Laminitis

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Laminitis has become a fashionable diagnosis in dairy cow lameness. Some fifteen years ago, Dutch colleagues vehemently denied the existence of this problem in their country. Nowadays they not only agree that it occurs, but they, along with some Scandinavians (Mortensen, Andersson, Nilsson), have been among the few critical observers of the clinical condition, and have also successfully reproduced laminitis in cattle. Let us pose a series of questions and attempt answers.

1. Does laminitis really occur in dairy cattle?
2. What is its cause?
3. How can I diagnose laminitis?
4. How do I treat it?
5. Can laminitis be prevented?

1. *Without doubt, laminitis does occur in dairy cattle.*

Laminitis is an aseptic pododermatitis affecting the laminar and solar corium. It is used incorrectly as a diagnosis in many dairy cows which walk tenderly. The facts are hard to obtain. Several nutritional models have been developed to produce an acute laminitis, all based on a relatively high intake of concentrate feed which may or may not be grain, giving a high carbohydrate intake, while the protein concentration and total intake have remained within normal limits.

The signs of *acute laminitis* are sudden in onset. The cow shows severe discomfort when standing or walking, is anorexic, reluctant to walk, has a pronounced digital pulse in all four legs (best felt over the fetlock), and has warm claws tender to hoof pincers. Histological changes in the dermal laminae include edema, congestion, thrombosis and a loss of onychogenic (hornproducing) substance in the stratum germinativum and str. spinosum. Acute laminitis is an emergency and treatment (below) must be adopted quickly if a favorable response is to be obtained.

Subacute laminitis is self-explanatory, but a newer phrase, *subclinical laminitis,* requires explanation. An abnormal stance or gait is not evident, although inspection reveals horn changes including a generalized softening of the solear horn, which becomes yellowish and may have extensive areas of hemorrhage. Some cases show erythema and mild edema of the coronary band. The cause may be a more prolonged intake of high concentrate feedstuffs under unsatisfactory environmental and husbandry conditions than a sudden insult to the cow's system.

Chronic laminitis has certan characteristic visible features, many being very similar to the horse. The dorsal angle is reduced from about 55° (normal) to 35-45°, the dorsal wall surface may be concave, and invariably there are more or less obvious horizontal lines (growth arrest lines) extending around the entire circumference of the wall. There may also be more obvious deeper linear indentations. These lines are symmetrical in all eight claws. The coronary band is flaky and the perioplic sheen is lost. The sole tends to be dropped, and one result is an increased liability to solar bruising, manifested as areas of hemorrhage. Their color is dark grey or black when long-standing or pink if recent or continuing. Some cases of acute and chronic laminitis have a tendency to develop solar ulcers some weeks or months later. Other complications of chronic laminitis develop slowly as a result of the uncontrolled claw deformity. Commonly the abaxial wall of the claw, particularly in the hind limb, overgrows to form a second sole. At the same time the position and shape of the underlying abaxial white line is altered, becoming wider, disrupted and a favorite site for entry of foreign material such as stones and gravel which work their way through an entrapped position between true sole and the overlying abaxial wall. Lesser degrees of disruption affect the axial wall. The existence of such problems in numerous claws makes control very difficult.

2. *What is the cause of laminitis?*

As in many problems (for example environmental mastitis) it is over-optimistic to think that there is a single, simple cause of laminitis. Certainly some strains of dairy cattle (e.g. Holsteins) are genetically predisposed to laminitis. The key is the smaller than average angle of the dorsal wall which, with only a modest amount of claw overgrowth, becomes dangerously smaller still. This change results in a tendency for weight to be transferred increasingly onto the heels. A vicious cycle develops as the toe horn is still less likely to be worn away, leading to a longer pointed toe. McDaniel of North Carolina in speaking to the AABP has pointed out the reduced "survivability" of first calf heifers with a significantly smaller dorsal angle than normal. He agrees that such feet are predisposed to trouble throughout the cow's life

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and that laminitic incidents play a large part in the deterioration of claw shape in these cattle. Other reasons for the development of laminitis have been recognized for many years. They include a tendency to feed large amounts of concentrate feed twice a day, often into a relatively empty rumen, permitting the development of a relative lactic acidosis. A tendency to put point-of-calving heifers or cows into a confined area where exercise is very limited, and onto rough irregular concrete surfaces exacerbates the potential for laminitis. Many surveys show that laminitis is particularly common around calving time and for about four weeks postpartum. Ironically, in New Zealand laminitis tends to develop following extensive exercise to and from pastures for parlour milking. Under this system the forage is of such high quality that little concentrate is fed. Excessive exercise has been blamed in such herds.

3. *How can I diagnose laminitis?*

Acute laminitis: a sudden onset of laminitis is seen in predisposed animals within one month of parturition under conditions of confinement and relative excess of concentrate feeding. The heifer or cow appears stiff, stays recumbent longer than expected, and walks with back arched. Erythema and possible slight edema is evident at the coronary band of all digits. The claws are hot and the digital pulse is appreciable. The cow is reluctant to raise a limb for palpation and exploratory hoof-paring. No evidence of digital sepsis or solar ulcer is evident. After a few days, in those cases with a flat sole, pink hemorrhage indicative of bruising is seen. The sole is usually composed of relatively soft, slightly yellow horn which can be depressed by digital pressure.

Subacute laminitis: less pronounced signs are seen than in acute disease, it is not necessarily related to parturition, and may be an exacerbation of the acute condition.

Subclinical laminitis: the solar horn is soft, yellow and may have extensive hemorrhages. Some cases have a widening of the white line and a liability to impact debris there. A cow may present as a secondary penetration of the sole by a foreign body, which causes trouble only because the sole is thin and of poor quality. The primary problem has then been acute laminitis with soft horn production, excessive sole wear and a flat sole.

Chronic laminitis: characteristic signs include the shape of wall, the ridging and the flat sole with possible overgrowth of the abaxial wall. Deviation of the distal phalanx, a consistent feature in equine chronic laminitis, is less pronounced and inconsistent in cattle. The reason is not known, but may be associated with the different anatomical relationship of the tip of the distal phalanx in the equine and bovine.

4. *How is laminitis treated?*

Acute laminitis requires intensive emergency treatment. The immediate aim is to get the cow moving. To this end she should receive flunixin meglumine (Banamine) to counter the endotoxic shock reaction resulting from the acidosis and rumenitis, corticosteroids, unless in advanced gestation, and possibly 500 gm magnesium sulfate to purge the animal. If unwilling to stand, anecdotal evidence suggests that local anesthetic block of all four feet may make the animal mobile. The concentrate intake should be drastically reduced and the animal put onto soft bedding. Chronic laminitis requires foot trimming but it is often basically not correctable due to irreversible changes. Those cases of chronic laminitis which have deep horizontal rings may become lame as the distal strip of horn moves in a hinge-like fashion as the cow walks. It is advisable to trim off this distal horn at the earliest possible time rather than wait for it to break off.

5. *How is laminitis prevented in dairy cattle?*

Laminitis is prevented by good husbandry. The increased size of dairying enterprises and the loss of the family dairy farmer with his or her attention to the individual has in some cases led to frustration of veterinarians in the attempts to improve management techniques. Several rules have been repeatedly reiterated in an attempt to avoid the rumen acidosis syndrome.

- All feeding changes should be made slowly.
- Dry cows should receive minimal amounts of concentrates (i.e. maximum 2 lb. daily.)
- Cows starting their lactation should have the concentrates increased slowly to lead to peak milk yield at about 5-6 weeks rather than the common aim of three weeks. There will be a significant saving in concentrates to compensate for the slight drop in total lactation yield.
- Concentrates should be fed in small amounts four times a day rather than in two feeds around milking time.
- Again, in an attempt to prevent the regular occurrence of a relative acidosis, cows should be encouraged to get back to roughage feed after concentrate consumption.
- Measures to increase saliva production by feeding of long-stemmed alfalfa will help.
- Addition of 1% (but not 3%) sodium bicarbonate in the concentrate will help to counter the incipient rumen acidosis.
- Systems which have a complete diet (as in Israel, but rarely seen in this country) will avoid the potential problems of acidosis.
- Cows should be given plenty of loafing space and exercise should be encouraged. In some places the location of feed bunks at opposite ends of a yard will help. Since freshly calved heifers appear to be at high risk, they should be handled gently, should not be bullied by the older cows, and should have plenty of bunk space to feed.

Experiments have shown that a high fiber diet (total roughage exceeding 33% of total intake) leads to less problems with clinical laminitis and a lower incidence

of sole hemorrhages, indicative of subclinical laminitis, than a high concentrate diet (e.g. 11 kg compared with 3 kg daily concentrate intake).

Many practitioners have observed herds with a poor usage of free stalls. Some premises have new concrete surfaces composed of a coarse type of aggregate. Cows are reluctant to move around freely on such surfaces. Sometimes, as explained by Cermak at the 1987 AABP meeting, cubicles may be new, uncomfortable, incorrect in dimensions, and too few for the herd size. Social rank may prevent heifers from using empty stalls. In one problem herd with laminitis, the replacement of wood shavings by straw bedding, lengthening the freestalls by moving the head rail 7" forward, hoof trimming before housing in October, and once weekly use of a 5% formalin footbath, without any dietary changes, reduced the lameness rate from 40/44 cows lame in year 1 to 4/44 lame in year 2.

In summary, the key to prevention is old-fashioned good husbandry and individual attention.

Health, welfare and fertility implications of the use of bovine somatotrophin in dairy cattle

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Twenty-two Friesian and Friesian cross Ayrshire cows and 16 first lactation heifers were paired. Twenty were injected subcutaneously with 500 mg recombinant bovine somatotrophin (sometribove) and 18 with a placebo at fortnightly intervals, starting 80 (±7) days after calving, through the winter of 1986-87. The cows' weights and changes in condition, milk yields, milk solids, health and fertility were recorded regularly. Compound cake was fed at a fixed stepped rate according to the number of days since the cow calved. Silage was available on an easy-feed basis. Ten cows had their daily silage intake measured. Sometribove treated cows produced on average 27·7 kg of milk per day, 4·5 kg more than the controls. Treated heifers produced 23·5 kg per day, 2·5 kg more than the controls. Milk quality was unchanged. Treated cows gained weight during the trial, but not by as much as the controls. Control heifers also gained weight but the treated heifers lost, on average, 3 kg. Local reactions at the injection site were not felt to be of welfare concern, nor was the general effect of the extra milk production. A small number of treated animals experienced mastitis and had poorer fertility but the differences were mostly not statistically significant. If bovine somatotrophin should become licensed for use in Britain it is recommended that clear guidelines should be issued on the management practices necessary for economic success and for the welfare of the treated animals.

Pasture study of two types of oxfendazole pulse release bolus for controlling nematodes in calves

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One group of first-season calves was dosed with an oxfendazole pulse release bolus at spring turnout (April 30) and on July 15 a second group received the front-loaded oxfendazole pulse release bolus. The objective was to test the boluses for the prophylaxis or control of nematodiasis. The control group consisted of calves to which no bolus was administered. The three groups occupied separate but adjacent plots. For the first five weeks of the trial, three calves, artificially infected with *Dictyocaulus viviparus* grazed in each plot. Parasitic bronchitis severely affected the control calves, necessitating repeated emergency treatments, whereas administration of the bolus at turnout almost completely prevented this condition. *D viviparus* infection increased markedly on the control herbage in July and August but was eliminated by the end of June on pasture grazed by bolus treated calves. Treatment in mid-season with the front-loaded bolus brought an outbreak of parasitic bronchitis under control. Gastrointestinal worm egg output was satisfactorily suppressed after the administration of both boluses, resulting in reduced levels of herbage infection. Calves treated with a bolus at turnout gained significantly more weight than either the controls ($P < 0{\cdot}001$) or the calves treated with a front-loaded bolus in mid-season ($P < 0{\cdot}01$). The weight-gain of the calves treated with a front-loaded bolus was slightly but not significantly greater than that of the control calves. On the basis of faecal egg counts, the first pulse released from the standard boluses was delayed and one front-loaded bolus failed to release a dose. However, under the conditions of the study, these events did not affect the efficacy of the boluses.