Rumen Acidosis, Heat Stress and Laminitis

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Introduction

Nutrition and feeding management are primary areas of interest when attempting to reduce lameness problems. This may or may not be the correct approach depending upon the specific types of lameness experienced. For example, it would be hard to influence the incidence of infectious foot diseases (foot rot, interdigital dermatitis, or digital dermatitis) by manipulation of the diet alone. Laminitis and claw disorders share a closer relationship to metabolic disease disorders which are often linked to nutrition and/or feeding issues. These are complicated by heat stress which has significant effects on rumen pH due to reduced salivary buffering. In addition, recent work by researchers from the United Kingdom suggest that peri-partum hormonal changes may be responsible for laminitis-like lesions observed during the early post-calving period. If these observations are accurate, we have yet one more explanation for how laminitis may occur despite proper nutrition, feeding, and heat stress management.

Rumen Acidosis

Acidosis is usually associated with the ingestion of large amounts of highly fermentable, carbohydrate-rich feeds which result in the excessive production and accumulation of lactic acid in the rumen. Grain overload, rumen overload, overeating disease and founder are all terms used to describe this condition. In its acute form, the disease is characterized by severe toxemia, ataxia, uncoordination, dehydration, ruminal stasis, weakness and recumbency. The critical pH threshold of the rumen in cases of acute acidosis is reported to be <5.0 (as cited Nocek, 1997). Mortality rate of the acute form of rumen acidosis is high.

The subclinical form of rumen acidosis (better known as SARA, for SubAcute Rumen Acidosis) is more common than the acute form of this disease. It is generally observed when recently calved cows are introduced to a diet considerably different than that fed during the dry period. Alternatively, it may occur when lactating cows are fed diets low in effective fiber. Major clinical manifestations would include variable feed intake, depressed fat test, poor body condition despite sufficient energy intake, mild to moderate diarrhea, and occasional cases of epistaxis (nose-bleed) or hemoptysis (the expectoration of blood from the mouth). Critical threshold of the rumen pH during bouts of subclinical acidosis is reported to be <5.5 (as cited Nocek, 1997). Conditions such as laminitis or undefined lameness, abomasal disorders, and liver abscesses are generally secondary observations (Nocek, 1997; Nordlund, 2002).

Although few studies have been able to establish a direct link between rumen acidosis and laminitis, most assume that the feeding program is a major underlying factor. In reality, much of the information ascribed to cattle is based on information from studies of starch overloading models in horses (Garner et al., 1975; Vermunt and Greenough, 1994). Recent work suggests

that an oligofructose overload model may be appropriate for the study of acute bovine laminitis. Researchers were able to successfully create classical symptoms of rumen acidosis and laminitis in animals treated with an alimentary oligofructose overload (Thoefner, et al., 2004). The following is an attempt to identify some of the more important predisposing factors relative to nutrition and feeding of dairy cattle.

Nutrition and Feeding Considerations
Rumen fermentation disorders that result in acidosis are typically traced to diets with excessive levels of highly fermentable carbohydrates and inadequate levels of effective fiber (Nocek, 1997). Even with high quality ingredients and proper formulation of the diet, what ends up in front of the cow is still at the mercy of those responsible for mixing, delivery, and management of the feed bunk. Add to these selective eating or feed sorting behavior of cows (Leonardi and Armentano, 2000) and it's easy to see that there's ample room for error. Equally important are dietary changes that naturally occur during the cow's lactation cycle. In recent years, nutritionists have concentrated their attention on transition cow feeding programs designed to ease the cow's adaptation to higher energy rations necessary to sustain milk production. A Florida study concluded that large differences in the fiber and net energy content of close-up and early lactation diets can contribute to an increase in the incidence of rumen acidosis and subclinical laminitis (Donovan et al., 2004).

Carbohydrate
Feeding rations high in non-structural carbohydrates to animals that are not sufficiently adapted have the potential to result in rumen acidosis. Research shows that volatile fatty acid production increases as pH begins to decrease when highly fermentable carbohydrates are fed. In conditions where there is an ample supply of highly fermentable carbohydrate, rumen pH continues to decline resulting in a change in the rumen microflora from predominantly gram-negative to gram-positive lactic acid-producing bacteria. Eventually, the bacteria that utilize lactic acid are outnumbered by those that produce it which leads to the accumulation of lactic acid in the rumen. Coincident with the change in rumen pH and microflora is the release of endotoxin from the outer cell walls of dying and disintegrating gram-negative bacteria. Aided by a damaged and dysfunctional rumen mucosa, bacteria *(Fusobacterium necrophorum)*, lactic acid, endotoxin and possibly histamine are absorbed into the blood stream. These products are rapidly dispersed to the micro-circulation of the corium, where directly or indirectly (through vaso-active mediators), blood flow is disrupted leading to the lesions observed in laminitis (Nocek, 1997; Vermunt and Greenough, 1994).

While there is little dispute that rumen acidosis may occur as described above, it is not clear that laminitis will inevitably occur as a consequence. Three studies observed no correlation between laminitis and the feeding of rations high in carbohydrate (Smit, 1986; Frankena et al.,1992; Momcilovic, et al., 2000). Despite conflicting information in the literature, one would still have to conclude that there seems to be an association (albeit complex) between carbohydrate nutrition, rumen acidosis and laminitis, but more research is needed to sort out the details of these relationships.

Protein
Feeding high levels of protein in the diet of dairy cows and the potential to cause laminitis or lameness is surely less well understood. Outbreaks of laminitis in calves fed milk replacer and

starter rations containing 18% digestible protein are reported from Israel (Bargai et al., 1992). Calves affected were 4 to 6 months of age and had lesions in their claws consistent with severe acute laminitis. Although this is an interesting observation, some might view the suggestion that high protein was the cause of this problem with significant skepticism, since milk replacers and rations containing 18% protein (or higher) are commonly fed to calves and young stock throughout North America without incident. Results of a Canadian study found no relationship between the level of protein fed and lesions associated with laminitis (Greenough, et al., 1990; Greenough, 1990). It may be that laminitis associated with protein feeding may be related to allergic histaminic reactions to certain types of proteins, or a link between protein supplementation and protein degradation end products (as cited Nocek, 1997). Regardless, at this time one would have to conclude that there is simply insufficient information to know what effects, if any, protein may have on foot health.

Heat Stress and Rumen Acidosis
The primary avenues for heat loss during periods of hot weather are sweating and panting. In severe heat, panting progresses to open-mouth breathing characterized by a lower respiratory rate and greater tidal volume. The result is respiratory alkalosis as a result of the increased loss of carbon dioxide. The cow compensates by increasing urinary output of bicarbonate (HCO_3). Simultaneously, the salivary HCO_3 pool for rumen buffering is decreased by the loss of saliva from drooling in severely stressed cows. The end result is rumen acidosis because of reduced rumen buffering and an overall reduction in total buffering capacity (Dale et al., 1954).

Rumen pH is largely determined by the balance between the acids generated from the fermentation of feedstuffs and the bicarbonate and phosphate buffers in saliva which neutralize these acids. Physically effective fiber stimulates chewing and chewing stimulates saliva secretion. Consequently, consistent intake of feedstuffs with effective fiber and cud-chewing are essential for rumen buffering. Saliva flow rates in beef and dairy cattle are estimated to be in the range of 108 to 308 liters (28 to 81 gallons) per day (Erdman, 1988). At these rates of saliva flow it is estimated that the cow can contribute in the range of 390 to 1115 grams (.86 to 2.5 lb) of disodium phosphate and 1134 to 3234 grams (2.5 to 7.1 lb) of sodium bicarbonate for rumen buffering daily. Reduced feed intake, a preference for concentrates rather than forage, a loss of salivary buffering from increased respiratory rates and drooling, and a reduction in the total buffering pool all contribute to a greater potential for rumen acidosis during periods of hot and humid weather, and may explain in part, why some herds experience more acidosis and lameness despite being fed properly formulated rations.

The effect of ambient air temperature on rumen pH was evaluated in lactating Holstein cows fed either a high roughage or high concentrate diet in both a cool (65^oF with 50% relative humidity) and a hot (85^oF with 85% relative humidity) environment. Rumen pH was lower in cows exposed to the higher temperatures and those fed the higher concentrate diets (Mishra, et al., 1970). These observations have been corroborated by others (Bandaranayaka and Holmes, 1976; Niles, et al., 1998) supporting the current view that increasing the energy density of rations to compensate for reduced dry matter intake during periods of hot weather is not without significant risk.

<u>**Laminitis and Claw Disorders in Cattle**</u>

Laminitis is an important predisposing cause of claw disorders in cattle. Inflammation of the corium results in the activation of tissue matrix metallo-proteinases (MMPs) that weaken the collagen fiber bundles that make up the suspensory apparatus within the claw. Coincident with this is the release of horn growth and necrosis factors that contribute to the inflammatory process and accelerate claw horn growth (Mulling and Lischer, 2002). The vascular disturbances associated with laminitis preclude the normal diffusion of nutrients and oxygen into the living-cell layers of the epidermis destined to become claw horn. This interrupts the normal differentiation of these cells and leads to the formation of weaker or softer claw horn. Some have suggested that "*claw horn disruption*" may be a better term for the condition of laminitis, because it more accurately reflects the nature of the anatomical and physiological lesions associated with laminitis (Logue et al., 1998). The following is a brief synopsis of the tissues and events that occur in the pathogenesis of this disease.

Anatomical/Histological Perspectives

The corium (or dermis) within the claw capsule consists of connective tissue with a rich supply of blood vessels and nerves. Immediately above the corium is the basement membrane and epithelial cell layers that comprise the claw horn: germinal epithelium, stratum spinosum, stratum lucidum, and finally, the stratum corneum or horn layer. Although it has no direct blood supply, cells within the lower layers of the epithelium (germinal epithelium and lower layers of the stratum spinosum) are "living cells" by virtue of nutrients and oxygen received from the corium by diffusion across the basement membrane. Cell death and cornification (formation cells into horn) occur as cells extend beyond the point to which they no longer receive nutrient and oxygen supply from the corium. Clearly, any condition resulting in a disruption in the flow of blood to the corium not only affects the corium, but also the epithelium and thus, the integrity of claw horn.

Pathogenesis of Laminitis - The Classical Description

The pathogenesis of laminitis is believed to be associated with a disturbance in the micro-circulation of blood within the corium which leads to breakdown of the dermal-epidermal junction between the claw wall, and the bone (otherwise known as the third phalanx (P_3) within the claw. Rumen acidosis is considered to be a major predisposing cause of laminitis and presumably mediates its destructive effects through various vasoactive substances (endotoxins, lactate, and possibly histamine) that are released into the blood stream in coincidence with the development of rumen acidosis. These vasoactive substances initiate a cascade of events in the vasculature of the corium including a decrease in blood flow caused by veno-constriction, thrombosis, ischemia, hypoxia, and arterio-venous shunting. The end result is edema, hemorrhage, and necrosis of corium tissues leading to functional disturbances including the activation of MMPs that degrade the collagen fiber bundles of the suspensory apparatus of the P_3. This is complicated still further by the activation of horn growth and necrosis factors that result in structural alterations involving the basement membrane and capillary walls.

Changes occurring in the epidermis are secondary to vascular disturbances that result in reduced diffusion of nutrients from the dermis to the living layers of the epidermis. This interrupts the differentiation and proliferation of horn cells (also known as keratinocytes) in the germinal layer, and the keratinization of horn cells in the stratum spinosum. The quality of claw horn is

dependent upon keratinization which gives the horn cell structural rigidity and strength. In conditions resulting in vascular compromise such as laminitis, the keratinocyte may become injured or inflamed from being deprived of oxygen and nutrients. The end result is the production of poorly keratinized (weak or inferior) horn that weakens the claw horn capsule's resistance to mechanical, chemical, and possibly even microbial invasion (for example, an increased susceptibility to heel erosion).

Inflammation leading to destruction of the dermal-epidermal junction also has particular consequences for cattle in that weakening of the suspensory apparatus within the claw permits downward displacement and rotation of the third phalanx (P_3). The result is compression of the corium and supporting tissues that lie between P_3 and the sole which predisposes to the development of sole ulcers (Lischer et al., 2002). In some cases this "P_3 sinking phenomenon" involves severe rotation of the toe portion of P_3 downward toward the sole. If compression of the corium by the toe is severe enough, a toe ulcer may develop. If, on the other hand, sinking of the P_3 is such that the rear portion sinks furthest, compression and thus sole and/or heel ulcer development will most likely occur. Sole ulcers are common claw lesions in dairy cattle and constitute one of the most costly of lameness conditions.

Peripartum Hormonal Effects
Recent work by researchers from the United Kingdom suggests another mechanism for weakening of the dermal-epidermal segment between the wall and P_3. Their work suggests that weakening of the suspensory tissue may be the result of hormonal changes that normally occur around the time of calving. Hormones, responsible for relaxation (relaxin) of the pelvic musculature, tendons, and ligaments around the time of calving, are thought to have a similar effect on the suspensory tissue of P_3 as well. These workers further suggest that although this weakening of the suspensory apparatus may be a natural occurrence, housing of animals on soft surfaces during the transition period (4 weeks prior to calving through 8 weeks after calving), may be sufficient to reduce or alleviate the potential for permanent damage to these tissues. Researchers observed fewer claw lesions in heifers housed in straw yards during the transition period as compared with heifers housed in cubicles (free stalls). Clearly, cow comfort issues matter around the time of calving. It would appear that first lactation animals in particular would benefit from softer flooring surfaces (Tarleton and Webster, 2002; Webster, 2002).

German researchers suggest that sinking and rotation of P_3 may be associated with as of yet unexplained structural alterations occurring on the surface of P_3 where the suspensory tissues are anchored. It is clear that despite the preponderance of information linking laminitis to feeding conditions that predispose to rumen acidosis, softer flooring surfaces and cow comfort cannot be overlooked as requirements for animals during the transition period (Mulling and Lischer, 2002).

Conclusions
Laminitis predisposes cows to claw disorders that are the major causes of lameness in dairy cattle. It is predisposed by rumen acidosis, heat stress and peripartum hormonal changes. Damage to the dermal-epidermal junction interferes with the diffusion of nutrients across the basement membrane into the living layers of the epidermis. Furthermore, disruption of the basement membrane and germinal epithelium restricts normal differentiation and proliferation of keratinocytes destined to become claw horn. The end result is weaker less resistant claw horn.

Current information suggests that laminitis is a disease affecting tissues at the cellular level. "Claw horn disruption" is the phrase preferred by some who believe that this more accurately describes the lesion of laminitis. Reduced keratinization is a major complication of laminitis and results in the production of soft weak horn that is less resistant to physical or mechanical forces. Sinking and rotation of P_3 is a secondary consequence of the damage caused by matrix metalloproteinase enzymes released during the course of the disease. These enzymes are responsible for degradation of the collagen fiber bundles in the suspensory apparatus of P_3 which creates laxity in this support system and permits sinking and rotation of P_3. A second mechanism is believed to be associated with the hormonal changes that occur around the time of calving. United Kingdom researchers have proposed that the same hormones responsible for relaxation of the pelvic musculature (relaxin) near the time of calving have a similar effect on the suspensory apparatus of P_3. These same researchers also found that housing animals on soft surfaces throughout the transition period permitted recovery of suspensory apparatus tissues, thus preventing permanent damage that may have predisposed to lameness in early lactation. In problem herds it is necessary to review the nutrition and feeding program, herd management, and cow comfort.

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