Laminitis in Dairy Cattle: Recognition of the Disorder and Management of the Causative Factors

Charles Guard, *DVM, PhD*
Ambulatory and Production Medicine Clinic
New York State College of Veterinary Medicine
Cornell University
Ithaca, New York 14853

Lameness in cattle is a common condition that can result in significant economic loss to a producer through a variety of mechanisms. Lame cows do not eat as much as healthy cows and thus produce less milk or less gain. They may become anestrous. Furthermore, they may be prematurely culled due to low milk production, delayed conception or emergency slaughter.

In a comparison with the other most common clinical conditions of dairy cattle, lameness was estimated to be the most costly on a herd basis.[1] Estimates were derived from published reports and data from the records of the Ambulatory and Production Medicine Clinic at Cornell University. With an average incidence of 30 cases per 100 cows per year, a case fatality rate of 2%, involuntary culling of 20% of cases; average increased days open of 28, treatment costs including veterinary fees, drugs and farmer labor of $23 per case, the total cost per 100 cows per year is estimated to be about $9000.

Laminitis is typically observed as chronic malformation of the claws with grooves and ridges, twisted axes, or flared claws. Acute lameness due to laminitis is most often due to white line abscesses or sole ulcers. Cows with peracute laminitis are rarer; they have no changes evident in the horn of the digit but are reluctant to rise or walk. Subacute laminitis is recognized by solar hemorrhages, yellow waxy solar horn, and broadened or bloody white lines.

Ruminal acidosis is the most common initiating cause of laminitis. Occasionally, acute febrile or toxic illness leads to laminitis. These cases are very rare compared to those following ruminal acidosis. The precise steps between ruminal acidosis and laminar vascular and inflammatory changes are not known. Environmental factors influence the final expression of lameness from laminitis given equivalent degrees of ruminal acidosis in different herd scenarios.

Pathophysiology of laminitis

Histopathological observation of experimentally produced laminitis showed a sequence of changes in the laminae.[2] Endothelial cell swelling and edema of the dermis occurred first, but several hours after lameness was apparent. Microvascular thrombosis and necrosis in the stratum spinosum were noted within a few days. Distortion of the laminae, epithelial hyperplasia, and hemorrhage developed within 10 days. These destructive changes disrupt the mechanical connection between the laminae of the hoof wall and the third phalanx. Some of the space created as P3 moves away from the hoof wall is filled with blood from torn microvessels. The hoof wall is thinner and more flexible near the heel which may account for this being the most common site for subsequent white line abscesses. Capillary permeability measured in the vascularly isolated digit of horses did not change during the development of laminitis.[3] However, several studies have shown that total vascular resistance of the digit increases early in the development of laminitis, mostly due to postcapillary venous pressure.[4] Despite the degree of increased resistance total blood flow may actually increase.[5,6] The increased flow is probably due to dilation of arteriovenous anastomoses in the coronary band. Catecholamines and some eicosanoids have similar effects on cutaneous circulation. In horses, endotoxin is considered by some to be important in the vascular changes of laminitis, however parenteral injection of endotoxin has not produced laminitis in horses.[7] The systemic or local injection of histamine leads to similar vascular changes. Local histamine injection combined with a rumen starch load or exposure to severely cold environmental temperatures produced more severe lameness than histamine alone.[8] Dutch researchers failed to induce changes typical of laminitis with digital arterial infusions of endotoxin.[9] Additional hormonal mediators are probably involved in the vascular changes in the hoof. The appearance of leukocytes in the dermis during laminitis is delayed for days or weeks depending on the degree of necrosis. Thus, the vascular changes are the primary derangement leading to laminitis and ordinary inflammation is a secondary event.

Clinical presentations of laminitis

Acute laminitis is evident by reluctance to rise or to walk. There is swelling and redness around the coronary band of all claws. There may be no detectable changes in the hoof if it is trimmed. Hoof testers applied to any digit usually demonstrate severe pain. Some herdsmen will misdiagnose this as foot rot affecting multiple digits and treat accordingly. The lameness subsides after a few days supporting the belief of the herdsman. A minority of these cows will develop severe laminar or solar separation with hemorrhage about a week later and again become severely lame. The lateral claws of the rear feet are most commonly affected. The hoof wall and sole are completely detached from the dermis at the heel, often with liquefactive necrosis of parts of the dermis.

The clinically apparent effects on the structure of the hoof of an episode of laminitis include: 1) solar hemorrhage with no detectable lameness, 2) claw growth abnormalities resulting in twisted claws or flared walls, 3) white line hemorrhage or widening, 4) white line abscess, 5) sole ulceration, 6) false sole, and 7) thimbling of the hoof wall. Despite the systemic nature of the causative factors of laminitis the lateral claws of the rear feet develop the majority of the lesions producing lameness.[9,10,11,12] Cows with chronic laminitis often stand with sickled hocks and both rear feet rotated outward. This abnormal posture helps reduce pressure on the painful lateral claws. When lesions occur in the fore feet they typically involve the medial claws. Cows may stand with their forelimbs crossed to relieve pressure on the painful claws.

The progression from the initial vascular changes to the lesions recognized by the veterinarian as chronic laminitis probably are the result of continued or multiple vascular insults rather than a single episode as in acute laminitis. Once the mechanical linkage between the hoof wall and P3 has been weakened, the hoof wall may pull away under forces exerted during normal walking and turning. This can result in hemorrhage which later undergoes sterile decomposition. The breakdown of blood proteins and cells results in a large increase in osmotic pressure which leads to fluid movement from the sensitive laminae into the abscess. The fluid pressure produces severe pain in the surrounding dermis. Resolution may be spontaneous by rupture to the outside at the white line or at the coronary band. Trimming away the hoof until the abscess drains has long been recognized as curative.

Sole ulceration is the result of necrosis of the dermis and subsequent failure to produce cornified epithelium. The location of sole ulcers invariably overlies the flexor process of P3. Edema surrounding P3 within the confined space of the hoof wall and sole forces P3 down and back. The dermis is thinnest between the flexor process and the sole in normal claws. Even slight relative movement of P3 will lead to ischemia and pressure necrosis of the dermis adjacent to the flexor process. As the remainder of the sole grows out, the site of the pressure from the flexor process may be hemorrhagic or ulcerated. Just as the white line may grossly thicken with poor quality horn as the wall separates from the sensitive laminae, there is often excessive horn production deforming the sole with growth into the axial space around a sole ulcer.

False sole and thimbling are the result of similar processes affecting the adhesion of horn tubules produced before and after an acute episode of laminitis. False soles may be discovered during routine trimming with no associated lameness. An apparently healthy layer of solar horn may underlie the completely separated older layer. Thimbling of the hoof wall is usually diagnosed in a lame cow when the distal, separating cone of hoof wall is only attached at the tip of the toe, usually the terminal 2 to 3 cm. A line of separation in the hoof wall, parallel to the coronary band, is usually about 6 cm from the coronary band when lameness occurs. The separated wall portion moves relative to the newer underlying hoof wall when the cow walks. The sensitive laminae of the toe tip are pinched during this movement.

Complications of sole ulcer or occasionally of white line abscess may produce severe lameness. Ascending infection of the deep flexor tendon, navicular bursa, or coffin joint require aggressive therapy or salvage. Claw amputation is usually indicated for septic arthritis. If the infection has ascended above the dewclaws in the flexor tendons, salvage is recommended, since the branches of the flexor tendons join at this level. As an alternative to claw amputation, I have been fenestrating the coffin joint with a 3/4 inch drill. After intravenous regional anesthesia, the drill is directed through the sole at the site of the ulcer, through the distal portion of the deep flexor tendon, through the navicular bursa, through the coffin joint, and out just above the coronary band on the dorsum of the digit. A nylon rope is passed through the hole and tied above the heel to promote continued drainage. A wooden block is attached to the sound claw. Systemic antibiotics are administered for 10 days and the rope is cut and removed at about 14 days.

Management effects on the incidence of laminitis

Ruminal acidosis

The inciting cause for the changes in the circulation in the claws is a low pH in the rumen. If the rumen pH gets low enough (normal is 5.9 to 6.5 for lactating cows), such as below 5.5 some chemical signal is released from the rumen that alters blood circulation. How does the rumen pH get below 5.5? The normal acids pro-

duced by fermentation in the rumen are acetic, butyric, and propionic. These acids are readily absorbed from the rumen and thus removed from the system. Absorption occurs in the undissociated form; the 3 volatile fatty acids have pK's of 4.8. Lactic acid with a pK of 3.1 is absorbed at about 10% the rate of the VFA's. Cattle fed all forage diets that consist mainly of cellulose make mostly acetic acid. If starch or sugar is added to the diet, certain bacteria ferment them to either propionic or lactic acid depending on how adapted the bugs are and the amount of rapidly fermentable carbohydrate that is consumed in a meal. At a constant level of starch feeding, adaptation occurs over 2 to 4 weeks that generally directs most fermentation products of starch and sugar to propionic acid.

Lactic acid is bad for the cow since it is very slowly absorbed from the rumen. Once absorbed the D and L isomers have very different rates of metabolism.[14] L-lactate is readily converted to pyruvate by lactate dehydrogenase and thus may be converted to glucose. D-lactate is more slowly metabolized by a mitochondrial enzyme to pyruvate. Excess D-lactate is excreted in urine. Lactic acid that is not absorbed must be buffered to maintain rumen pH in a healthy range or metabolized to propionate. The lactilytic bacteria *Megasphaera elsdenii* is the main converter of D-lactate to propionate. Even after 4 weeks on a high concentrate diet there is still significant lactate present in rumen fluid.[15]

Cud chewing is a great source of extra buffer for the rumen, but it is stimulated by coarse fiber which diets that induce lactic acid production generally lack. Of practical importance in formulating diets or solving problems of ruminal acidosis is that starches differ in the rate they ferment. Corn starch, if from dry corn, is relatively slow to ferment since it must be hydrated from a crystalline state before the microbes can attack it. High moisture corn starch ferments quite rapidly as do barley and wheat starch. The particle size of the grain as it is consumed will also influence the rate of lactic acid production. Coarse grinding will result in slower fermentation but may lead to reduced total digestibility. Steam flaking of grain also increases the rate of fermentation. In addition to stimulating cud chewing, large forage particles form a floating mat at the top of the rumen. As new feed is consumed it is dumped on top of this mat. The mat traps grain particles in a microenvironment that only slowly ferments starch. During regular rumen contractions the fully hydrated, dense particles drop slowly to the bottom of the rumen where most of the starch digestion takes place.

The absolute amount of starch consumed per day or per meal has been shown to influence the development of laminitis. Cows were allocated 7 or 11 kg of concentrates per day through computer feeders and free choice grass silage from weeks 3 to 22 of lactation.[16] The high concentrate group had 4 times as many episodes of lameness as the low concentrate group. In a 2 lactation study, sole ulcers were about twice as frequent in the cows fed up to 11 kg concentrate per day even though a 3 wk period was allowed for adjustment.[17]

A low proportion of Israeli calves developed laminitis in winter beginning at 3.5 months-of-age on a calf growing farm that raised 600 head per year during a 5 yr period.[18] The only factor found to differ between this farm and others in the area was the 18% crude protein concentrate fed *ad libitum* until 4 mo. Winter feed consumption was higher than the rest of the year. Changing to a 15% protein feed apparently solved the problem. Others have associated laminitis with feeds high in protein such as lush spring grass or grass silages with high ammonia content.[2] The evidence is not nearly as strong that protein or nitrogen intake *per se* is as important a contributing dietary factor as starch. Early spring grass can have a very high sugar content. The presence of ammonia from grass silage may facilitate the growth of lactate producing bacteria.

Providing and maintaining adequate effective fiber intake during the transition from dry and close-up feeding to early lactation rations is the most common challenge in preventing ruminal acidosis. Defining fiber requirements in easily measurable units must go beyond ADF and NDF. Separating forage particles with graded sieves and determining the rumination time per unit of dry matter of a forage consumed are both considered valuable indicators of fiber effectiveness. I consider the development of a standardized system for evaluating forages for effective fiber as important as determining the relative rates of carbohydrate and protein digestion.

Feeding management and cow environment

Indirect but compelling evidence suggests that time spent standing on concrete exacerbates the effects of ruminal acidosis on the hoof structure. Two farms in Somerset, England were under the same management.[19] One farm had a 3 year history of severe laminitis in first calf heifers. If lame heifers were moved to the other farm they recovered. Heifers stood in the alleys or half in-half out of the stalls a greater proportion of the time at the farm where they became lame. The barns were considered identical in layout and dimensions of stalls. The major difference between the 2 farms was in stall maintainance. Four times as much bedding was used each day in the stalls at the farm where laminitis did not occur. In a large cross-sectional study of dairy calves in the Netherlands, the odds ratio of developing sole hemorrhages was 0.2 for calves raised in a straw yard versus slatted floors.[20] Management strategies to encourage adequate lying time include: proper stall design

and dimensions, adequate bedding, minimal overcrowding in freestall barns, and minimizing parlor holding area time to 3 hours per 24.

Even when the feeding management appears to provide adequate effective fiber, outbreaks of laminitis may occur. Five herds without previous problems of laminitis were investigated because of sudden problems in first calf heifers.[21] In all herds there was some abrupt change in the routine for the heifers. They were either newly introduced to concrete or to competing with older cows. Social stress may lead to altered behavior both in eating patterns and in lying time. In many herds with laminitis problems, the first calf heifers are most severely affected. Introducing the heifers to the environment, the feed, and the milking parlor before calving may lessen these social stresses. Many freestall herds separate first lactation heifers into a separate group or pen. This makes them all of similar size and lessens the stress of competition with mature cows.

In the past few years I have investigated several herds with problems of laminitis that occurred in the late summer. These herds did not have significant laminitis problems throughout the year and were not seasonal calving. The common scenario has been changes in cow behavior in response to hot weather and flies. The cows were reported to stand in a bunch at one end of the barn during most of the day. Most feed consumption was at night and in the early morning. Acidosis was occurring due to the slug feeding being practiced by the whole herd. Laminitis was exacerbated by the time spent standing during the day to avoid stable flies and during the night to eat. Recognizing these behavioral changes in a herd should lead to efforts to control fly populations, cool the cows to encourage more even feed consumption, and increase the effective fiber of the diet.

Summary

Laminitis is a common cause of lameness in dairy cattle and calves. Prevention of damage to the claws will improve productivity and longevity. The problems of acidosis and laminitis are not restricted to the highest producing herds. Management errors occur at all levels of production. However, as we develop strategies to feed heifers to reach calving size by 20 mo and cows capable of producing 30,000 lb of milk per year, ruminal acidosis and laminitis will become increasingly important in the health management of our herds.

References

1. Guard CL. 1994. Costs of common diseases of dairy cattle. Cornell Centennial Conference for Veterinarians, Ithaca, NY. 3/20-22/93. 2. Nilsson SA. 1963. Clinical, morphological and experimental studies of laminitis in cattle. *Acta Vet Scand* 4 suppl 1:1-301. 3. Allen D, ES Clark, JN Moore, KW Prasse. 1990. Evaluation of equine digital Starling forces and hemodynamics during early laminitis. *Am J Vet Res.* 51:1930-1934. 4. Coffman JR, JH Johnson, MM Guffy, *et al.* 1970. Hoof circulation in equine laminitis. *J Am Vet Med Assoc.* 156:76-83. 5. Robinson NE, JB Scott, JM Dabney, *et al.* 1976. Digital vascular responses and permeability in equine alimentary laminitis. *Am J Vet Res.* 37:1171-1176. 6. Hood DM, MS Amoss , D Hightower, et al. 1978. Equine laminitis. I. radioisotopic analysis of the hemodynamics of the foot during the acute disease. *J Equine Med Surg.* 2:439-444. 7. Hunt RJ. 1991. The pathophysiology of acute laminitis. *Comp Cont Ed Vet.* 13:1003-1009. 8. Takahashi K, BA Young. 1981. Effects of grain overfeeding and histamine injection on physiological responses related to acute bovine laminitis. *Jap J Vet Sci.* 43:375-385. 9. Boosman R, CWAAM Mutsaers, A Klarenbeek. 1991. The role of endotoxin in acute bovine laminitis. *Vet Quart.* 13:155-162. 10. Choquette-Levy L, J Baril, M Levy, H St-Pierre. 1985. A study of foot disease of dairy cattle in Quebec. *Can Vet J.* 26:278-281. 11. Arkins, S. 1981. Lameness in dairy cows: Part 1. *Irish Vet J.* 35:135-140. 12. Russell AM, GJ Rowlands, SR Shaw, AD Weaver. 1982. Survey of lameness in British dairy cattle. *Vet Record.* 111:155-160. 13. Toussaint Raven E. 1989. Cattle Footcare and Claw Trimming. Farming Press Books, Ipswich, United Kingdom 14. Giesecke D, M Stangassinger. 1980. Lactic acid metabolism. in *Digestive Physiology and Metabolism in Ruminants.* ed Y Ruckebush, P Thivend. AVI Publishing Co, Westport, Conn, USA. pp 523-540. 15. Ogimoto K, 1977. Microbial investigation of the lactic acid metabolic system in the rumen of feedlot cattle. *Proc Anim Nutr Metab.* 21:74-86. 16. Manson FJ, JD Leaver. 1988. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. *Anim Prod.* 47:185-190. 17. Peterse DJ, S Korver, JK Oldenbroek, FP Talmon. 1984. Relationship between levels of concentrate feeding and incidence of sole ulcers in dairy cattle. *Vet Record.* 115:629-630. 18. Bargai U, I Shamir, A Lublin, E Bogin. 1992. Winter outbreaks of laminitis in dairy calves: aetiology and laboratory, radiological and pathological findings. *Vet Record.* 131:411-414. 19. Colam-Ainsworth P, GA Lunn, RC Thomas, RG Eddy. 1989. Behaviour of cows in cubicles and its possible relationship with laminitis in replacement dairy heifers. *Vet Record.* 125:573-575. 20. Frankena K, KAS van Keulen, JP Noordhuizen, EN Noordhuizen-Stassen, J Gundelach, D-J de Jong, I Saedt. 1992. A cross-sectional study into prevalence and risk indicators of digital haemorrhages in female dairy calves. *Prev Vet Med.* 14:1-12. 21. Bazeley K, PJN Pinsent. 1984. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. *Vet Record.* 115:619-622.