The Management and Husbandry Aspects of Foot Lameness in Dairy Cattle

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Introduction

There have always been cases of foot lameness in dairy cows. Even in prewar years there were lame cows, but the majority of these suffered from foul-in-the-foot (infectious pododermatitis), and from the complications of its neglect, whilst other developed pus-in-the-foot due to picked up nails and other penetrating foreign bodies. The incidence was relatively low, and the position of minor economic importance only.

In the post war years of intensive development in dairy managment, however, and particularly since the introduction of loose winter housing, the picture has changed and foot lameness has assumed alarming proportions, becoming a major economic problem in many dairy herds. It is estimated that 10% of all dairy cows in intensive management become lame each year, and that one half of these, i.e. 5%, are lame as a result of laminitis. A rough estimate suggests that lameness costs the dairy industry some £15,000,000 annually, but the true cost may be considerably higher, for many lame cows are recorded as culled due to the resultant poor milk yield, weight loss, or infertility, rather than to the primary lameness itself.

Until comparatively recently very few farmers or veterinary surgeons were genuinely interested in foot lameness, which, by and large, was regarded as an inescapable fact of life, however, the position is now far too serious to allow for futher complacency, and considerable efforts are being made to come to grips with this alarming problem, and to work out satisfactory preventive measures based on a study of the causative factors.

Incidence and Etiology

Long experience with lameness in dairy cows shows that some 90% of lame cows are lame in the foot. It follows that only 10% are lame in other sites, which are therefore of little overall economic importance.

Purely as a matter of interest, it is probably worth reporting that the most common such site is the hip and then considering the causes of hip lameness. It is commonly believed that dietary calcium-phosphorus imbalance on farms with low soil phosphate levels is important in this connection.

The writer has never been able to convince himself that such cases, if they occur, are of any importance and would prefer to consider a) *fluorosis* which produces a high level of hip lameness in adult cows exposed to abnormal fluoride levels for long periods. This may occur in areas of naturally high soil fluoride levels, and occasionally by wind-borne distribution of dust with a high fluoride level on the leeward side of new motorways, industrial plants, etc. Usually, however, it results from the use of cheap mineral mixtures, for many crude phosphate minerals have an unacceptably high fluoride level. b) trauma, osteoarthritic change not infrequently occurs in the hip of the older cow as the result of regularly occurring slight injury. When an unduly high incidence of hip lameness is present in old cows in a herd it is always worth while checking doorways, steps, corners, and gateways with a view to eliminating any hazard which may lead to frequent slipping, sharp turning, or colliding with posts, walls, and other obstacles.

It must be remembered that, quite apart from hip damage, the modern dairy cow is almost continually exposed to injury in modern yard and parlour arrangements. Collecting yards are particularly dangerous, for farmers seldom wash them down adequately, and especially in dry weather the slimy faecal coating smeared over the concrete is extremely slippery, particularly for cows heavily pregnant, recently calved, crowded, hurried, or in oestrus.

Hygroma of the knee due to deficiency of bedding in the cubicles, or "shippon hock" following abrasions on the lateral aspect of the hock due to friction on the cubicle edge, also occur fairly frequently. Nevertheless approximately 90% of lame cows are lame in the foot and approximately 90% of foot lameness is hind foot lameness. This at first is surprising, for the cow takes some 60% of her weight on her fore legs. But weight bearing does not cause as much stress as propulsion and the hind legs are involved in propulsion during which the hind feed suffer much friction, particularly along the white line of the outer aspect of the outer claw. Friction is the enemy. It is also true that the hind feet have a smaller ground area than the fore feet which in itself exaggerates any stress. They also tend to be more exposed for a longer time to the softening and erosive effects of slurry. In fact very few lameness conditions are specific to the front feet. "Foul in the foot" ("infectious pododermatitis") affects the fore or hind feet without discrimination and therefore one knows that a cow lame in a front foot is probably a case of "foul in the foot". This condition is, of course, the result of the infection of injuries and abrasions in the interdigital cleft and heel by *Fusiformis* necrophorus, which causes acutely painful inflammatory reactions at these sites followed by linear necrotic change and secondary bacterial invasion in the cleft, often underrunning the horn at the sole-heel junction. Fusiformis can survive and accummulate in wet deep litter, muddy gateways, wet tracks, and other commonly used moist areas of the farm, and so a great deal can be done to control the disease by simple measures of hygiene and drainage, even as simple as the temporary use of alternative gateways while walking the herd between pasture and buildings. One should remember that there has to be an abrasion to allow entry of the organism, so that traumatic hazards such as stony tracks, kale stumps, and corn stubble should be avoided if possible. Foot baths are useful in this, possibly more than any other, type of foot lameness. It must be remembered however, that a foot bath improperly designed and sited is both useless and expensive. Foot baths must be placed so that the cows can be diverted, automatically, through the bath on their exit from the parlour; there must be a wash bath through which the cow walks before she reaches the medicated bath, which must be long enough to avoid any possibility of the cow hopping over it, deep enough to immerse the foot to the coronet, but not so deep that the preparation splashes the udder and teats. The foot bath must lead on to a dry clean area of concrete where the cows may stand for some twenty minutes before proceeding back to the field or cubicle yard. The writer advocates the use of formalin at 2-4% or copper sulphate at 10%, and suggests that two foot treatments weekly should be ample.

Laminitis may affect any or all feet. In the fore feet the inner claws are generally worse affected, probably because they bear the greater weight, while in the hind feet the outer claws, exposed to the greatest friction, suffer more. Laminitis is such an important disease that it merits full discussion in its own right.

Bilaterally symmetrical fracture of the third phalanx is a genuine fore-foot lameness, very seldom affecting the hind feet. When one fore-foot alone may be affected, and occasionally bilaterally asymmetrical fractures occur, it is usual in "spontaneous" fracture of this bone for both inner claws to be affected, presumably as the greater weight bearers. When bilateral, the condition is clinically very similar to acute laminitis of the front feet, and probably radiography will be needed to confirm the diagnosis. The condition is most commonly met with in the high fluoride areas, but also occurs occasionally elsewhere. Possibly low copper levels may predispose, but there are almost certainly other factors involved as for example significant overweight in a young and growing animal.

Whilst in recent years there has been much surmise as to

the effect of minerals and vitamins e.g. zinc and biotin, in rendering the horn more resistant to stress, no deep consideration seems to have been given to the various stresses which may affect the bovine pedal bone.

So 90% of foot lameness is hind foot lameness, and 90% of hind foot lameness is outer claw lameness.

The outer claw is often smaller in area than the inner claw and it sustains greater friction forces as the cow propels herself forwards, particularly along the lateral white line.

It is obviously worth considering the background factors predisposing to this high incidence of foot lameness, hind foot lameness and outer claw lameness.

1. Conformation - this is an obvious factor. In particular the straight and upright hocks of the modern Friesian must accentuate impact and friction forces. The modern Friesian has lost its shock absorbers. Friesian feet, big, soft and often showing congenital deformities of the outer claws are obviously much more prone to damage than the small hard and compact feet of the Jersey, or the Shorthorn, now unhappily almost extinct. Probably there are other factors related to conformation involved in lameness. Many breeders are well aware of the possibilities and it is work suggesting that it is sensible to cull vigorously the offspring of a chronically or continually lame cow.

2. Nutrition - the correlation between nutrition and lameness has been an important subject for some years, the link being, of course, the condition known as laminitis. *Laminitis* has become a very important and serious cause of lameness in the dairy cow.

The writer's experience supports that of Maclean (1965) who carried out an important survey nearly twenty years ago. Some 20% of the writer's lameness cases are clearcut laminitic cases, but it is now becoming clear that laminitis is even more important as a predisposing cause of other foot conditions including solar ulcer, solar "bruising", some white line lesions, and various foot deformities or overgrowth such as "Turkish slipper" and "shovel" feet. It is probable that some 50% of foot lameness is *primarily* laminitic, while the writer's experience again supports very strongly the finding of Maclean (1965) that it is virtually impossible to examine postmortem the foot of any dairy cow about six years of age in an intensive management without finding laminitic lesions.

Laminitis is due, of course, to toxic insults to the vascular structure of the feet, and may therefore result from toxic illnesses such as acute metritis, retained membranes, and acute mastitis particularly summer mastitis.

It is also associated with overeating of certain specific foodstuffs, particularly those with a high energy content. Wheat, barley, maize, rape, clovers, and even young grass with its high soluble energy content may all be dangerous.

But undoubtedly the most important association is with heavy concentrate feeding at or after calving. In the herds for which the writer is responsible the highest incidence occurs in regimes where heavy steaming up is followed by a rapid increase in the concentrate ration in the days after calving. It does not occur to anything like the same extent in herds which do not steam up above twevle pounds of cake, which do not exceed the steaming up level for the first week after calving, and which aim for peak yield at 6 weeks post partum rather than the traditional four.

Farmers, like veterinary surgeons, are gradually becoming aware that it is the energy fraction of the diet, and not the protein intake which is critical, and *that the disease is just one manifestation of a ruminal and systemic lactic acidosis* associated with the overwhelming of the normal mineral flora by ingestion of highly fermentable energy foods. Unfavorable organisms such as *streptococcus bovis* become predominant in the rumen, and to the lactic acidosis is added the endotoxic effect of the disintegration of vast numbers of normal rumen bacteria. Silage lactic acid may be contributory.

An artero-venous shunt develops at the coronet leading to congestive and anoxaemic changes in the foot with necrosis of soft tissue and rotation of the third phalanges.

We are all familiar with the classical clinical picture of acute laminitis in the cow. But, for the occasional cow which exhibits the acute picture clinically, there may be many, often the whole group coming up to peak yield, where similar pathological changes are taking place within the feet without clear cut symptoms of lameness.

It will be eight or more weeks later when all changes are irreversible, and long after the offending nutritional regime has ceased to operate, that severe clinical signs will appear.

These will be the sign of *chronic laminitis* - the front wall will show transverse ringing and cracking, the soles will become convex, degenerate and soft, thinning and even peeling back from the white line at the toe; haemorrhages will appear on the ground surface of the sole as blood and exudate slowly work through the horn. The heels become high, the toes long and either shovel or Turkish slipper shaped.

Above all, the dreaded solar ulcer lesion will appear at the sole-heel junction of the outer claws of the hind feet near the axial border, often before the more chronic deformities are well marked.

3. Environment - the cubicle yards, collecting yards, tracks and lanes etc. Here the enemies are friction, erosion and *infection*. All these factors will have exaggerated effects if the feet are already damaged by laminitic change.

a) Friction - friction in this connection relates to concrete, the most evil of the necessary evils met with in animal housing. When smooth it is slippery; when rought enough to give a grip is causes damage to horn. The ill effects are less if it is possible for the cow to move gently and slowly upon it. When she is forced to hurry, to twist and turn, and when she is crowded, it becomes destructive indeed. Concrete is the greatest enemy.

b) *Erosion* - one might justifiably assume that slurry would lubricate the abrasive effects of concrete upon horn. In fact, clinical evidence suggests that slurry is, in itself, chemically erosive and may cause severe ulceration of the

fibrous heel - so called "slurry heel" and softening of solar horn.

c) Infection - the worn and eroded areas of the sole and heel described are obviously prone to infection. But levels of infection in modern dairy cow accommodations are notoriously high - cubicle and collecting yards are filthy places, and the damaged feet become infected. Development of granulation tissue occurs, there is overgrowth of horny sole, and stones, gravel, and sand work into the soft, sodden, diseased horn.

Foot lameness in dairy cows is inextricably associated with management. The highest incidence is in the autumn during and immediately after transition from pasture to winter housing. The cows are at grass by day, they are housed by night. They are at, or just past, calving on a high plane of nutrition, and are acidotic and laminitic. They are made to walk twice daily along stony tracks just becoming muddy as the rainfall increases; at night they stand on concrete at its most abrasive, in slurry at its most erosive.

The worst sufferers are the heifers calving down for the first time, as yet unused to the perils of the collecting and cubicle yards, and of their milking ration. It is interesting in this connection that the later heifers calve, the better chance their feet have of remaining healthy. If they calve at 2 years old, lameness, environmental with a laminitic background, will be a problem. Those calving at three years old may well remain foot sound. Two and a half years would be an ideal compromise between economics and foot health, but unfortunately calves are born in the autumn in the intensive herd; heifers must calve in the autumn to be economic, and therefore must calve at either two or three years of age.

Obviously cows in oestrus and those which ride them are at greater risk: scuffling increases the friction forces exerted upon the hind feet. Equally obviously cows and heifers low in the social order and frequently bullied are at greater risk as they scrabble to escape and turn rapidly away.

It is necessary for herdsmen and owners to consider the possible repercussions upon the cows's feet any change in management, or alteration in policy, which they may make.

Preventive Measures

This is a difficult area. From what has been said a number of preventive procedures come immediately to mind. Unfortunately, as in so many cattle problems of diverse and complicated etiology, it does not follow that normal preventive advice will always be successful. Obviously, therefore, there are still many etiological facets not yet properly elucidated or understood, so that we can do no more than suggest a number of useful procedures which normally repay consideration, while retaining an open mind in case the results are disappointing.

1. Never hurry cows. It has been said that if cows are allowed to move with grace and elegance they will not become lame. Let them take their own time, and above all do not crowd them. All cattle accommodation should be designed so that sudden changes in direction are unnecessary. A smooth flow is essential, avoiding bottle necks and dead ends.

2. The cubicle length must be right. Cows must be able to stand comfortably with their hind feet on the cubicle bed. If they habitually stand with their hind feed in the dung channel the horn will soften and erode and the cow's weight will be improperly distributed. For the same reasons the step up must be as low as is feasible. Allow plenty of room behind cows in cubicles so that cows do not have to turn sharply when they back out of cubicles.

3. Keep the concrete surface in good order and lay new concrete in accordance with the advice of the agricultural advisers of the industry. The right concrete blend properly laid may save much disease and considerable expenditure. Keep in touch with research on newer composite flooring materials.

4. Avoid long walks. Avoid moving cows over rough tracks with a covering of stones, gravel, sand or grit.

5. Keep yards, collecting areas and pathways clean. Use plenty of water. Keep in touch with new developments for continual cleansing systems for cubicle yards. Consider the washing and medication of cows' feet in properly designed foot baths.

6. Avoid large cow units. Suggest that large herds be divided and maintained in units of some seventy cows. As soon as there are so many cows in a unit that the patterns of social dominance break up, one gets anxious apprehensive cows continually taking evasive or aggressive action and thus predisposing to foot damage.

7. Take especial care with feeding in the pre and postpartum periods. Weaver (1979) put forward seven suggestions for dietary care in the prevention of laminitis. These suggestions have proved so useful and so effective in the majority of cases that they are now widely described as the **Weaver rules.** they are worth repetition word for word.

(a) there should be no sudden feeding changes in the four weeks before and four weeks after calving.

(b) there should be a relative reduction of the rate of concentrate feeding after calving so that peak yield is reached at six weeks and not at three to four weeks.

(c) there should be immediate and adequate access to forage and bulk after concentrate feeding.

(d) Free access to iodised or rock salt should be ensured to increase the saliva flow and improve the pH buffering capacity of the rumen.

(e) the provision of grass cubes or lucerne nuts in rations fed pre- and post-partum will ensure a further increase of the rumen buffering capacity.

(f) the inclusion of sodium bicarbonate at the rate of approximately 1% of the bovine mixed ration will improve the rumen pH.

(g) the feeding of concentrates in a number of feeds, i.e. more than two, daily to freshly calved cows will, like the other suggestions, minimize the risk of peaks of rumen acidosis, while ideally the system of complete diet feeding brings the risk to an absolute minimum.

Weaver added two further rules to be used in association with those already described.

(h) downcalving heifers should enter concrete floored yards several weeks beforehand in order to become accustomed to the surfaces.

(i) there should be plenty of exercise for stock in the prepartum month and the immediate post-partum period.

8. Finally, it is very important that farmers should be encouraged to build well designed units for the inspection and dressing of lame cows. All veterinary surgeons engaged in cattle practice are only too well aware that dairy farmers with no proper facilities for foot care usually have a lameness problem. As soon as a well designed unit is built so that the care of feet becomes a pleasure rather than a chore the position invariably improves.

Above all, it should be stressed that the feet of each and every cow in a dairy herd should be examined and trimmed as a matter of routine each spring and autumn. This will only be done if it is easy to do i.e. the foot care unit is efficient, and the farm staff are properly trained in foot care, a fascinating and rewarding subject.

References

Maclean, C. W. (1965) Vet. Rec. 77 24. p. 662. - Weaver, A. D. (1979) Bov. Pract. 14 p. 70.