# LAMINITIS: A MAJOR CAUSE OF LAMENESS IN FLORIDA DAIRY CATTLE

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## INTRODUCTION

Laminitis is characterized by damage to the sensitive laminae of the hooves. It is manifested by severe lameness in the acute form, abnormal hoof growth in the chronic form, and deterioration of hoof horn quality predisposing to various secondary disorders of the foot in the subclinical form of the disease.

Survey data on hoof characteristics and foot lesions indicate that laminitis is a leading cause of lameness in Florida dairy cattle.[1] While there are other precipitating factors including a genetic predisposition (recorded in Jersey heifers)[2] and trauma to the feet associated with excessive exercise[3] or housing on concrete, laminitis is most commonly linked with feeding conditions which induce rumen lactic acidosis.[4]

## EPIDEMIOLOGICAL FACTORS ASSOCIATED WITH LAMINITIS

### Nutrition and Feeding

Laminitis, particularly that associated with lactic acidosis, is of minimal concern for pastured cattle which are limit fed concentrate feeds. However, where cattle are being challenge fed high levels of concentrate feed to achieve maximum milk production potential for rumen acidosis and laminitis increases. Therefore, incidence of laminitis is highest during the first 60 days of lactation, and first lactation animals are usually the worst affected.

During periods of severe heat stress feed intake drops markedly and the rate of nutrient passage through the gastrointestinal tract slows.[5] To circumvent this problem, feeding strategies call for increasing the energy density of rations. The result is a tendency toward rumen acidosis and subsequently, laminitis.

Presented at the 3rd Florida Ruminant Nutrition Symposium, Gainesville, January 23-24, 1992

## Peripartum Disease

Peripartum diseases such as retained placenta, metritis, udder edema, and mastitis have also been incriminated as predisposing causes of laminitis. However, because of the variability in incidence rates within and between herds, the exact relationship between peripartum disease and laminitis remains unclear.

## Confinement Housing

Finally, confinement-type housing continues to grow in popularity as dairies endeavor to comply with environmental regulations and achieve the goals of heat stress abatement. The down-side to confinement housing is that it restricts cows to smaller areas for longer periods of time. This generally means less opportunity for exercise and confinement of cows on concrete which is a harder, wetter, and more abrasive flooring surface.

Cattle hooves are continually wet during much or all of the year under confinement housing conditions due to the continual application of water from sprinkler and floor flushing systems. Consequently, hoof horn tissue remains soft. When combined with laminitis, which also softens the hoof horn tissue, the rate of hoof wear on concrete increases dramatically, particularly if the concrete surface has a rough or abrasive finish. Indeed, it is not uncommon for cattle in such conditions to wear the soles of their feet through to the underlying corium tissue after only 1-2 weeks on rough concrete. The syndrome outlined in the following case report is typical of those seen under such conditions.

## CASE REPORT OF A SEVERE LAMINITIS OUTBREAK ON A FLORIDA DAIRY

During the summer of 1990 laminitis on 1 Florida dairy resulted in enormous financial loss and the premature culling of more than 100 cows. Peak losses associated with reduced milk production, involuntary culling, increased labor expense, and drug and veterinary costs were incurred during the months of July, August, and September. Of cows sold from the herd for lameness, more than 60% were 1st lactation animals less than 60 days in milk.

The clinical syndrome varied, but for most animals lameness began very early in lactation. Several animals developed symptoms typical of acute laminitis as evidenced by pain in all 4 feet and an extreme reluctance to stand or walk. Some never recovered from this stage, whereas others improved with time. Still others did not show any of the typical signs of laminitis but became acutely lame in 1 or 2 feet only, presumably due to secondary foot disease (sole abscesses, sole bruises, etc.). Hemorrhages were readily visible on the soles of many of the affected cattle. Hoof horn tissue was soft and pared away effortlessly when cut with a hoof knife.

The rate of hoof wear was accelerated in the majority of animals. Hoof soles were thin and in some cases had either worn through or separated at the white line exposing the underlying sensitive laminar tissues. Affected cattle generally progressed to more severe forms of foot sepsis which extended to adjacent soft tissues and joints of the foot and fetlock. Most of these did not recover and ultimately had to be destroyed or prematurely sold from the herd.

Review of the feeding, housing, and management practices up to the point of the outbreak revealed several potential contributing factors. The milking herd ration was a nutrient dense ration which in retrospect lacked sufficient fiber. Long hay was not available to lactating animals and the introduction of 1st lactation heifers to the milking ration may not have been gradual enough to avoid lactic acidosis and the laminitis that subsequently developed. A second factor that likely influenced the problem experienced in this herd was the placement of 1st lactation animals on concrete in a newly constructed shade barn in May, approximately 1-2 months prior to the outbreak. The surface of the concrete was rough and abrasive. The combined effects of the high concentrate to forage ratio, rapid introduction of high energy feedstuffs, and confinement on new concrete are believed to have been major contributors to the laminitis, elevated rate of hoof wear, and related foot problems that developed in this herd.

## THE PATHOGENESIS OF LAMINITIS

Laminitis is generally regarded as a local manifestation of a systemic disturbance, but there are at least 2 possible explanations for the lesions which result. One hypothesis suggests that the lesion is initiated by an apparent disruption of blood flow through the laminar tissues (corium) of the foot. Specifically, capillaries become engorged with blood due to a constriction of the vasculature (blood vessels) on the venous side. With the flow of blood impeded, pressure builds up within the capillaries forcing fluid to move through the vessels into the surrounding tissue (interstitial) spaces. The effect of this movement of fluid into the interstitial spaces increases resistance to blood flow still further ultimately rendering the laminar tissues ischemic (deficient of blood). Deprived of blood and thus nutrients tissues of the corium proceed to dysfunction and death evidenced by breakdown of the epidermal horn bonding the hoof to the underlying laminar tissues.[4]

Reduced blood flow to laminar tissues results in a reduction in the net flow of nutrients (methionine, cysteine and cystine) which are necessary for the synthesis of keratin.[6] The incomplete formation of keratin reduces the integrity of the onychogenic attachment between the laminar epithelium and the hoof horn thereby predisposing them to separation. Separation of the hoof horn from

the digital cushion and pedal bone leads to further pooling of blood and serum in the laminar tissues. As a result the toe begins to rotate downward toward the sole. In particularly severe cases, it may actually penetrate the sole.

Although the exact mechanism is unknown, lactic acid and endotoxin are alleged to be a couple of the prime causative agents responsible for the venoconstriction and subsequent engorgement of terminal blood vessels in laminar tissues.[7,8,9] Previously held concepts that the vascular engorgement was a direct result of the action of histamine are in question.[10]

The second hypothesis suggests that the vascular changes as described are secondary to a primary defect in the formation of the epidermal horn which weakens the bond between the hoof and corium. This predisposes to traumatic separation of the hoof wall from the sensitive lamina and results in vascular damage which accounts for the engorgement of blood in laminar tissues. Current information would suggest that the former hypothesis is more likely.[4]

## TREATMENT AND PREVENTION OF LAMINITIS

Therapy for the acute laminitis condition would include treatment with anti-inflammatories and movement of the animal to a grass pasture, dirt lot, well-bedded stall, or other area free of concrete, gravel or stone. The animal should be fed hay only and reintroduced to concentrate feed gradually over several days. Beyond regular hoof trimming there is no specific treatment for chronic laminitis.

Rations should be formulated and fed to maximize intake but minimize potential for lactic acidosis. The feeding of a total mixed ration (TMR) not exceeding 60-65% concentrates with 5-15 lbs. of chopped hay is advised for feeding of early lactation cows. Limited evidence suggests that various micronutrients namely; sulfur, zinc, and copper are important to maintenance of hoof health.[11,12,13,14] Finally, the addition of 1% sodium bicarbonate is also recommended to buffer rumen contents.

Formalin (5%) footbaths are recommended by some as an aid to harden hooves (See appendix 1). Concrete should be finished smooth to reduce abrasiveness and grooved to prevent injury due to slipping. Cows should have access to dirt lots or pasture for at least 10-12 hours per day. Rubber belting (conveyor-type belts) has been used with some success in feed barns where cows do not have access to free stalls or suitable dry lots.

Finally, one of the most important considerations regarding treatment, control, or prevention with any lameness problem is to determine the cause or causes. Laminitis is a very debilitating disease and it may occur as an acute, chronic or subclinical

problem. Don't wait to see if it gets better, cut the cow out of the herd and examine the foot. Prompt attention not only improves the likelihood of a cure for the individual affected, but also increases the probability of recognizing a particular condition before it becomes a major herd problem.

This is particularly true for subclinical laminitis in which the clinical signs of disease are subtle or in fact misleading. The classical symptoms of "founder" are absent. Instead, observations of any of the following: 1) soft, yellowish, hoof material, 2) hemorrhages, particularly noticeable on the sole, 3) an increase in the number of lame cows with sole abscesses or ulcers, 4) records which would indicate an annual incidence of herd lameness, excluding that associated with footrot, above 5%, or 5) herd records demonstrating that over 50% of herd lameness is occurring within the first 60-90 days of lactation, should signal to the observer that laminitis may be the underlying lameness problem.[15]

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APPENDIX 1.

## FORMALIN FOOTBATH

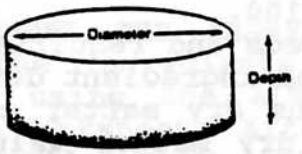
A. 5% Solution = 1 gallon of 36% formaldehyde in 19 gallons water.

1. It is very irritating when inhaled and should only be used or mixed outdoors. It is also irritating to the skin and feet and should not be used repeatedly as a footbath at greater than 5% concentration. Formalin tends to harden the hooves with repeated use. Any residue solution should be discarded and a fresh batch mixed before each use. Otherwise, the mixture may become too concentrated from evaporation.

2. Formalin is monitored by the Environmental Protection Agency under SARA Title III and all suppliers and/or distributors are required to keep records of sales. No more than 500 lbs. should be kept on site at any one time.

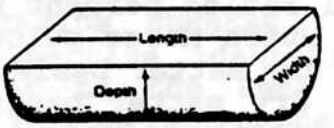
## FORMULAS FOR CALCULATING THE CAPACITY OF TANKS AND TROUGHS (in gallons)

**CIRCULAR TANK**



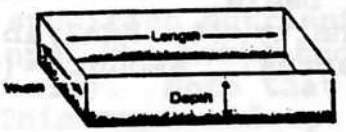

**Diameter x Diameter x Depth x 5.86 = Number of Gallons**

**ROUND END TANKS**



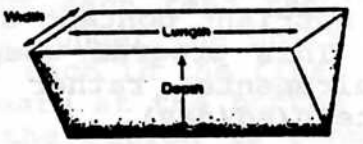

**Width x Depth x Length x 2.83 = Number of Gallons**

**RECTANGULAR TANK**




**Width x Length x Depth x 7.46 = Number of Gallons**

**V-END TROUGH**




**Width x Depth x Length x 3.73 = Number of Gallons**

**As a general rule, cattle will consume approximately 1 gallon of water per 100 lbs. of body weight. However, in hot weather water consumption may increase by as much as 30-100%.**