are typically seen after abrasions and lacerations of the skin, and after intramuscular injections, when spores are introduced deep into the muscle. Six deaths of horses treated with ivermectin have been brought to the attention of clinicians at the Large Animal Clinic of the New York State College of Veterinary Medicine since the injectable form became available in 1983.

The most serious manifestation of clostridial infection, malignant edema, is an acute disease with muscular edema and hemorrhage, bacterial invasion of cells, and production of enzymes and toxins which may result in anemia, leukopenia, massive muscle necrosis and death. Eight cases of malignant edema have been seen in the NYSCVM Large Animal Clinic in the past five years. One of these was associated with an intramuscular injection of ivermectin.

Merck & Co. report that there have been approximately 1.5 deaths per 100,000 horses treated with injectable Eqvalan. Dr. Ed Boraski, Director of Technical Services for MSD/AGVET, U.S. Operations, a division of Merck & Co., said in a phone interview that the company's information and feedback came primarily from veterinarians. All reports were subsequently forwarded to the Food and Drug Administration, as is standard procedure for new drugs on the market.

In the face of these adverse reactions, Merck & Co. withdrew the injectable Eqvalan from the market. Dr. Boraski explained that they will not be recalling the doses which have already been sold because there is no specific problem with the drug itself. While Dr. Boranski said that Merck & Co. could not recommend or condemn it, a practice of injecting 2 cc of penicillin along with the ivermectin to prevent clostridial infections has been used with anecdotal success.

There have been no problems reported with the use of the oral paste form of ivermectin in horses, and it appears that this will be the method in the future by which horse owners may benefit from this new antiparasitic drug.

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A Newsletter for Horsepeople

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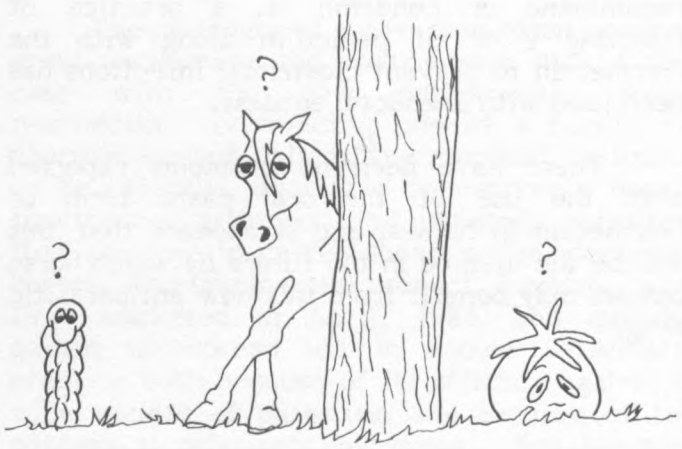
TOMATOES, EARTHWORMS, AND HORSES

by Elizabeth Uhl, '88
with special thanks to John E. Lowe, DVM



What do tomatoes, earthworms and horses have in common? They are all harmed by contact with black walnut trees (Juglans nigra). Tomato plants and earthworms die if exposed to a toxic substance called juglone, which is produced by the black walnut tree. Horses can suffer acute laminitis (founder) from contact with black walnut shavings in their bedding. The agent which causes laminitis has not been identified but may be related to juglone.

Winter is approaching, and many horses will be brought inside until spring. One of the most commonly used bedding materials is soft wood shavings. Unfortunately (usually around mid-winter), the supply of soft wood shavings can run short, and dealers may substitute hard wood shavings. According to Dr. John Lowe, Director of the Equine Research Park, if these hard wood shavings contain any black walnut (even as little as 20%), it can be disastrous to horses.

The following examples were taken from actual cases compiled by Dr. Lowe:

Stable #1 - Black walnut shavings were used to bed down ten horses. Within 24 hours all ten required treatment for acute laminitis.

Stable #2 - A load of wood shavings containing 20% black walnut by weight was delivered and used as bedding. All seven horses exposed to the shavings showed signs of toxicity (including two horses that had been exposed to only 10 kg of the shavings).

Stable #3 - Twenty-eight of thirty-two horses were treated for acute laminitis within 24 hours of their initial exposure to black walnut shavings.

Stable #4 - Thirty-five horses were bedded with black walnut shavings. Within 24 hours thirty had acute laminitis.

Signs of toxicity usually occur within 12-24 hours of contact with the black walnut shavings. Affected horses show an increase in temperature, pulse and respiratory rate that corresponds to the severity of the laminitis. The legs are often edematous (swollen) from the knee or hock down. The severity of the signs ranges from slightly swollen legs to animals that refuse to move. In some horses the hind legs are more affected than the front, giving these horses a gait that resembles myositis or "tying up". Foals and yearlings are often unaffected or recover quickly. If horses are treated for laminitis as soon as the first signs are observed, most will recover without any lasting effects.

Outbreaks of black walnut poisoning have affected 37-100% of the horses exposed. This variation is probably due to many factors, including: length of exposure, amount and toxicity of the compound present, percentage of black walnut in the shavings and type of storage (exposure of the cut shavings to air seems to decrease toxicity).

Horses exposed to living black walnut have also been adversely affected. Two farms in the midwest had to take the drastic action of cutting down all the walnut trees, removing the stumps and then replacing the soil in paddocks and stables, after the development of acute laminitis and respiratory disorders in their animals. The owners and the loggers who cut down the trees also suffered respiratory disorders. The reactions to the living trees seems to be allergic in nature, and may not affect all horses or all people.

Black walnut trees are among the most beautiful and valuable trees in our forests. However, those considering planting black walnut trees, especially around horse pastures, should also be aware of potential problems. Horse owners and stable managers should inquire as to the composition and source of shavings used for bedding.

CALCIUM AND PHOSPHORUS NUTRITION

by Michelle Seavey, '86
with special thanks to Dr. Harold Hintz

Calcium and phosphorus are two minerals required in relatively large amounts by horses and other animals. Although calcium and phosphorus are best known as components of the skeleton, both have other vital roles in the body. Calcium is involved in muscle contraction and blood clotting, while phosphorus is essential for energy storage and metabolism.

Levels of calcium in the blood are regulated by three hormones. Parathyroid hormone acts to raise blood calcium by increasing resorption of bone and phosphorus excretion by the kidney. The active form of vitamin D enhances absorption of calcium from the gut and calcium removal from bone. Calcitonin acts to decrease blood calcium by decreasing bone resorption. Low levels of calcium or phosphorus result in a failure to mineralize bone, a problem called rickets in growing animals and osteomalacia in adults. The weakened bones are prone to bending and breaking. This is especially apparent in the leg bones because of their role in supporting the body. The effect on the other body systems is sometimes not readily apparent.

The amounts of calcium and phosphorus in the diet affect the levels in the body. It is important to have not only enough of each mineral, but also the proper ratio of calcium to phosphorus. This ratio ranges between 1:1 and 2:1. Diets containing high levels of phosphorus and low or marginal levels of calcium trigger secretion of parathyroid hormone, which stimulates resorption of bone to supply the blood with calcium. As the bones lose calcium over long periods, fibrous connective tissue invades the demineralized area. Fibrous tissue increases the size, but not the strength, of the bones. The swelling is most obvious in the head, thus leading to the name "big head disease".

How can you tell if your horse's calcium and phosphorus needs are being met? Signs of a problem may not be obvious until the bone abnormalities are advanced. Analysis of blood samples won't help, since homeostatic mechanisms are working to keep blood calcium at a normal level at the expense of bone strength. Hair analysis is also useless; research has shown that calcium and phosphorus levels in hair vary more with the season than with diet. Urine analysis may help, since low calcium intake causes low urinary excretion of calcium, while high intake promotes urinary calcium excretion.

The best method of determining a horse's mineral status is to compare its needs with calcium and phosphorus levels in its diet. The National Research Council (NRC) publishes tables listing nutrient requirements of horses in various stages of life. Requirements of both calcium and phosphorus are lowest in mature horses; they are increased by pregnancy, lactation, and growth. Research has shown that exercise increases the rate of turnover of bone, but not skeletal mass, indicating that working horses do not need higher levels of calcium or phosphorus. Although minerals are lost in sweat, an increase in feed intake to meet increased energy needs will supply the additional minerals.

Once the horse's needs have been determined, a balanced diet can be formulated. The NRC also publishes tables of the mineral content of various feeds. In general, grass hays are low in both calcium and phosphorus. Legume hays are high in calcium but low in phosphorus, and grains are high in phosphorus and very low in calcium. Mineral supplements should be added if calculations show that the diet is low in calcium. Since young horses cannot be depended on to consume enough of the appropriate mineral when it is offered free choice, the supplement should be mixed with the grain. A county extension agent or veterinarian can be consulted to help plan your horse's diet.

NUTRITIONAL MYOPATHY IN FOALS

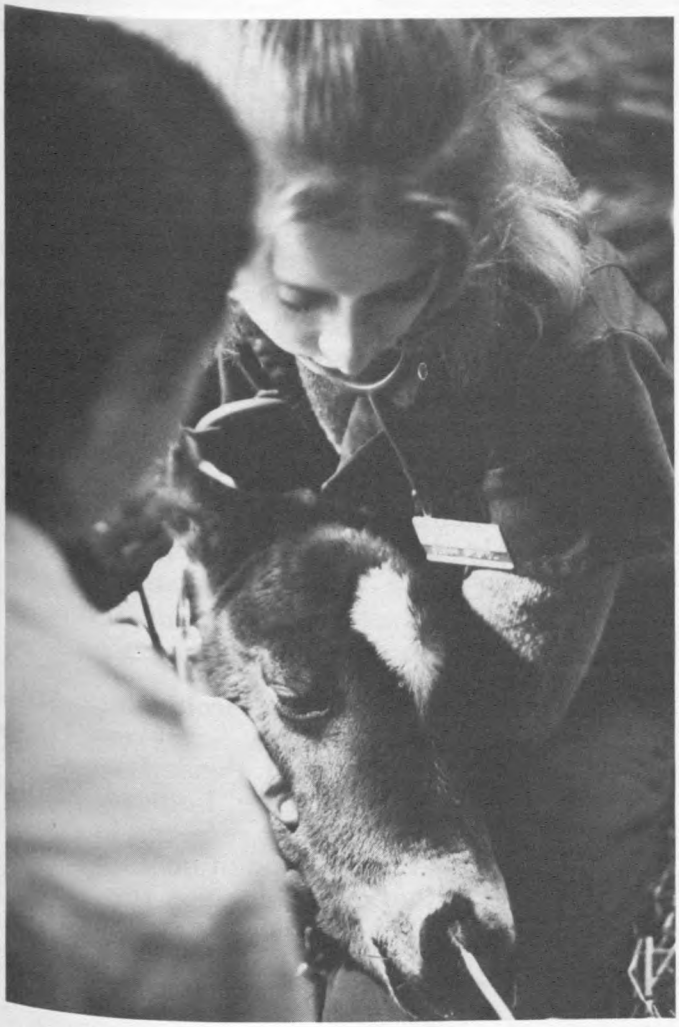
By Pamela Livesay-Wilkins '86

With Special Thanks to Dr. George Maylin and Dr. Donald Lein

Nutritional myopathy--also known as dystrophic myodegeneration, polymyositis, or white muscle disease--is a non-inflammatory degeneration of skeletal and cardiac muscle. Natural cases of the disease have been reported in Australia, the Netherlands, New Zealand, Great Britain, Canada and the United States. It is a disease reported primarily among young, rapidly growing animals of many species, and although the incidence of this disease is low in foals, it is seen more frequently in selenium deficient areas. Most of New York State and a large portion of the Northeast are selenium deficient areas.

Foals develop the disease most commonly in the first few months of life, usually in the first week. The foal may be affected suddenly, usually following a period of increased muscular activity, or clinical signs of lethargy, muscular stiffness or a stilted gait may be noticed first. Most frequently, the foal fails to suck and is recumbent, depressed, lethargic, stiff and lame if it does rise. The temperature is usually normal, although heart and respiration rates may be elevated. In the acute form, death of the foal can follow in as few as five hours, due to exhaustion and circulatory or cardiac failure when the heart muscle (myocardium) is severely affected.

The less acute form is the more common presentation, and because the extent and severity of muscular damage vary considerably, so do the clinical signs. Most foals are depressed and lethargic, but some remain bright and responsive. In the early phases some foals develop a steatitis (inflammation of fatty tissue) that results in a painful swelling below the skin that is most obvious along the crest, the ventral abdominal wall and over the gluteal region of the rump. Myoglobinuria (muscle



myoglobin in the urine) is seen in some foals, and the mucous membranes may be pale. Foals that are unable to stand may nurse if helped to their feet, but affected foals usually have difficulty sucking and swallowing which can lead to death from inhalation pneumonia or starvation.

Selenium/vitamin E deficiency syndromes have a variety of manifestations, but nutritional myopathy is the only well-documented disease entity observed in foals and horses. For all domestic animals, selenium and vitamin E (tocopherol) are essential nutrients that function in concert to maintain the functional integrity of cells. Vitamin E is a biological antioxidant that reduces peroxide formation in tissues. Selenium is incorporated into an enzyme, glutathione peroxidase, that is found in erythrocytes (red blood cells) and other cells in the body and serves to destroy peroxides that manage to slip by the vitamin E system. If peroxides increase their concentration in tissues, the end result is cellular destruction. Both selenium and vitamin E are required by the body to prevent all selenium/vitamin E related diseases since the two anti-peroxide systems are present in varying concentrations in different tissue types in the body. However, nutritional myopathy is a selenium responsive disease that can occur in the face of normal levels of vitamin E.

In selenium deficient areas, selenium and vitamin E are frequently administered in the management of musculoskeletal weakness, lameness, azoturia ("tying up") and reproductive problems in horses, as well as a preventative measure for nutritional myopathy. However, information regarding the exact status of these nutrients in equine nutrition is scanty, with much being extrapolated from other animal studies, making an accurate diagnosis of deficiency difficult. In an effort to address this problem, Dale S. Rubin in association with Dr. George Maylin and Dr. Donald Lein conducted a study at Cornell University to establish baseline data on selenium and vitamin E in the blood of New York State horses. In deficient areas, selenium/vitamin E deficiency syndromes appear most frequently at farms using only locally grown feeds, primarily during the spring and summer months. Onset of the disease depends on many variables including stress and such dietary factors as high levels of unsaturated fatty acids in the diet and decreased vitamin E levels in hays that have been stored for long periods or are weather damaged. Also, soils can appear to contain sufficient selenium levels, but it may be a form that is not bioavailable, meaning the horse can't use it.

Rubin et al. found that levels of vitamin E in horses declined through the winter months and rose again in the early spring, which is consistent with feeding stored hay throughout

the winter and turning the horses out to grass in the spring. Selenium levels stayed constant. A decrease in vitamin E levels throughout the winter, if combined with low selenium levels, could be a factor contributing to the onset of nutritional myopathy. Supplementation of selenium at the rate of 1 mg/day increased blood selenium levels above those associated with nutritional myopathy. This study also demonstrated that only limited amounts of inorganic selenium can cross the placenta, while mares supplemented with organic selenium during late gestation produced foals with selenium levels approaching that of their dams. Other studies have shown that excess selenium administered to lactating mares is not eliminated through the milk, so a deficient foal cannot be supplemented through the mare. (This study was supported by a grant from the Harry M. Zweig Memorial Fund for Equine Research.)

Thus, with nutritional myopathy, early recognition and treatment of the disease along with prophylaxis in selenium deficient areas are of utmost importance. Large broodmare farms feeding grains and hay grown locally may want to consider supplementation of their mares and foals. The effectiveness of treating foals that do develop the disease depends on how far the disease has progressed: animals that are recumbent and unable to suck are given a poor prognosis; foals with extensive myocardial degeneration can't be expected to recover.

It is probably best to consult with your local veterinarian to determine if your area is selenium deficient and to help you develop a prevention program for your animals, as too much selenium can also cause severe disease. In horses, selenium toxicity can result in alopecia (hair loss), weakness, tachycardia (increased heart rate) and may damage the hoof so that it is sloughed. Selenium is an essential nutrient, but its dietary levels should be judiciously monitored - too much or too little can result in severe problems for your horse.

OUR GUEST EDITORS: Dr. Harold Hintz, PhD, Cornell and **Dr. Herbert Schryver, DVM, Cornell, PhD, University of Pennsylvania,** are acknowledged experts in the field of equine nutrition. They both conduct research in equine nutrition and have joint appointments with the College of Veterinary Medicine and the College of Agriculture and Life Sciences.

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