Lameness in Cattle: Causes and Consequences

Jan K. Shearer
College of Veterinary Medicine, University of Florida

Introduction

Lameness is one of the most important health problems in dairy cattle. Involvement by veterinarians varies from diagnosis and treatment of lameness conditions to consultation with dairy owners and managers on procedures for treatment, control and prevention. In large herds, the sheer number of lame cows is beyond what most veterinarians can cope with on a daily basis. Therefore, throughout much of North America foot care and claw trimming tasks are performed by outside trimmers or farm employees. Regardless of who performs these tasks, the key to achieving a successful outcome from treatment is prompt examination and therapy of lameness conditions. In addition to reducing losses to lameness, early recognition and treatment accomplishes the objectives of limiting animal suffering and preventing lameness disorders from continued progress toward more serious forms of disease that may require surgical correction and an extended period of recovery.

Over 90% of lameness in dairy cattle involves the foot, and of that, more than 90% involves the rear feet, with the majority of disorders affecting the outside claw. The most common causes of lameness are ulcers and white line disease. This indicates that more than just nutrition and feeding management errors are responsible for lameness disorders. Logically, if all lameness were simply a consequence of rumen acidosis and laminitis, then risk and incidence of disease should be similar for all claws of each foot. Indeed, the unique pattern of lameness in cattle suggests that it is more complex. Beyond ration adjustments and correction of feeding management errors, short and long term improvement of foot health generally requires attention to housing and cow comfort, and other management factors.

The infectious claw diseases: digital dermatitis (DD) and interdigital dermatitis (ID) are common maladies in today's modern dairy operations. Treatment and control options exist, but none are completely satisfactory or effective, and recurrence rates are generally high (around 50% or more). Foot rot is generally less common and often confused with other forms of deep digital sepsis. This confusion sometimes leads to treatment delays allowing the infection to ascend (or extend) into adjacent tissues of the foot including the distal interphalangeal joint and retro-articular space. This usually reduces options for treatment to surgery, culling, or in the worst case scenario, euthanasia.

The following is a brief review of some of these conditions with specific comments on how to manage them.

Laminitis and Claw Disorders in Cattle

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 horn capsule. Coincident with inflammation of the corium is the release of horn growth and necrosis factors that contribute to the inflammatory process and

accelerate claw horn growth. 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 normal differentiation of these cells and leads to the formation of weaker or softer claw horn. This has caused some to suggest 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 this condition. 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. It is otherwise known as the quick or the sensitive tissue layer. Adjacent to the corium (moving toward the exterior) is the basement membrane and a series of epithelial cell layers that comprise the claw horn: germinal epithelium, stratum spinosum, stratum lucidum, and finally, the stratum corneum or horn layer. Cells within the germinal epithelium are specialized cells known as keratinocytes; that is, they have the ability to produce and accumulate keratin within their interior. Keratin is a fibrous scleroprotein that imparts strength to the keratinocyte giving it resistance to physical and mechanical forces. Between each of these cells is intercellular cementing substance, a lipoprotein which binds the cells together, like the mortar between bricks in a wall. Although they have no direct blood supply, cells within the 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. Since the natural progression and movement of keratinocytes is outward from the corium and away from their nutrient source, keratinocytes begin a process of slow death within the upper layers of the stratum spinosum and stratum lucidum. Horn is formed in the stratum corneum by the death and cornification of these keratinocytes. 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 matrix metalloproteinases (MMP) 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.

As described above, the 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 in the germinal layer, and the keratinization of horn cells in the stratum spinosum. Vascular compromise (such as that which occurs with laminitis) leads to injury of the keratinocyte from the deprivation 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. 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 suggest 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.

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.

White Line Disease

Areas of hemorrhage are often most noticeable and severe in the white line region of the sole. This corresponds to the primary weight-bearing region of the claw. Because it is an active area of hoof formation it is also highly vascular, and a frequent site for hemorrhage during bouts of laminitis. These areas of hemorrhage are not visible during the acute stage of laminitis, but instead, gradually rise to the surface of the sole as it grows outward over a period of 6-8 weeks. At this point they become visible and useful as indicators of previous disease of the corium (subclinical laminitis).

Another possible outcome associated with laminar necrosis and separation of the white line is the formation of subsolar abscesses (otherwise known as white line disease). There are a couple of reasons why cows that have had laminitis are at greater risk for white line diseases. For one thing, horn of the white line is naturally softer, but especially so when formed by a diseased corium. The softer it is the more vulnerable it is to damage or separation and penetration by foreign material from the environment. Secondly, weakening or elongation of the dermal-epidermal segment that leads to distorted claw horn growth often results in widening of the white line. Since the white line contains the softest horn within the weight bearing surface the wider it is, the more likely it is to be breached by foreign material from the environment.

Infectious Diseases of the Skin of the Foot

Infectious claw disorders represent some of the most important causes of lameness in dairy cattle. However, unlike the lesion associated with a sole ulcer or white line disease which specifically affects the claw, these diseases affect the "skin" of the interdigital space, heel bulbs, and interdigital cleft (on the back of the foot above the interdigital space). Although there are some differences in the way these conditions develop and the way they appear, they all have at least one thing in common: they are believed to be caused by infectious agents capable of inducing inflamation and lameness.

Digital Dermatitis (Hairy Heel Warts or Footwarts). This disease has occurred in near epidemic proportions throughout the United States since the early 1990s. Although not conclusively proven, it is the opinion of most that

digital dermatitis (DD) are caused by 3, or possibly 5, different bacterial spirochetes belonging to the genus *Treponema sp.* Although lameness caused by DD may be severe, it tends to be an inconsistent feature of this disease. Florida studies indicate that only about half of the animals affected exhibit lameness. Cows naturally avoid contact with the ground or flooring surface by walking on their toes. In fact, stubbed toes from excessive hoof wear is one indicator of a DD lesion in affected cows. Despite obvious indications of discomfort, research indicates that cows affected with DD are likely to suffer reduced reproductive performance and a tendency for lower milk production.

Lesions associated with this disease are typically round or oval and located on the back of the foot adjacent to the interdigital cleft. Some lesions are located on or above the heel bulbs and still others may be found adjacent to or near the dew claws. Early lesions are red, raw, and flat. They are extremely sensitive and cows react painfully to spraying with water or other direct contact. Even a mild disturbance of the inflamed tissue tends to result in mild to moderate bleeding. As lesions mature most will enlarge. Hairs at the skin margins remain long and erect serving as distant evidence of what might otherwise be an obscure lesion. On closer inspection one will observe a lesion with characteristics similar to the early lesion with a slightly more raised surface, characterized by some as granular or terrycloth-towel-like. These more mature lesions may be red, tan, or grey. Similar to the early lesion, they are very sensitive and tend to bleed easily if sufficiently disturbed.

The natural way for skin or hoof horn to respond to chronic irritation is to increase its rate of growth. Consequently, the chronic lesions of DD are characterized by a thickening of the skin accompanied by the development of filamentous or conical (hair-like) projections of whitish, grey, or even brownish keratin (the tough protein substance of skin). These filamentous extrusions of skin are responsible for the characteristic hair-like appearance of some lesions and thus, the term "hairy heel warts". Since hoof horn is a modified version of skin, lesions that develop near the site of hoof horn formation (near the coronary band) often have the effect of accelerating hoof formation and thus lead to overgrowth. Coincident with this is the fact that many of the bacteria believed to be associated with this condition are capable of producing enzymes which can eat away hoof horn. Therefore, it is not uncommon to find evidence of both overgrowth and erosion in the same foot.

Treatment of DD (Hairy Heel Warts Footwarts). Approaches to therapy include:

1. Surgical excision (removal)
2. Footbaths
3. Topical treatment with various disinfectants, caustic chemicals, and antibiotics
4. Cryosurgery (freezing), and electrocautery (burning)
5. Topical treatment under a bandage
6. Systemic antibiotic therapy.

Most of these treatments have a place in the management of this condition, however the less invasive forms of treatment are preferred by these authors and highlighted in the following.

Topical spray-on treatment with antibiotic and some non-antibiotic preparations has been shown to be very effective when used in a scheme of consistent daily treatment for a period of 810 days over 2-week period. The major disadvantage to topical treatment is that lesions occurring in the interdigital space are missed. Topical treatment under a bandage is particularly effective with most cows showing remarkable improvement within 24-48 hours. Furthermore, when properly applied this approach to treatment has the advantage of reaching interdigital lesions. Theoretically at least, the same is true for well-managed footbaths.

Foot rot and Super Foot rot. Footrot is an infectious disease of the interdigital skin characterized by the presence of an interdigital lesion, swelling, and moderate to severe lameness. Fever ranging from l03-105°F (occasionally higher) is a consistent finding during the acute stages. Although evidence is inconclusive, most believe that footrot develops following injury or abrasion of the interdigital skin. This interdigital injury is secondarily infected by *Fusobacterium necrophorum* alone, or in combination with *Bacteriodes melaninogenicus,* organisms which encourage progression to a more severe and necrotic-type of lesion. Failure to institute treatment early in the course of the disease may lead to complications involving surrounding soft tissues (tendons, tendon sheaths, joint capsules, and bone) ultimately resulting in deep digital sepsis. At this

stage, response to medical therapy is quite often unrewarding, thus limiting one's options to either surgery, or possibly euthanasia, in particularly severe cases.

In recent years, clinicians from the United Kingdom and the United States have observed a more extreme form of this disease referred to as "super foot rot". It is characterized by acute onset of lameness and swelling of the foot that progresses rapidly to an ascending cellulitis (a spreading infection). The interdigital lesion associated with this form of foot rot tends to be especially severe and successful treatment particularly challenging. Readers are advised to contact their veterinarian for specific advice on treatment options if they suspect this form of the disease.

Treatment of Foot rot. Foot rot is responsive to most antibiotics in common use for cattle. In fact, dose and duration of treatment are likely more important than antibiotic selection. The key to achievement of a successful therapeutic outcome is dependent upon prompt recognition and early implementation of treatment procedures. Systemic therapy plus topical treatment of the interdigital lesion have long been the preferred methods of treatment. In uncomplicated cases, improvement is noticeable within 24-48 hours with good recovery attainable in 3-4 days from the onset of treatment. Treatments of choice are Naxcel (Ceftiofur Sodium), Penicillin, Albon (Sulfadimethoxine), and tetracyclines (extra-label in dairy cattle). Some prefer to simultaneously treat the interdigital lesion as well. Various antiseptic-type products may be used as topical treatments. Bandaging of the foot is unnecessary. Regardless, the secret to success is early detection of the disease.

In Conclusion

Laminitis predisposes 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 nutrition and feeding, herd management, and cow comfort. Infectious claw diseases are also important causes of lameness in dairy cattle. Foot rot and "super foot rot" represent conditions that cause severe inflamation and swelling of the foot. Closer examination will reveal a lesion in the interdigital skin as well. Prompt identification and antibiotic treatment are crucial to achievement of a successful therapeutic outcome with footrot. Heel erosion, accelerated skin and hoof horn formation, lesions in the interdigital space, and the presence of bacterial spirochetes are consistent findings with DD. Although lameness is inconsistent, the lesions are painful and have a negative impact on overall performance. These diseases are most effectively treated and controlled by applying topical treatments either under a bandage, as a topical spray, and/or in a well-managed footbath.

References

Argaez-Rodriguez, R.J.D., W. Hird, J. Hernandez, D. H. Read, and A. Rodriguez-Lainz. 1997. Papillomatous digital dermatitis on a commercial dairy farm in Mexicali, Mexico: incidence and effect on reproduction and milk production. Prev. Vet. Med. 32:275-286.

Choi, B. K., H. Nattermann, S. Grund, W. Haider, and U. B. Gobel. 1997. Spirochetes from digital dermatitis lesions in cattle are closely related to treponemes associated with human periodontitis. Int. J. Sys. Bact. 47:175-181.

Greenough, P. R. 1990. Observations on bovine laminitis. In Practice, 12:169-173.

Hernandez, J., J. K. Shearer, and J. B. Elliott. 1999. Comparison of topical application of oxytetracycline and four non-antibiotic solutions for treatment of papillomatous digital dermatitis in dairy cows. J. Am. Vet. Med. Assoc. 214:688-690.

Hoblet, KH, and Weiss, W. 2001. The Veterinary Clinics of North America, Food Animal Practice, Volume 17, Number 1, p. 111-127.

Logue, D. N., S. A. Kempson, K. A. Leach, J. E. Offer, and R. E. McGovern. 1998. Pathology of the white line. Proceedings of the 10th International Symposium on Lameness in Ruminants. Lucerne, Switzerland, p.142-145.

Mulling, C. K. W., and C. J. Lischer. 2002. New aspects on etiology and pathogenesis of laminitis in cattle. Proceedings of the XXII World Buiatrics Congress (keynote lectures), Hanover, Germany, p.236-247.

Nocek, J. E. 1997. Bovine acidosis: Implications on laminitis. J Dairy Sci., 80(5):1005-1028.

Lischer, C. J., P. Ossent, M. Raber, and H. Geyer. 2002. The suspensory structures and supporting tissues of the bovine 3rd phalanx and their relevance in the development of sole ulcers at the typical site. Vet Record, 151(23):694-698.

Shearer, J. K., and J. B. Elliott. 1998. Papillomatous digital dermatitis: treatment and control strategies - Part I. Compend. Cont. Educ. Pract. Vet. 20:S158-S166.

Shearer, J. K., J. Hernandez, and J. B. Elliott. 1998. Papillomatous digital dermatitis: treatment and control strategies - Part II. Compend. Cont. Educ. Pract. Vet. 20:S213-S223.

Tarleton, J. F., and A. J. F. Webster. 2002. A biochemical and biomechanical basis for the pathogenesis of claw horn lesions. Proceedings of the 12th International Symposium on Lameness in Ruminants, Orlando, Fl, p. 395-398.

Vermunt J. J., and P. R. Greenough. 1994. Predisposing factors of laminitis in cattle (Review). Br Vet J, 150(2)151-164.

Webster, J., 2002. Effect of environment and management on the development of claw and leg diseases. Proceedings of the XXII World Buiatrics Congress (keynote lectures), Hanover, Germany, p. 248-256.