Protein-Energy Malnutrition in Ruminants

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The term protein-energy malnutrition (PEM) has been used[18,19] to describe insufficient provision of feed, in terms of quantity, quality, or both, to ruminant livestock. The term corresponds to the established nomenclature in both the human and agricultural literature. It does not convey the negative connotations of the word starvation, yet it still accurately describes an affected animal's condition. Other terms that are used to describe underfeeding include unintentional starvation, inanition, thin pregnant cow syndrome, emaciation, hypoalimentation syndrome, starvation syndrome, and a variety of informal colloquial terms often used by laypersons. A consistent syndrome of clinical signs accompanies PEM.[18-20]

Complete starvation secondary to total absence of food is quite rare, and usually involves either overt animal cruelty or gross errors in management. On the other hand, incomplete starvation (which includes PEM) is quite common, but is not often openly discussed by either livestock producers or veterinarians. When conditions of general underfeeding, such as PEM, exist, the resulting protein and energy deficiencies cannot be strictly separated. Energy is the nutrient whose intake is most severely restricted in most cases of PEM. However, pure energy deficiencies are usually complicated by protein deficiency because of the typical composition of ruminant feedstuffs (high in energy and low in protein) and the high expense of supplemental protein. Similarly, pure protein deficiency (kwashiorkor) may accompany unusual feeding conditions, but results in appetite depression and ultimately a secondary energy deficiency. Vitamins and minerals usually do not play a major role in PEM, as most are required in proportion to the amount of energy metabolized.

It may be difficult to distinguish between primary PEM (simple

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underfeeding) and secondary PEM, which is attributable to anorexia resulting from a primary disease condition. In secondary PEM, adequate nutrition is available to the animal, but it is not willing or able to eat. Disease further complicates secondary PEM by increasing basal metabolic rate as a result of fever and conditions of inflammation, tissue destruction, or stress.[8] This article will emphasize primary PEM rather than the much broader but more easily recognizable condition of secondary PEM.

PATHOPHYSIOLOGY OF PROTEIN-ENERGY MALNUTRITION

When only suboptimal supplies of protein and energy are available, they are distributed within the animal in such a way that requirements for maintenance receive highest priority. The animal can further respond by decreasing its maintenance requirements through lowering its basal metabolic rate. Decreased thyroid hormone concentrations mediate this effect. Decreasing organ size may further reduce basal metabolic rate.

Requirements for growth and reproduction take a lower priority. Both can be curtailed drastically in order to increase the animal's likelihood of survival. If conception occurs before the onset of PEM, however, the products of conception have the same priority for nutrients as the dam does for maintenance. Because of this, pregnant females suffering from PEM will mobilize additional maternal nutrients to meet fetal requirements.

Body stores of protein and energy begin to be mobilized when nutrient intake falls below maintenance requirements. Carbohydrate stored as glycogen is the first energy store to be catabolized in response to negative energy balance; however, glycogen stores in the ruminant are limited and are depleted rapidly. Body stores of lipid and protein must then be catabolized to meet energy requirements. Lipolysis begins when insulin concentration declines in relation to glucagon concentration. This results in mobilization of nonesterified fatty acids from depot fat. Large amounts of energy are released if these acids can be completely oxidized in the tricarboxylic acid cycle. Incomplete oxidation and ketone body formation may result if the energy deficiency is sudden and there are high absolute demands for glucose, such as in pregnancy or lactation. However, in PEM, the onset of energy deficiency is gradual, and in nonpregnant or early-gestation animals, the demand for glucose is moderate; thus, extensive ketone body formation does not occur in PEM.

Proteins may also be catabolized to meet energy requirements and particularly to maintain critical blood glucose concentrations. Only a small amount of blood glucose is derived from lipolysis, that being from the glycerol portion of the hydrolyzed triglyceride. Therefore, the major substrates for hepatic gluconeogenesis become amino acids from muscle breakdown. Protein catabolism is initiated and maintained by decreased insulin and increased glucocorticoid secretion (see the arti-

cle by Herdt entitled "Fuel Homeostasis in Ruminants" elsewhere in this issue).

The liver is the organ that undergoes the most profound physical and functional changes during PEM. Because hepatic proteins are very labile, they are quickly catabolized during protein deficiency. The liver loses weight faster than other organs during prolonged PEM. Fatty infiltration of the liver is often evident in acutely starved animals, but is rarely observed in ruminants with PEM because the latter mobilize fat slowly and are able to utilize it before it accumulates in the liver. Hepatic enzyme function is impaired by PEM; this helps to explain the tendency for malnourished animals to be more susceptible to the effects of toxins than their well-fed counterparts.[15]

Loss of muscle mass and muscular power occur insidiously during PEM, and are eventually evident as lethargy and weakness. More than 60 per cent of the total body protein lost during protracted starvation is from muscle and skin.[8]

Ruminants, because of their large alimentary tract capacity, have a fairly effective buffer against the consequences of short-term malnutrition; however, long-term malnutrition eventually exhausts the store of digestible nutrients in the rumen. As PEM progresses, ruminal microorganisms decline in number because, like their host, they are being deprived of substrates for metabolism.[5] Protozoa are particularly sensitive to nutrient deficiencies and may be absent in animals suffering from PEM. The digestive and absorptive capacity of the rest of the alimentary tract is also adversely affected by PEM. Maldigestion secondary to lack of digestive enzyme synthesis, and malabsorption resulting from edema of the bowel wall result in less efficient utilization of whatever feed is available. This contributes greatly to the rapid clinical deterioration observed in animals with PEM. These digestive impairments also help to explain the tendency for ruminants to recover very slowly from periods of PEM; malabsorption and maldigestion remain while ruminal microbial populations become reestablished. Digestive enzymes must also be replenished and lower intestinal tract function must be restored before the animal can recover. When adequate diets are offered to malnourished ruminants, diarrhea commonly ensues, probably as a result of the osmotic activity of unabsorbed nutrients in the large intestine.

Reproductive capabilities of ruminant livestock may be drastically impaired by PEM. Malnutrition in developing animals results in delayed sexual maturity and decreased libido after puberty does occur.[1] Prolonged PEM may cause partial or complete cessation of the estrous cycle. Once conception has occurred, however, fetal development is unlikely to be affected adversely. Most pregnant females with PEM do not abort, even if they eventually die. A severely emaciated animal will only occasionally abort or deliver prematurely, with that occurring just before its own death.

Beef cows that are malnourished during the last trimester of pregnancy have significantly decreased milk production, decreased calf birth weights, increased calf mortality, decreased calf weaning weights,

and increased age of their offspring at puberty.[7,22] Similar reproductive impairment may be expected in malnourished ewes and goats. Malnutrition may also increase the incidence of dystocia secondary to muscular weakness of the dam. Mortality of calves born alive to malnourished dams is increased; this may be attributed to calf weakness at birth and probably, more importantly, to poor maternal colostrum and milk production. Low maternal protein intake during the last trimester of pregnancy has been directly associated with weak calf syndrome in beef cattle.[4]

Subclinical PEM causes major economic loss in the cow-calf industry by increasing the interval from parturition to first estrus and by impairing fertility when estrus does finally commence. Inadequate protein and energy intake in sheep and goats around breeding time likewise impairs fertility and also causes the development of fewer ova, which ultimately lowers the birth rate.

The effects of malnutrition on the immune system are extensive. Malnutrition generally increases host susceptibility to bacterial infection via reduced cell-mediated immune response, reduced antibody production, and reduced complement concentration.[10,25,28] Resistance to parasitic infection may also be decreased by PEM. However, poorly nourished animals are usually more resistant to viral infections than their well-nourished counterparts. This may be explained by impairment of those host metabolic functions that are essential for viral replication within the host.[26]

CLINICAL HISTORY AND EVALUATION

An accurate history is especially important when attempting to establish a diagnosis of PEM in the field. Questions should be asked to determine the factors that may have been responsible for insufficient protein and energy intake. Specific factors to consider include drought, overgrazing, floods, and heavy snow cover. Large hay packages, such as round bales or compressed stacks, are frequently associated with less than expected nutrient intake because they are prone to weather deterioration and waste. In addition, the weight of large bales is commonly overestimated. Sometimes the roughages available to feed ruminant livestock are of such poor quality that the animals cannot consume enough to keep from starving, even when such roughage is available free choice. Poor forage quality may be the result of excessive maturity of harvested hays, weather damage of harvested hays, improperly managed dormant winter pastures, or winter grazing of crop residues without adequate supplementation. In some instances, a complete ration evaluation may be necessary to establish nutritional inadequacy. Accurate feed consumption data, which are often difficult to obtain, may also be necessary. Keep in mind that owners tend to overestimate the nutritional quality of their forages and the dry matter intakes of their animals.

A useful history should also include facts about the animal that could influence its nutritional requirements. Protein-energy malnutrition is most common in ruminant livestock during periods when animals

have relatively high requirements for energy and protein. Late pregnancy is the time in a breeding female's life cycle when her nutrient requirements are high because they include the nutrient needs of the fetus. It follows that most clinical cases of ruminant PEM occur in females during the last trimester of pregnancy. The probability of PEM is compounded by growth requirements if the animal is in her first pregnancy and has not reached adult body size.

Cold temperatures compound the high nutrient requirements of late gestation, and unfortunately, the coldest months of the year usually correspond to the final months of gestation. Cold stress dramatically increases an animal's energy requirement as more energy is spent simply to maintain body temperature. Young animals, animals with short haircoats, animals exposed to wind along with the cold, and animals with wet or mud-caked hair are particularly susceptible to the nutritional drain of cold weather.[17]

A veterinarian is usually not called to a malnourished herd or flock with PEM until one or more animals have become recumbent. Animals affected with PEM often appear to have been normal one day and to have then deteriorated and become recumbent the next. This quick onset of clinical signs of PEM is common, despite the fact that PEM is a chronic, progressive disease. Digital palpation over the backbone and the ribs is often necessary to reveal the marked loss of body condition that has been progressing, unobserved, for weeks or months.

The alert mental state of a recumbent animal suffering from PEM helps to differentiate it from other chronic, debilitating diseases. Rectal temperature is often decreased in animals suffering from PEM, even if the animal is housed in a warm environment. Hypothermia occurs as the result of lowered basal metabolic rate and continued loss of body insulation. Animals with PEM usually have normal or avid appetites, which is not characteristic of other chronic diseases. Heart and lung sounds are typically normal, although lung sounds may be slightly harsh on the dependent side of the animal as a result of passive congestion. The rumen often has decreased motility and is usually full of sparingly digestible, poor-quality roughage. Diarrhea secondary to maldigestion and malabsorption may be present in advanced cases, particularly if the owner has attempted to renourish the animal with high-concentrate feeds. A thorough physical examination should be performed on animals suspected of having PEM. Special attention should be directed toward finding conditions that could cause anorexia and secondary PEM, such as blindness, trauma, chronic degenerative joint disease, foot problems, or other musculoskeletal problems.

Beef Cattle

The most common class of ruminant livestock diagnosed with PEM is the beef cow in late gestation. Spring calving herds are much more likely to be affected than are herds that calve in the fall. Beef cows with PEM are commonly not presented to the veterinarian until after they become recumbent. Although they usually continue to eat avidly, these cows typically die in about 7 to 14 days after becoming recumbent, with or without treatment.

Protein-energy malnutrition in beef cows may be complicated by abomasal impaction, particularly if the cows were consuming very coarse forages or had limited access to water. Hypocalcemia is occasionally associated with inadequate feed intake and may complicate PEM.[11] The differential diagnosis of recumbency in overwintering beef cows also includes Johne's disease, bovine lymphosarcoma, chronic peritonitis, internal or external parasitism, chronic pulmonary disease, vitamin E or selenium deficiency, locomotor difficulties, dental disorders, behavioral problems, pregnancy ketosis, nonparturient hypocalcemia, and toxicoses.[19]

When pregnant beef cows suffering from PEM do survive through parturition, their inability to produce sufficient colostrum and milk may result in clinical signs of PEM in their calves. This problem is most severe in malnourished first-calf heifers, whose calves often die of PEM. Such calves typically try to nurse persistently and eat dry feed, drink surface water or urine, and bawl for several hours at a time. They eventually become recumbent and die. In beef calves, PEM can be differentiated from neonatal diarrheas and septicemia by the absence of diarrhea or fever.

Dairy Cattle

Standard management and nutritional practices for adult dairy cattle usually include intensive management and a high plane of nutrition; thus, PEM is a rare condition in adult dairy cattle. If dairy cow nutrition is inadequate, it is usually promptly detected because of poor milk production or, occasionally, because of primary ketosis. Pregnant dairy heifers that are fed poor-quality roughages during the winter months are the most likely candidates among adult dairy cattle to develop PEM. The term 'thin pregnant cow syndrome' has been used to describe dairy cows in poor condition during late pregnancy.[23]

Protein-energy malnutrition may also occur in nursing dairy calves fed artificial milk replacers during periods of cold weather. This condition has been termed emaciation[27] or clinical cold stress.[2] Deaths are most frequent when very low-quality milk replacers are fed, or when an insufficient quantity of a good-quality replacer or milk is fed to calves housed outdoors during cold weather. Milk replacers of good quality use milk sources of protein, such as skim milk powder, dried whey, or casein, rather than nonmilk sources, such as soy flour, wheat flour, or distiller's dried solubles.

Dairy calves affected with PEM are typically 3 to 4 weeks of age. They become progressively thin, inactive, and lethargic before death. Hypothermia is a consistent clinical feature; rectal temperatures range from 32.2°C (90°F) to 36.7°C (98°F). Affected calves may die suddenly in convulsions from hypoglycemia. Appetite remains normal or avid in affected calves; they may even behave in an excessively friendly manner, as though they are looking for food and warmth.[2] Diarrhea resulting from maldigestion and malabsorption is common just before affected calves die; this often confuses the diagnosis because of its resemblance to colibacillosis, viral diarrheas, or cryptosporidiosis. If diagnosed early enough, malnourished calves can recover quickly with

proper feeding and shelter from the cold. Whole milk is the best food source for malnourished calves during recovery from PEM.

Goats

Protein-energy malnutrition is less common in goats than in other ruminant species, probably because they are not often stressed for production and because they have a superior ability to digest roughages. Goats are able to store large amounts of fat internally, which also enhances their ability to survive long periods of nutrient shortages. However, these qualities are occasionally overestimated by goat owners, who might place goats in unimproved or woodland pastures where the amount of forage is severely inadequate. When forage is sparse, pastured goats must expend large amounts of energy just traveling to get to their food. Maintenance requirements of stable-fed goats are, therefore, considerably lower than those of their pastured counterparts. It is a commonly held but mistaken belief that dairy goats should be fed high levels of oats. Addition of greater than 25 per cent of oats to goat rations will, in fact, seriously curtail digestibility of the total diet[9] and may eventually culminate in PEM.

Because goats maintain a rigid social order, it is particularly important that each animal be given adequate housing surface area and feeder space. Otherwise, malnutrition of individuals low in the social order will result. Nubian goats, because of their submissive social nature, should not be mixed with other breeds in intensively managed herds.

Clinical signs of PEM in both adult and neonatal goats are very similar to those previously listed for cattle. The differential diagnosis of protein-energy malnutrition in goats includes systemic caseous lymphadenitis, Johne's disease, internal or external parasitism, chronic pulmonary disease, chronic degenerative joint disease (usually resulting from mycoplasma or caprine arthritis-encephalitis infection), chronic peritonitis, cobalt deficiency, dental disorders, locomotor difficulties, behavioral problems, and toxicoses.[19]

Sheep

Protein-energy malnutrition is common in sheep but is infrequently called to the attention of veterinarians; this is a reflection of the low economic value of individual ewes. Sheep owners are often aware of the presence of PEM in their flock, but cannot justify the cost of an intervention such as supplemental feeding. Protein-energy malnutrition in sheep has the unique effect of reducing wool quantity and quality (for example, breaks in the wool); this represents an additional economic loss associated with PEM in sheep.

Excessive dental attrition is a common cause of secondary PEM in range ewes. Dental attrition may be caused by soil ingestion while grazing, dietary calcium deficiency, calcium–phosphorus dietary imbalances, or all three. Dental attrition alone reduces the productive life of range ewes by 2 years compared to their farm-fed counterparts.[12]

Complete starvation (often with secondary pneumonia) is a major

cause of death in newborn lambs. A newborn lamb that never nurses will survive only 3 to 4 days. Clinical signs in lambs and adult sheep with PEM resemble those mentioned earlier for cattle. The differential diagnosis of emaciation or recumbency in adult sheep includes chronic progressive pneumonia, pulmonary adenomatosis, internal or external parasitism, systemic caseous lymphadenitis, chronic peritonitis, Johne's disease, copper deficiency, cobalt deficiency, locomotor difficulties, subacute blue tongue infection, behavioral problems, and toxicoses.[19] Ovine pregnancy toxemia (ketosis) is caused by sudden onset of nearly complete starvation or anorexia in ewes carrying multiple fetuses. The presence of ketonuria in cases of ovine pregnancy toxemia makes it relatively easy to distinguish from PEM.

DIAGNOSTIC AIDS

Careful evaluation of the animal's history and ration, as well as a physical examination, is usually sufficient to establish a tentative diagnosis of PEM. However, substantiating this diagnosis is both important and difficult. Few livestock owners readily accept a diagnosis of PEM; most believe that their animals are not being starved, but rather are suffering from some other condition. Producers usually suspect diagnoses such as an unusual infectious agent, a toxicosis, or an exotic nutritional deficiency. Because affected animals seem to deteriorate so rapidly, the diagnosis of a chronic condition such as PEM becomes especially difficult to accept. Efforts should always be made to rule out chronic diseases that could cause emaciation by themselves, or that could cause anorexia and secondary PEM. Exclusion of conditions that could cause secondary PEM lends important support to the diagnosis of primary PEM.

Clinicopathologic procedures are, unfortunately, of limited value when one is attempting to support a diagnosis of PEM in a live animal. Malnourished ruminants do have significantly lower packed cell volumes, insulin concentrations, and thyroid hormone values than well-fed animals.[13,14] However, changes in these blood values are usually clinically minor, within accepted normal limits, or attributable to other conditions that resemble PEM. Studies monitoring serum glucose, urea nitrogen, free fatty acid, and creatinine concentrations in malnourished ruminants have demonstrated that none of these tests has value in the definitive diagnosis of PEM.

The accuracy of blood tests intended to support a diagnosis of PEM can be increased by collection of samples from a representative group within a herd or flock. However, the value of such metabolic profile tests used to predict the nutritional status of apparently healthy ruminants is controversial. Application of these tests is discussed in depth in the article "Metabolic Profile Testing."

Most laboratory tests that could be expected to be of value in detecting PEM in ruminants are, in fact, not at all affected by malnutrition. For example, large body weight losses in cattle have been shown

to have no significant effects on blood concentrations of lactate dehydrogenase, aspartate aminotransferase, alkaline phosphatase, or creatinine phosphokinase.[21] Even when undernutrition is prolonged and severe, dairy cattle have been capable of maintaining most measures of blood composition within normal ranges.[23] Hyperketonemia rarely develops in ruminants when malnutrition is gradual and glucose requirements are moderate. Marked abnormalities in blood constituent concentrations probably indicate a failure of homeostasis rather than malnutrition itself.

Antemortem diagnostic tests can support, but not confirm, a diagnosis of PEM, and they cannot distinguish between primary and secondary PEM. Their greatest value is in ruling out other differential diagnoses, thus improving the likelihood that the diagnosis of primary PEM is correct.

PATHOLOGIC FINDINGS

Necropsy is required to confirm the diagnosis of primary PEM. Necropsy findings in ruminants that die from PEM include decreased muscle mass, lack of normal body fat, and serous (brown) atrophy of the fat that remains. Serous atrophy is caused by replacement of triglycerides in lipid vacuoles with proteinaceous fluid. It is most evident in the coronary grooves, around the kidneys, and in the bone marrow cavity. When PEM is primary and is attributable to inadequate forage quality, the rumens of affected animals are typically packed full of dry, firm ingesta. Ruminal contents will lack the pungent odor characteristic of normal fermentation and will often have an alkaline, rather than an acidic, pH. If PEM is secondary, or if it is caused by insufficient quantity of feed, then the rumen may be small or nearly empty. Special attention should be given necropsy findings that suggest a primary disease condition, which could cause secondary PEM.

TREATMENT

Once an animal affected by PEM has become recumbent, treatment is usually unrewarding. It is extremely difficult to reverse any advanced catabolic state in a ruminant animal. Owners of recumbent animals should be informed that recovery is unlikely and that therapy will be expensive and prolonged.

Therapy for PEM must be intensive and comprehensive to be successful. Intravenous fluid therapy may be necessary to correct secondary dehydration or electrolyte imbalances. The addition of glucose or amino acids to intravenous solutions provides much-needed energy and protein in a form that does not require a functional gastrointestinal tract; however, large and often cost-prohibitive quantities are required to be effective.

Ruminants affected with PEM usually have excellent appetites and should be offered high-quality legume hay free choice. Concentrate feeds, such as ground corn or soybean oil meal, should either be avoided or provided in small, frequent feedings. Sudden supplementation with such rapidly fermentable feeds usually results in ruminal acidosis and osmotic diarrhea in animals not accustomed to digesting such feeds. Indigestion may even lead to enterotoxemia in susceptible animals. Anorectic animals may benefit from force-feeding with a gruel, such as alfalfa pellets mixed with water, which is pumped into the rumen via a large-diameter tube. Oral electrolyte solutions may be added to the gruel, as necessary, to help correct dehydration. Propylene glycol, administered at a dose of 33 ml per 100 kg of body weight and given orally twice a day, can be useful in providing additional energy to the affected animal. Response to oral nutritional supplementation is often disappointing, however, because of impaired digestive function that hinders digestion and absorption of whatever nutrients are supplied. Ruminants generally recover from starvation more slowly than monogastric animals because of the prolonged time required to reestablish ruminal microbial populations. Ruminal transfaunation may hasten this process and ultimately speed the animal's return to normal ruminal digestion.

Other potentially useful therapeutic measures for individual animals with PEM include appropriate anthelmintic treatment if gastrointestinal parasites are present, and the use of insecticides if lice or other external parasites are present. Antiparasitic agents with a wide therapeutic index, such as the benzimidazoles, ivermectins, or pyrethrins, should be selected for use in cases of PEM. Organophosphate anthelmintics, such as haloxon, and organophosphate insecticides, such as coumaphos or trichlorfon (metrifonate), may be poorly tolerated by malnourished animals. Tetrahydropyrimidines (pyrantel and morantel) should also be used with caution in malnourished animals.

In certain cases of PEM, the life of the dam may be saved by cesarean section or by hormonal induction of parturition. Multiple B-vitamin injections may be given to help replace vitamins lost as a result of digestive disturbances or dehydration. Good nursing care becomes essential if recumbent animals are to survive. Frequent rolling from side to side, provision of soft bedding and secure footing, frequent massaging of muscles that the animal lies on, and meticulous attention to feeding and watering will all be necessary if the animal is to recover.

PREVENTION

When a case of PEM is diagnosed in a herd or a flock, the owner's attention should be directed toward the rest of the animals, rather than to the affected individual. Immediate supplemental feeding may be necessary to prevent further losses in the group. Supplemental feeding may prove to be especially difficult in range sheep, who are often reluctant to consume feeds to which they are unaccustomed. In these

situations, the sheep may require confinement to a small area or co-mingling with sheep accustomed to eating the supplement(s) before they will begin eating adequate quantities themselves.

High-quality legume hay is an excellent source of supplemental protein and energy for ruminants with PEM. Natural protein supplements, such as soybean or cottonseed meal, may also be useful. Increased dietary protein intake has the extra advantage of increasing digestibility and voluntary intake of forages. Nonprotein nitrogen sources, such as urea, are poorly utilized when fed in conjunction with low-energy roughages. Nonprotein nitrogen supplements should be avoided as a source of feed for animals recovering from PEM. Feeds high in energy and low in protein, such as ground corn or milo, will depress voluntary intake of forages and are usually not cost-effective as supplements when adequate forage supplies are available.

Long-term prevention of PEM requires both good management and good nutrition. Management factors that help prevent PEM should be encouraged and implemented. For example, provision of windbreaks and dry shelters will decrease the effects of cold weather on energy requirements. Adequate bunk or feeding space ensures that even submissive animals will have a chance to consume adequate rations. Primigravid animals should not be fed together with mature females; rather, they should be separated in the fall and supplemented according to their own requirements. Animals in thin condition at the end of the grazing season should be fed separately or with the primigravid females. Animals with bad teeth should be culled while they still have some salvage value.

Routine scoring of body condition or weighing of animals during the months of most severe nutritional stress can be used effectively to help prevent PEM.[22,24] Veterinarians are often able to assist livestock producers in using these scoring systems. Condition scores are usually assessed for beef cows at weaning time and before calving season begins. Ewes are most effectively scored before the mating season and before lambing. Body condition scoring at these strategic times allows livestock producers to make nutritional adjustments before production efficiency is decreased or deaths occur.

Laboratory analysis of the nutrient content of forages is the cornerstone of a good nutritional program, especially when harvested forages are used to maintain ruminants through the winter months. Reliance on visual appraisal or "book" values for forage composition is ineffective because of the many variables that affect forage quality. These variables include forage variety, fertilization rate, maturity at harvest, and weather. When the nutrient content of the forage is known, rations can then be balanced according to nutritional requirements established by the National Research Council (NRC). When conditions include cold weather without shelter, muddy lots, or sparse pasture, energy needs will be higher than NRC standards and appropriate adjustments should be made when formulating rations. Large, round bales or compressed hay stacks must be managed carefully to prevent underfeeding as a result of weather deterioration and wastage.

Adequate supplemental feed must be provided when dormant pastures or crop residues are grazed past midwinter or when animals are in their last trimester of pregnancy.

Adherence to proven management techniques and a scientific nutrition program are sound investments for the livestock producer. They not only prevent deaths resulting from PEM, they also pay additional dividends by increasing reproductive performance, disease resistance, and overall herd or flock productivity.

SUMMARY

Protein-energy malnutrition occurs when feed is provided to ruminant livestock in insufficient quantity, quality, or both. The clinical syndrome that results from protein-energy malnutrition is not difficult to recognize, but it may be difficult to convince owners of the diagnosis. Development of clinical signs, such as recumbency and hypothermia, may occur rapidly owing to the sudden failure of homeostatic mechanisms that maintain the supply of cellular fuels. The ruminant is unique in its response to malnutrition because ruminal microorganisms become malnourished just as their host does. Ruminal maldigestion hastens the onset of clinical signs and makes recovery very difficult and prolonged.

Clinical signs of PEM are similar in adult beef cattle, dairy cattle, sheep, and goats; however, the typical history of affected animals varies for each of these species. Neonatal ruminants may also be severely affected with PEM if they do not receive sufficient colostrum and milk. Definitive diagnosis of primary PEM requires necropsy of an affected animal.

Diagnosis of PEM in an individual animal usually indicates a herd or flock problem that requires immediate attention. If the affected individual is already recumbent, then treatment will likely be difficult and unsuccessful. Changes in management of the herd or flock that involve ensuring adequate feed intake, minimizing cold and social stress, and meeting the animal's specific nutritional requirements will prevent PEM and maximize production.

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