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NUTRITION AND PRODUCTIVITY PRACTICAL PROBLEMS RELATED TO NUTRITION

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If you really can't figure out the problem it must be the feed. Many of us in the nutrition and feed manufacturing business have felt the frustration of having our formulas and our feed blamed for a myriad of problems on the horse farm and in the training stable. Many people engaged in the production of horses attribute miraculous healing and preventative qualities to feed and/or feeding management. This is all too easy to understand as the easiest change to make in response to a problem is to change the feed. The old saying goes, 'if it ain't broke, don't fix it.' It seems that the second part of the saying is: 'If it is broke, fix it.' Even if you don't know what's broke, fix it by changing the feed! Certainly much of the overall success of a breeding farm or racing stable can be attributed to accurate feeding programs and good feed management systems. This alone is why we as feed manufacturers and horse nutritionists have jobs. However to attribute all or a majority of the problems on a farm to nutrition is foolish. It will be the purpose of this paper to try to identify areas of production that are nutrition responsive and to try to explain the manner in which nutrition may be related to various problems that exist on the farm or in the training barn. Obviously time and space do not permit a thorough discussion of all of the various problems that may have a nutritional variable but we will try to discuss some of the more important ones. Problems of the performance horse were discussed in the 1992 proceedings of this course. A discussion of myositis, thumps, anhidrosis, COPD, and degenerative joint disease will be found in that publication. The problems and conditions below are still only a partial list of the problems that may have a nutritional origin or the manner in which nutrition is thought to influence productivity on the horse operation.

Nutrition and the broodmare

It is a common occurrence for people to erroneously attribute many fertility problems in the brood mare to vitamin deficiencies and to neglect to consider the single most important nutritional variable in reproductive efficiency - energy balance. More progress can be made in terms of increasing conception rates by insuring appropriate body condition than by using any other nutritional tool. This is not to say that meeting

vitamin, mineral and protein requirements is not important but from a practical standpoint energy balance tends to be more a concern. There is really no reason to assume that any species of animal is designed to conceive, carry a pregnancy to term and re-breed while in a negative energy balance. It is surprising that energy intake should be a major problem as it is really one of the easiest nutrients to assess.

Broodmares should be kept in reasonable flesh throughout the year. I would ask clients to try to practice what I would refer to as straight line broodmare nutrition. By this I mean that an optimum condition should be established for each mare and appropriate adjustments made in the feeding program to maintain this condition. This means that coming into the last trimester of pregnancy the mare should begin receiving more feed in preparation for lactation and that during lactation, feed intake should be increased to support the production of milk without excessive loss of body condition. During the final half of lactation the energy intake of the mare should be adjusted depending on condition at that point in time and on forage availability, again in hopes of maintaining optimum condition. Once a foal is weaned and the mare "dries up," an assessment of condition should be made and this period should be used to adjust the mare's condition back to optimal. This means that for the heavy milking mare that has gone through a tough lactation, we may want to continue to feed her at pre-weaning levels of feed intake and allow her to gain condition and for the mare that is fat we may want to not feed any feed and allow her to meet needs on good quality forage alone. Work at Texas A&M would indicate that mares that are a little too fat are far more reproductively efficient than are mares that are too thin.

For barren and maiden mares, plane of nutrition should be increased as the mares come into the breeding season. It makes good sense to increase nutrient intake when mares are put under lights so that an increase in condition is achieved at the same time as an increase in day length. This practice is referred to as nutritional flushing and from experience is effective in shortening the transitional period and increasing conception rates. This practice, done on mares to be bred in February and March, would mimic what would occur in nature when the spring flush of grass would result in mares increasing in body fat stores.

Dr. Walter Zent, considered one of the better reproduction practitioners, once remarked that if he were limited to one practice to increase conception rates in mares it would be "suturing" the mare. However, he added that poor conformation of the external genitalia of the mare is often as much a function of body condition as anything else. Thin mares are especially more prone to sunken vulvas that are apt to result in fecal contamination or wind sucking (pneumovagina). Especially in older mares, adding body weight may improve conformation and hence reproductive performance. Like Dr. Zent, as a nutritionist if I could do but one thing to the broodmare from a nutritional point of view it would be to insure energy intake to insure optimum body condition.

Another nutrient that I am always looking at in terms of reproduction is selenium. Maylin at Cornell reported that mares receiving inadequate selenium were much more prone to reproductive problems than were mares receiving adequate selenium. I try to insure that selenium intake in the mare is from 2 -3 mg/day.

Developmental orthopedic disease

Developmental orthopedic disease (DOD), also referred to in some circles as metabolic bone disease, consists not of a singular but of several related disorders affecting the young horse during the critical developmental stage. Included in the disease are: Epiphysitis (Physitis), Osteochondrosis (OC), Osteochondritis Dissecans (OCD), Wobbler Syndrome and acquired flexural deformity. At the outset it must be understood that all of these conditions are multifactorial in cause and are, for the most part, a related set of diseases. Furthermore, it is important that one realize that there is a strong genetic component which must not be disregarded. Whether the genetic component is a specific gene, or several genes that do not behave in a normal fashion, or simply as a result of selection pressure for one trait that resulted in the increase in the incidence of the problem, is not well understood. Certainly it is known that faster growing, earlier maturing, larger horses exhibit a greater tendency toward the disease than do their slower growing, smaller counterparts. The selection for early and rapid growth along with speed has seemed to bring with it an increased tendency to develop DOD and a greater need for understanding the problem. In other species, the problem has been diminished by eliminating offending individuals from the gene pool and from the standpoint of the purist this is not a bad idea. However, this approach is not feasible in most horse breeds. Those breeds that have put selection pressure on elimination of the problem have had some success yet this is the exception rather than the rule. The solution to the problem rests in an understanding of the possible causes, a realization of the genetic component and the establishment of management and feeding techniques that minimize the expression of these potentially catastrophic disease states.

Physitis

Actually the term epiphysitis is in itself a misnomer. The condition should more appropriately be referred to as physitis or metaphysitis. Defined simply, physitis is an inflammation of the physis (epiphyseal plate) which exist as a centers of ossification or growth in the bones of all of the mammalian species. Epiphyseal plates are found both at the proximal and distal ends of all of the long bones and are the means by which longitudinal growth occurs. The epiphyseal cartilage is dynamic and new cartilage is being formed as old cartilage cells are converted to bone. Physitis occurs when the cartilage growth plates become inflamed or when normal maturation of cartilage to form bone is disrupted. Although any growth plate can be affected, those most commonly thought of in relation to the disease are at the distal ends of the metacarpal bones, the metatarsal bones, radius and tibia. The incidence of the problem seems also to be in the order in which the sites were listed.

The "lesion" appears as a bony enlargement at the level of the metaphysis and is more frequently seen on the medial rather than lateral side. Epiphysitis may be accompanied by palpable amounts of heat in the affected areas and in some cases by lameness, though many young horses show varying degrees of clinical epiphysitis without exhibiting lameness. Causes of epiphysitis include but are not limited to:

Angular limb deformity	Very rapid growth
Nutritional deficiencies	Injury
Nutrient excess	Nutrient imbalance
Concussional damage	Genetic predisposition

One must be sure to understand those changes in joint architecture that are normal and distinguish between normal changes and physitis that is potentially threatening to development. It is entirely possible that subtle changes in the shape of joints is in the realm of what is normal in the developmental process and should not be misconstrued to be a real threat. It is also entirely possible that some mild physitis is a normal process or at least that what some people might be calling physitis is not outside the normal range of what is acceptable.

Treatment of physitis is varied depending on the severity and the cause (if one can be determined). In most instances it is advisable to reduce caloric intake such that the rate of growth of the affected horse is slowed. One of the most critical errors that is commonly made is to slow growth excessively and not only reduce the intake of energy but also of other nutrients critical to bone growth. It is far more prudent to try to continue to meet the horse's requirement for protein, minerals and vitamins than to simply quit feeding the affected horse. One of the ways in which this can be nicely done is by utilizing a protein, vitamin, mineral supplement fed at approximately 2 lbs.(1 kg.) per day. This feeding rate will of course depend upon the type of product that is available and how it is formulated. In addition to this "supplement" good quality hay should be offered ad-libitum. Once feed intake has been reduced, a professional assessment of the diet should be obtained. Nutrients of critical concern in doing a ration evaluation for young horses include protein, calcium, phosphorus, copper and zinc. If, after evaluation of the ration, one can find no reason from a nutritional standpoint that a problem should exist then other causes of the problem should be considered such as angular limb deformities and so on (see above). Even if the ration appears to be balanced in all respects it is probably best to proceed conservatively in terms of feeding rates until the problem is resolved. Many times it seems that mild physitis goes away in 60 days if it is treated and 2 months if it is not. In the final analysis it is not particularly alarming to see some inflammation of the physes just prior to closure. The major concern about physitis is that it may be the visual indicator that a more serious metabolic problem such as osteochondrosis exists.

Osteochondrosis

One of the more serious aspects of the DOD complex is osteochondrosis. Unlike physitis, osteochondrosis may result in a debilitating lameness in many instances reducing or eliminating any chance of an athletic career. Osteochondrosis is a disorder involving the maturation of joint cartilage. Aberrant cartilage formation results in the separation of cartilage from the subchondral bone resulting in the formation of subchondral cysts and cartilaginous flaps. When these lesions are observed, usually by taking X-rays, the condition is referred to as osteochondritis dissecans (OCD). It is most frequently seen in the ankle, hock and stifle joints. The lesions may involve the articular or non-articular surfaces, with the former posing the greatest immediate risk of lameness. Articular lesions which become detached from the subchondral bone may be surgically removed with variable degrees of success.

A number of causes of OC have been proposed, yet the specific etiology of the disease is far from understood. Interest in OC has been generated by a perceived increase in the incidence of the condition and a report from Ohio State University which suggests that the occurrence of OC may be the result of mineral deficiencies or imbalances. It is difficult to determine if indeed the incidence of the problem has increased or if the condition is being diagnosed more frequently due to superior radiographic techniques and equipment. With respect to the micro-mineral theory, careful balancing of rations containing liberal concentrations of copper and zinc have resulted in only a slight alteration in the occurrence of OC, emphasizing the multifactorial cause of the problem. Other factors currently under consideration include hypercalcitoninism as a result of high calcium intake in mares and hyperthyroxemia due to excessive carbohydrate intake. It is likely that genetic, nutritional and endocrine components contribute to the etiology and to obtain a decrease in incidence all must be considered.

Until the cause of OC is better elucidated it is appropriate to take a conservative approach to the nutritional management of affected populations of horses. Rations should be designed that meet but do not significantly exceed the horse's requirements for all nutrients, with specific emphasis on copper, zinc, manganese, calcium and phosphorus. Once formulated the ration should be fed in a manner that results in moderate growth rates and maximizes the forage rather than the grain part of the diet.

Wobbler syndrome

One of the earliest accounts of the wobbler syndrome was written in 1939 by members of the Department of Veterinary Science at the University of Kentucky. They described 47 cases which had occurred in central Kentucky between 1937 and 1939. Cases occurred particularly among Thoroughbreds and Saddlebreds, breeds which have long necks. Foals around weaning developed a lack of coordination in the hindlimbs,

which progressed to include the forelimbs, eventually causing the animal to stumble and fall. Well grown weanling and yearling colts seemed particularly prone, with three colts affected to every one filly. As a result of necropsy studies, it was suggested the condition was related to abnormalities of the vertebrae of the neck, which caused damage to the spinal cord. These observations were confirmed some 20 years later by Dr. James Rooney, also from the University of Kentucky, who identified more precisely the sites and nature of the lesions in the cervical vertebrae. Rooney suggested the overgrowth of the articular processes on which vertebrae move upon each other causes distortion and narrowing of the spinal canal and results in pressure and damage to the cord. The most frequent sites of lesions are between cervical vertebrae C3 and C7, although the presence of lesions does not always result in clinical signs of wobbler disease. When the neck is flexed, the lesions may cause pressure to be exerted on the spinal cord.

Clinical signs associated with wobblers may be related to other causes including trauma, parasitic infection of the spinal cord, and infection with equine rhinopneumonitis virus. In terms of prognosis it is therefore important to differentiate and establish an accurate diagnosis. Currently a true wobbler, the condition of which has recently been given the name cervical vertebral malformation (CVM), is confirmed by taking X-rays of the neck region. To do this, the horse must receive a general anaesthetic so that a technique know as myelography can be performed. This involves the injection into the spinal canal of a contrast fluid so that the space between the cord and the surrounding bony mass of the vertebra can be readily visualized. Narrowing of this space due to lesions of CVM can then be located. The procedure is not without its hazards and should only be undertaken by those who are experienced with the technique and its interpretation. Two types of lesions have been identified. The first typically affects horses from 4 to 12 months of age and occurs most frequently between vertebrae C3 and C4, and C4 and C5. It causes pinching of the cord only when the neck is flexed. The second affects horses between 12 and 36 months of age and occurs between vertebrae C5 and C6, and C6 and C7. Compression of the spinal cord is not relieved or exacerbated by flexion or extension in this region. Injury to the cord results from pressure which interferes with blood flow, causing damage to the cells comprising the cord. It is this injury which results in signs of incoordination, the severity of which are related to the extent and site of damage.

In the young horse destined to become a wobbler, osteochondrosis intervenes, allowing cartilage within the vertebra to develop in the absence of bone formation. The blood supply becomes inadequate leading to death of the surrounding tissue and the subsequent development of chronic joint lesions between the cervical vertebrae. What triggers these pathological changes at this critical growing period is still a matter of considerable debate. The initial suggestion that wobbler syndrome was an inherited condition linked to certain families has not been proven, although genetic influence has not been eliminated. By breeding two wobbler parents it has not been possible to

increase the incidence of wobblers in their offspring. It was noted, however, that the incidence of other bone deformities was increased.

It is interesting to compare the development of similar bone lesions, including spinal deformities, in other species, particularly in poultry and pigs, both of which have been subjected to intensive genetic selection and high planes of nutrition to improve growth rate and feed conversion. It is apparent that within these populations, genetic selection has contributed to an overall increase in skeletal problems. A similar situation may well have evolved in the Thoroughbred, with the current commercial incentive to produce a well-grown but nevertheless skeletally immature yearling in time for the summer and autumn (fall) sales. Foals and yearlings which receive a diet high in protein and energy have a critical demand for the correct balance of vitamins and minerals. This feeding level occurs at a time when the skeleton is still not capable of bearing increased muscle mass, nor is it able to respond to the strains and pressures imposed upon it. As a consequence, lesions of osteochondrosis may develop, causing CVM.

The prognosis for a wobbler has always been poor because of the progressive nature of the condition. However, within the last ten years a number of wobblers have been treated surgically. It has been reported that clinical improvement does occur in some cases but there is still considerable concern as to whether such animals should be allowed to participate in athletic competition. An alternative but less dramatic approach is to try to eliminate factors which might promote the wobbler condition, primarily by reducing the level of nutrition in the young horse.

Research workers at the New Bolton Center, Pennsylvania, have developed criteria for the early recognition of 'potential' wobblers using radiographic techniques devised by Mayhew of the Animal Health Trust, Newmarket. Treatment of the 'suspect' horses includes complete stall rest and a level of nutrition only slightly in excess of maintenance requirements. Horses on this program with which the author has had contact appear neurologically normal after completion of the treatment regime and by late in their two-year old year have achieved growth similar to that of their contemporaries.

Acquired flexural deformities

Acquired flexural deformities, also acquired contracted flexor tendons, appear frequently as a sequel to DOD. Rapidly growing horses that are erect in their pasterns are at most risk. The exact cause and an effective treatment remain elusive. Reducing growth rate, bandaging, surgery and tetracycline therapy have all been tried with varying degrees of success. The incidence of acquired contractures is high among horses that have been on a restricted diet and are then fed more liberally, a trend observed among horses in the USA and UK.

Fescue toxicity

Fescue toxicity is a condition primarily affecting pregnant mares grazing endophyte infected tall fescue pastures. Fescue (*festuca arundinacea*) is a cool season grass found in the central and northern regions of the United States that has desirable agronomic properties such as drought resistance, resistance to over grazing and insect damage and yields greater than most pasture species. However, mares grazing endophyte infected pastures exhibit a variety of reproductive problems that make the grass unsuitable for use for that class of horses. Symptoms of toxicity include prolonged gestations, tough thick and rubbery placentas, agalactia, premature placental separation and small weak foals. The causative agent in the toxicity is thought to be the endophyte (*acremonium coenophialum*) which inhabits the tissue of the fescue plant.

The endophyte is a potent vasoconstrictor and a strong prolactin antagonist, an antagonism which may be mediated via the prolactin antagonist melatonin. The entire mechanism by which the endophyte exerts its effect is not clearly understood. However, human placenta deprived of vascular supply shows the same tendency to become thickened and lose its characteristic fragility at parturition. This mechanism may explain the toughened placenta in mares grazing affected pastures. The depressed prolactin levels in affected mares would account for both the prolonged gestations and agalactia. At this time, the best recommendations for preventing the problem is to graze pregnant mares on pastures other than fescue. If fescue is the only pasture species available, mares should receive the predominant amount of their feed intake in the form of a good quality concentrate and properly cured non-fescue hay. This is particularly important in the last 90 days of gestation. If mares do not begin to make a significant udder within 10 days of their due date a toxicity problem should be suspected and the mares immediately removed from the pasture and fed liberal quantities of grain and a high quality legume hay as the condition of the mare allows. Because one of the major causes of foal loss due to the problem is neonatal asphyxia, a foaling attendant should be present at all births to assist the foal in breaking through the placenta. A ready supply of colostrum and a high quality milk replacer should be available in case the mare does not produce any milk. Many times affected mares will begin to produce adequate quantities of milk for the foal several days post-partum, but in the interim the foals' nutrient needs must be met using another source.

Nutritional secondary hyperparathyroidism

Nutritional Secondary Hyperparathyroidism (NSH), also known as millers disease, bran disease and big head disease, is caused by excessive mobilization of calcium from the bone under the influence of parathyroid hormone. The maintenance of blood calcium homeostasis is critical to the function of the muscular and nervous systems

and is therefore under close regulation of the hormones parathormone and calcitonin. When blood calcium levels fall below normal, calcium is resorbed from the bone under influence of parathormone in order to re-establish normal blood levels. If this occurs over an extended period of time, the bones become depleted of calcium, and lose their structural integrity resulting in lameness. The facial bones, depleted of calcium, become fibrotic and enlarged, hence the name big head disease. The condition occurs due to a dietary calcium deficiency, excess levels of phosphorus, an inverted calcium:phosphorus ratio in the total diet and high levels of oxalates in forages which interfere with digestion and absorption of calcium. In early times, millers fed the horses they used to work their mills the wheat bran that was a by-product of the mill. Bran is very high in phosphorus and low in calcium and many of the millers' horses developed this condition, thus the name millers disease or bran disease. In modern times the problem is most prevalent in instances when high levels of grain are being fed without calcium supplementation or adequate intake of good quality forage or when horses are grazing pasture high in oxalate content. Due to the strong homeostatic mechanism controlling blood calcium, analysis of the blood for calcium is not very valuable as a diagnostic tool. Instead the most accurate method of determining if this problem exists is by evaluation of the ration. The calcium intake of the horse should be determined and compared with the requirement of the specific class of horse in question. Additionally feed phosphorus should be determined to make sure that its level is not greater than that of calcium. Provided that an inverted calcium:phosphorus ratio is not being fed and that calcium levels are adequate this problem should then be limited to areas in which there are high levels of oxalates. Horses respond very nicely to supplemental calcium in the form of calcium carbonate and as such the problem of NSH should not be of concern on the well managed stud feeding a properly formulated diet.

Blister beetles

Blister beetle poisoning is confined to horses eating hay produced in the southwest where blister beetles are found in large concentrations. The toxin responsible for the expression of toxicity is cantharidin, which is present in the beetle and relatively stable over extended periods of storage. It takes only a few blister beetles, when ingested by the horse, to be fatal. Beetles are baled with hay, transported to the horse and then eaten inadvertently resulting in severe illness or death. The problem of blister beetles has become more pronounced since the replacement of the sickle bar mower with mower conditioners that crush the beetles rather than allowing them to crawl from the windrow as was the case for the older method of cutting hay.

Cantharidin, the toxin in blister beetles, is an extreme irritant to the digestive tract causing necrosis of the gut mucosa, the gastric mucosa and the lining of the esophagus as well as irritation to the urinary tract. Affected horses usually show severe colic

and discomfort, an elevated respiratory and heart rate, diarrhea and dehydration. Death usually occurs within 48-72 hours after ingestion of the beetles. The only treatment is that which reduces insult to the gut and includes tubing with mineral oil, fluid therapy and the use of analgesics such as butazolidin. The best way to control the problem is to feed hay produced in areas where blister beetles are not found or that produced from certified blister beetle free fields.

Colic

It is beyond the scope or intent of this paper to cover colic in its entirety. However, some mention of colic with respect to nutrition related causes is warranted. Since the advent of modern anthelmintics (dewormers), the most important cause of colic in horses is nutritional mismanagement. Nutritionally induced colic can be grouped into two categories: 1) improper forage:grain ratios or inadequate amounts of forage and 2) the use of tainted feedstuffs.

Far and away the more important of these two is the former. The horse is a wandering herbivore, a continuous grazer and has an absolute requirement for long stem hay or pasture. Failure to realize this simple fact probably results in more colics than any other single cause. The fact that hay or pasture is limited in some instances is made worse by the fact that in many of these cases the horses are on a high level of grain intake as well. On high grain diets rate of passage of ingesta is increased allowing more readily fermentable carbohydrate to reach the hind gut. Fermentation of this material many times results in a decrease in cecal and colonic pH under the influence of the lactic acid that is produced. This decrease in gut pH causes a shift in the microflora of the hindgut, which may result in the release of toxins and subsequent clinical colic. Additionally, in this type of etiology there is frequently an increase in the gas production in the hind gut that may lead to a flatulence and subsequent colic. The most effective way to reduce the incidence of colic on the farm or on the race track is to feed liberal quantities of good quality forage, limit grain intake to that necessary to maintain acceptable body condition and feed the grain portion of the daily ration in small meals and as frequently as is practical. Additionally it is a good idea to have hay available to mares even in the spring or other times when pasture growth is lush. In many instances there is simply not enough ‘gut-scratch’ factor in young lush growth. This combined with the increased rate of passage associated with a low dry matter feed leads to the appearance of more soluble forage constituents in the hind gut which again can lead to gas build-up and abdominal discomfort. There are very few if any instances when over feeding dry hay can lead to a problem, but there are numerous problems that can develop if hay is limited.

Although everyone is taught of the perils of feeding moldy hay to horses, this practice is not nearly as important a cause of colic as is under feeding forage. Nonetheless we would be remiss in not mentioning the potential for problems of the

gut if tainted or moldy feedstuffs are fed. The horse has a much lower threshold of pain than do cattle and as such can not tolerate GI tract insult inflicted by certain molds or the toxins they produce. Moldy hay and/or grain should not be fed to horses as certainly the risk of colic is increased. It is common in some parts of the world to see large round bales of hay and/or silage fed to horses. Although many times this is done successfully one must realize that there is an increased risk of gastric upset when either of these practices is used. Horses generally will not ingest moldy feedstuffs if given a choice, but moldy hay or spoiled silage can be deadly if the horse is forced to eat either. High quality forage products should be fed to horses. Feedstuffs fed to horses should be free of mold and other foreign debris, be of a forage species appropriate for the horse and should be properly cured and stored in a manner that will prevent their contamination.

Black walnut toxicity

The use of wood shavings from various species of trees is becoming more commonplace as labor cost of keeping horses becomes a greater concern. In most instances the use of shavings or saw dust provides an economical alternative to the use of wheat or rye straw or hay. The time necessary to clean stalls bedded in shavings is less than that required for straw and generally it is less expensive to use. Many soft woods and hardwoods alike are appropriate for use as a bedding material for horses. One type of shavings that should not be used for bedding is black walnut as severe founder (laminitis) has resulted from bedding horses on these shavings. Although the exact toxin has not been identified, it is probably the compound juglone in the walnut shavings that is the culprit.

Cystitis

Cystitis is an inflammation of the urinary tract that may be caused by the consumption of certain sudangrass and sudan-sorghum hybrids. Cystitis is characterized by an increase in urination or a dribbling of urine, incoordination and continuous estrus behavior in mares. The problem is most severe when horses are allowed to graze pastures that have been stressed. Drought, freezing temperatures and injury to the plant such as by trampling seem to increase the danger of these pastures when grazed by horses. Cystitis is caused when a glycoside in the plant is converted to prussic acid in the gut. Besides cystitis, prussic acid poisoning may cause muscle tremors, nervousness, respiratory distress and ultimately respiratory failure and death. Symptoms of prussic acid poisoning are also seen when wilted wild cherry leaves are ingested (hydrocyanic acid poisoning).

To prevent this problem one should not allow horses to graze sudan or sudan-

sorghum pastures such as sudex. Hays made from these pastures appear not to be toxic to the horse, but they generally are stemmy and of poor quality when compared to hays traditionally fed to horses. If one is forced to use sudangrass or sudan-sorghum pastures every attempt should be made to discontinue their use in periods of stress such as during a drought or immediately following a freeze.

Enteroliths

Enteroliths, also called stones or calculi, may form in the intestine of the horse when several criteria are met. Generally they require the presence of a foreign object such as a nail, stone, ball of hair, or other piece of debris. These objects serve as centers for the formation of the enterolith which is composed primarily of salts. Stones found in the gastrointestinal tract of the horse usually consist of magnesium ammonium phosphate.

The enterolith may cause symptoms ranging from weight loss and mild abdominal discomfort to an obstruction requiring surgical intervention. Size of the stone can range between the size of a pea to larger than a bowling ball. Smaller stones may be passed by the horse and cause no symptoms, but larger ones tend to cause an obstruction at the pelvic flexure of the large intestine or at the interface of the large and small colons. An affected horse may have only one enterolith or may have a large number of enteroliths of variable size.

The exact cause of the development of the stones remains uncertain and no specific dietary cause has been identified. The problem appears to be of the greatest magnitude in areas where access to fresh forage is somewhat limited rather than in areas where horses have access to an abundant supply of good quality forage. Some have indicted wheat bran, alfalfa hay and diets very high in iron as possible causes of the stones, yet no experimental evidence exists to corroborate that feeling. H.F. Hintz of Cornell University has suggested that feeding of vinegar, acetic acid, may reduce the occurrence and severity of the problem.

Wood chewing

The exact cause of wood chewing is not known. Horses that chew wood should be distinguished from the classical cribber in that they actually eat wood from fences, barns and other wooden structures rather than grabbing hold and sucking in air. Wood chewing may result from boredom, as in a stabled horse, from the learned habit, or possibly from a nutritional inadequacy. Experience suggests that frequent and vigorous exercise of stalled horses will minimize this behavior if it is boredom induced. Isolation of horses that chew wood from the rest of the herd in a paddock lined with electric fence will both reduce the behavior in the offending horse and

prevent other horses from picking up the habit. Although the two causes discussed above account for a fair percentage of horses that chew wood, the majority of horses that eat fences, trees and other wooden objects do so for other, less obvious reasons. Some of the more reasonable explanations are presented below.

Horses have an absolute requirement for long stem hay or fiber. When they are deprived of this requirement the frequency of wood chewing increases. Horses in the stall that are allowed minimal hay intake and horses in areas where fibrous feeds are in short supply due to inadequate rainfall or expense of importation are most inclined to begin chewing wood. In these instances the obvious and many times effective remedy is allowing for more fiber intake. As this will decrease the incidence of both colic and wood chewing it is probably most economical in the long run. Work at the University of Kentucky by Willard and others has shown a direct relationship between wood chewing and amount of fiber in the diet. Horses that were fed a concentrate diet low in fiber ate significantly more wood than horses allowed more hay. The class of horses that chew wood which are the most difficult to understand are horses on abundant, high quality pasture. These horses have access to plenty of fiber, are not in a stall most of the time and get plenty of exercise. So why do they chew wood and even more intriguing why does this behavior seem to be somewhat season related? Horses are most apt to exhibit wood chewing behavior in the times of the year when forage growth is rapid and lush grass is available. When this occurs the dry matter content of the forage is low, the crude fibre content is low, and the rate of passage is high. It is the author's opinion that the pH of the gut is decreased during this time due to a greater amount of soluble sugars in the forage reaching the hind gut. Additionally, the fiber content of the forage is so low that the effective fiber is inadequate to meet the horse's requirement for long stem fiber. In an attempt to increase the amount of effective fiber in the diet the horse eats fences, tree bark and other wooden objects. One way in which one can reduce the frequency of wood chewing in pastured horses is to continue to provide long hay to the horses during the period when rapid growth of low dry matter forage occurs. It is surprising how much hay horses will continue to eat when there is an abundance of lush pasture growth available. In addition to reducing the wood chewing problem it has been the author's observation that flatulence colic associated with horses grazing these lush pastures is reduced as well when hay is offered free-choice during extreme pasture growth phases.

Urea toxicity

Urea and biuret are non-protein nitrogen sources commonly used in the diets of ruminant animals. These feed ingredients provide nitrogen to the intestinal (ruminal) microflora for use in the synthesis of microbial protein which may then be used to meet a significant portion of the ruminants' protein requirement. The arrangement of the horse's gastrointestinal tract makes the use of urea questionable in terms of

contributing to their nitrogen requirements. Studies conducted at universities in the US indicate that the horse receiving marginal protein intake can benefit from urea, especially mature horses on a forage diet. However, urea may also be toxic to the horse.

Contrary to popular belief, horses are not extremely sensitive to urea and in fact show greater tolerance to urea than do ruminant animals. Urea is generally broken down in the stomach and small intestine of the horse, absorbed and excreted via the kidneys prior to the time it arrives in the cecum. Therefore, urea is not broken down to form carbon dioxide and ammonia, the latter of which causes the toxicity to occur in the ruminant animal. This does not mean that urea is not potentially toxic to the horse. Hintz and co-workers fed ponies a diet which consisted of twenty-five percent urea and resulted in the death of the ponies to which it was fed. The classical symptoms attributed to urea toxicity in the horse are incoordination, wandering and head-pressing (horses will stand pressing their heads against solid objects). Although it is generally not recommended, horses are tolerant of urea-containing ruminant diets and indeed older horses may make use of some of the non-protein nitrogen. Urea should not be fed to young horses at all. A note: feeding rations designed for one class of livestock to another class of livestock for which it is not intended is never a good idea. For instance, even though horses can tolerate the NPN levels practical in ruminant diets, they are intolerant of ionophores which might be present in those diets.

Ionophores

Ionophores are antibiotic-like compounds used in ruminant diets to alter rumen fermentation. These compounds cause a shift in the volatile fatty acid (VFA) ratio that favors the production of propionate and reduces the molar percentage of acetic acid. The net result of the action of ionophores in ruminant diets is greater feed efficiency. The two most common ionophores used are MONENSIN SODIUM (rumensin) and LASALOSID (bovatec). Both of these ionophores are extremely toxic to the horse. As such feeds containing ionophores should NEVER be fed to horses. There have been several instances in the U.S. when ionophores have mistakenly gotten into horse feed and have resulted in the death of horses. Litigation in these cases is ongoing.

Levels of rumensin as low as 1 mg/kg of body weight have resulted in the death of horses. However the LD-50 (level at which 50 % of horses would be expected to die) is reported to be 2-3 mg/kg of body weight. This means that 50% of horses that consumed 1,000 mg of rumensin would be expected to die. The normal inclusion rate of rumensin in cattle feed is 3 -5 grams per ton (3.3 - 5.5 ppm). This means that a 500 kg horse would have to eat from 300 -500 kg of a feedlot cattle ration for it to be toxic. These feeds should not result in toxicity. The real hazard with ionophores is errors by feed manufacturers, or with horses consuming pasture cattle supplements

or poultry feed which could contain 400 and 100 ppm ionophore respectively. Affected horses show restlessness, colic, sweating, and incoordination and generally die within 12-36 hours of the onset of toxicity symptoms. Post-mortem examination shows severe damage to the cardiac (heart) muscle. In horses that have shown signs but recovered, cardiac damage is frequently evident such that a return to athletic performance is questionable.

Rumensin and other ionophores are frequently used in cattle feed as a coccidiostat, especially in the diets of replacement dairy heifers. Additionally, ionophores are used in poultry feeds to control coccidiosis. These facts and the extreme toxicity of ionophores to horses make feeding cattle feed or poultry feed to the horse risky. Many feed mills that make a significant amount of horse feed will not make ionophore containing feed due to the possibility of contaminating horse feeds and the potential for liability that exists.

Heaves (Chronic Obstructive Pulmonary Disease)

Heaves has long been associated with horses not sound of wind. It is characterized by hyper-reactive airways, and an allergic response similar to asthma in humans. Affected horses show coughing, forced expiration of air and a heave line in the region of the base of the ribs associated with muscular hypertrophy caused by effort of expelling air from the lungs. Horses with this condition frequently have a nasal discharge. The lungs lose their elasticity, which results in a heightened respiratory distress. Affected horses tend to be very hard keepers and their athletic ability is impaired, especially in events where maximal effort is required.

The exact cause of heaves is not clearly understood, nor is there an effective cure. Hence, management of the disease is the sole way in which it can be coped with. Most of the time horses with heaves are best kept outside in the pasture rather than in a barn. Keeping the horses' environment as free from dust and mold spores as possible is the most effective way to treat the disease. Heavey horses should be bedded on paper, clean wood shavings or even on a rubber mat in preference to bedding on straw or hay. Hay should be wet prior to feeding. This practice minimizes the extent of dust, mold spores and other airborne particulate matter. Frequently the use of fiber sources other than long hay can minimize allergic reactions. Cubed grass or lucerne hay, shredded beet pulp, and mashes made with oaten or wheaten chaff are alternatives to long hay.

In addition to nutritional management, several other things can be done to reduce the problem of the disease to affected horses. Never clean the barn with a heavey horse inside. Sweeping, dusting, moving hay into and out of the loft, and other activity in the barn increases the amount of airborne particulate matter which results in an increase in allergenic response of affected horses. Provide for good ventilation in the barn. Airflow increases the fresh air available to the horse and helps to dissipate the

ammonia in the stall. Ammonia, like dust, can serve as a severe respiratory irritant and as such pose a problem for the horse with hyper-reactive airways. Chronic, heavey horses may also benefit from the use of expectorants to reduce coughing and of bronchodilators to allow for eased breathing. When possible, do not restrict the heavey horse to the stall. Horses maintained in pastures are less likely to develop heaves, and are better able to cope with the disease once affected.

Goiter and iodine deficiency

Goiter, an enlargement of the thyroid gland, may occur in response to deficient (hypo-iodine goiter) or toxic (hyper-iodine goiter) levels of iodine in the diet. Iodine is a component of the hormone thyroxin produced by the thyroid gland. When iodine levels in the diet are inadequate to meet the horse's requirement the thyroid gland becomes enlarged, which is indicative of thyroid disfunction. Thyroxine is responsible for regulation of metabolic rate and as such hypothyroid horses may exhibit slowed skeletal growth, rough hair coat, delayed shedding of hair and muscular weakness. Foals born to thyroid hormone, iodine deficient, mares are frequently dysmature appearing and lack hair and may or may not exhibit an enlarged thyroid gland. Supplementation of horses with iodized salt is generally adequate to meet iodine requirements, although most modern diets contain levels of iodine higher than those that would be obtained from consuming iodized salt alone.

If goiter appears in the presence of what appears to be adequate levels of iodine in the diet, then an iodine toxicity must be suspected. Workers at Cornell University in the US have reported goiter in foals born to mares consuming high levels of a supplement containing kelp, a seaweed high in iodine. Levels of iodine intake as low as 50 mg/day by pregnant mares have resulted in goitrous foals. As such random supplementation of mares with high iodine supplements is not a good practice and the iodine provided from these supplements should be considered in assessing the iodine status of the horse's diet. The symptoms of hyper-iodine goiter are very similar to those seen in instances of hypo-iodine goiter and as such evaluation of the diet is the first diagnostic tool to use in determining the cause of the problem. The current NRC publication, Nutrient Requirements of Horses, suggests the iodine requirement of the young growing horse is 0.1 mg/kg of diet while 5 mg/kg of diet is thought to be the potentially toxic level of dietary iodine.

Goiter may also be caused by goitrogenic substances in the diet that exhibit an antithyroid activity. Feedstuffs known to exhibit antithyroid activity include un-cooked soybeans, cabbage, kale and mustard. The enzyme responsible for the antithyroid effect is inactivated by heating and cooking and as such properly prepared soybean meal is safe to feed. The goitrogenic effect of these feeds is not effectively inhibited by supplemental iodine, suggesting that, with the exception of properly heated soybean meal, they should be avoided in equine diets.

Selenium deficiency and white muscle disease

In areas of the world where selenium levels in the soil and forages are inadequate, selenium supplementation is necessary to prevent the appearance of selenium deficiency. Although the current NRC requirements suggest that the selenium level of the diet be 0.1 ppm, most well formulated equine diets contain a level of from 0.2-0.4 ppm. The most common manner in which selenium supplementation is achieved is by adding sodium selenite to the grain mix.

Selenium deficiency is characterized by white muscle disease in foals. The skeletal muscle is pale to white in color due to degeneration of the muscle cells. The cardiac muscle is also affected. Foals exhibiting white muscle disease are frequently weak at birth, suffer from respiratory distress and have difficulty nursing. Due to the loss of muscle integrity respiration is impaired and foals often die of respiratory failure. The serum selenium level and plasma glutathione peroxidase (a selenium dependent enzyme) concentration of mares has been effectively used as a predictive diagnostic tool in averting the incidence of white muscle disease in foals produced by mares grazing selenium deficient pastures. The expected normal range of serum selenium in selenium adequate mares is from 0.06 to 0.15 ppm (mg/100ml), although mares at the lower end of the expected range have been known to produce foals with white muscle disease. Plasma glutathione peroxidase concentrations of less than 20 units are indicative of marginal selenium status and horses with this low value should be treated by either addition of dietary selenium or by injection of selenium. It is worth noting that Se-E injections have resulted in anaphylactic shock on occasion and as such should be administered with care.

Myositis or tying-up

Myositis, or tying up, is an exertional myopathy common to performance horses. Another condition with much the same etiology and effect is azoturia. Some would disagree with grouping, for purposes of discussion, myositis and azoturia. However in practice these two conditions appear to be a continuous progression of muscle dysfunction rather than distinctly different disease entities. Symptoms common to the two conditions are an elevated level of the muscle enzymes AAT (aspartate amino transferase) and CPK (creatine phosphokinase), decreased muscle pH, stiffness over the loin and back, an unwillingness to move, and some degree of myoglobinuria (myoglobin is a muscle pigment which appears in the urine after damage of the muscle membrane and muscle fibers). The urine of affected horse takes on a characteristic coffee color indicating the presence of myoglobin in the urine. The exact cause of these conditions is not known.

Myositis or tying-up occurs most frequently in athletic horses of light horse breeds. It may occur at any time from the beginning of an exercise bout to several hours

post-exercise. Most frequently there is an obvious change in the horse's gait which rapidly progresses to a reluctance to move at all. Affected horses should not be forced to move as this makes the damage to the already compromised muscle worse. Definitive data that would clearly elucidate the mechanism for this muscle problem are lacking but clinical evidence suggests that affected horses may benefit from Se-Vitamin E injections. The dual roles of these two nutrients in maintaining muscle membrane integrity and preventing tissue peroxidation suggest that they may be involved in the problem. Elevated levels of AAT and CPK characteristic of horses having been through a bout of tying up are indicative of muscle membrane damage and leakage of these two enzymes into the circulation and it stands to reason that selenium and vitamin E therapy in horses prone to tie-up is probably warranted. Even though some horses respond to supplementation, others do not. Myositis is a management related problem in many instances. Failure to allow a horse to warm up properly, intake of high carbohydrate diets in conjunction with irregular and inappropriate exercise programs and asking a horse to do more than it is fit enough to do all contribute to the occurrence of tying-up syndrome. Additionally nervous fillies are more inclined to develop the problem than are colts and once afflicted, horses are much more inclined to tie up again. As a general rule the following management practices should reduce to an absolute minimum the incidence of tying-up.

1. On days that a horse is not to be worked cut the feed allowed by one-half and allow at least some exercise even if it is only 10-15 minutes on a longe line or 20 minutes of hand walking.
2. Try to meet at least 50% of the horse's requirement for DE using fibrous feeds.
3. Allow for adequate time to properly warm up and cool down the horse. An effectively managed cooling down period allows for the muscle to return to resting state more rapidly, encourages the lactic acid in the muscle to diffuse into the blood more quickly which in turn decreases muscle acidity and will effectively prevent some of the muscle soreness associated with intense exercise.
4. Use training techniques that minimize physical and psychological stress while allowing the horse to get fit enough to perform the task for which it is intended.