General Sessions

Moderators - Michael Vaughn
Tom Edwards

Cattle Hoofcare

R.W. Blowey, *BSc, BVSC, FRCVS*
124 Stroud Road, Gloucester
United Kingdom

Lameness remains a major problem in cattle, particularly for dairy herds. Estimates of incidence vary from 10 to 50 cases per 100 cows per year, with the higher incidence being seen in cubicle-housed cattle. This paper outlines some of the common causes of lameness and the factors which can lead to an increased incidence. To appreciate the aetiology of lameness, it is necessary to understand the structure and function of the hoof.

The Structure of the Foot

The foot consists of two digits, the outer or lateral claw and the inner, or medial claw. In hind feet the lateral claw is larger than the medial and is the major weight-bearing surface. In front feet this is reversed, with the inner claw being the larger and weight-bearing. This is thought to be an important reason why the majority of lesions occur in the outer claw of hind feet and the inner claw of front feet.

Each claw consists of three tissue components as shown in Figure 1, namely hoof, corium and the bone.

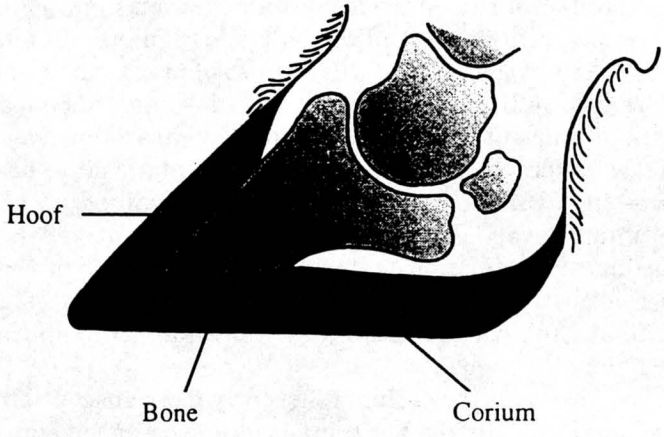

Figure 1. The three basic tissues of the foot: hoof, corium and bone.

The **hoof** can be subdivided into the wall, the white line and the sole. It provides the hard outer casing of the foot.

The **corium** ("quick") is a support tissue carrying nerves and blood vessels which transport nutrients for both hoof and bone. Pain is experienced when the corium is compressed, penetrated or infected.

The **bones** comprise the pedal bone and the navicular bone.

The wall of the hoof is produced at the hoof/skin junction, of the coronary band. It passes slowly down over the laminae at approximately 5mm per month. The sole is a separate structure, produced by the corium of the sole. Where the wall and the sole join there is a cemented junction known as the white line. Being a cemented junction this is a point of weakness and a common place for penetration by stones.

Hoof Overgrowth

Although often omitted from texts on lameness, hoof overgrowth is probably one of the most common factors leading to discomfort when walking. Overgrowth occurs primarily at the toe. Elevation of the toe destablises the foot, rotating the claw backwards and placing additional weight on the heel, as shown in Figure 2. Cows with overgrown claws have a poorer gait and walk with significant discomfort compared to well-trimmed animals. In addition, extra weightbearing on the rear edge of the pedal bone may predispose to sole ulcers. It is vital that both veterinarians and herdsmen have a good understanding of the anatomy of hoof overgrowth before embarking on hoof trimming. Hooftrimming procedures will be described.

Figure 2. Overgrowth at the toe leads to a backward rotation of the pedal bone and pinching of the corium between the pedal bone and hoof.

White Line Abscess

The white line is a cemented junction between the wall and the sole of the hoof and as such it is an inherent point of weakness. The white line can be further weakened by aspects of management, housing, feeding and particularly by calving. Once weakened, it is easy for a stone or dirt to become impacted. Growth of horn may dislodge the foreign body from the base of the foot, but it is also possible that the foreign body will penetrate to the corium. Bacteria carried in with it will multiply and it is pressure from the pus which leads to lameness. Infection escapes from the corium by taking the line of least resistance. This usually leads to a discharge through the softer horn of the heel.

Sometimes pus tracks up the wall to discharge at the coronary band. If infection accumulates in the toe, then lameness can be particularly intense because there is no route for drainage. Treatment consists of draining the abscess, removing all the under-run horn to expose the underlying corium. New horn will then grow from that corium. Ideally a block, for example a Cowslip (Giltspur Ltd.), should be applied to the sound claw to remove weightbearing from the affected claw. This will promote healing, reduce lameness and considerably enhance the welfare of the cow.

Sole Ulcers

Sole ulcers are caused by a pinching of the corium between the pedal bone above and the hard horn of the sole beneath.

Pinching of the corium produces bleeding and this blood then mixes with the horn as it is produced, to be seen on the surface of the sole some 1-2 months later. This is often referred to as bruising. Sometimes the compression and damage to the corium can be so severe that horn formation is totally disrupted. This then leads to a sole ulcer. Treatment consists of removing all underrun and damaged horn and preferably applying a block to the sound claw to minimise weightbearing on the ulcer site, thereby promoting healing.

Prevention of White Line Abscesses and Sole Ulcers

As both conditions are caused by similar factors, causes and control are dealt with together. The corium is in its most fragile state and most susceptible to bruising at the time of calving. The rings on the horns of beef cows, one for each calving, show that there is always a disruption in horn formation at parturition. It also explains why peak incidence of lameness is seen some 4-8 weeks after calving, this being the length of time it takes for the damaged horn to grow to the surface of the sole.

It is not known whether it is the process of calving itself which leads to increased fragility of the corium, or the initiation of milk production. Recent work has suggested it may be the sudden increase in milk production, leading to a massive demand for sulphur-containing amino acids, which could be a critical factor. Sulphur-containing amino acids are essential both in the construction of keratin, the main component of horn, and for milk production. Clearly at the time of calving there is a sudden repartition of nutrients towards milk production and perhaps this leads to the formation of poor quality horn. A second feature of parturition is the decrease in the rate of rumination shown by all cows. Dairy farmers will have been aware of this for many years. A cow late at night sitting chewing her cud probably will not calve that night. However, if she is not cudding and is sitting motionless, perhaps with a glazed look in her eyes, then you know that parturition is imminent. Clearly there is an increased risk of rumen acidosis with ruminal atony present. This has been linked to coriosis, i.e. increased fragility of the corium. The problem is compounded by rations which are low in long fibre. It has recently been suggested that the rate of resumption of rumen contractions immediately post-calving is considerably affected by the quantity of long fibre in the diet. Cows on high concentrate diets have a much longer period of ruminal atony and are much more susceptible to rumen acidosis than cows on high, long fibre (e.g. straw, hay and big bale silage) diets. Could this be one of the reasons why the use of precalving rolls works so well? Precalving rolls are designed to be fed with minimal or no grazing and the cows are often given straw as forage. Straw is an excellent material to ensure that rumen contractions are maintained at an optimum level. Finally, the increased fragility of the corium at parturition may simply be a feature of the rise in foetal cortisone production, as this produces a dramatic increase in circulating maternal acute phase proteins.

Not only is the corium inherently more susceptible to damage at calving, but there is often a range of additional factors superimposed on dairy cows immediately

after calving which can lead to further damage to the corium and increase the incidence of lameness. These include:

Excessive standing

Poor cubicle comfort, due to suboptimal dimensions or lack of bedding, and heifers which have not been trained to use cubicles will both lead to increased standing. Lack of loafing area and blind-ending passageways which heifers particularly feel reluctant to enter, further exacerbate the problem.

Inappropriate feeding

As discussed above, there is a marked reduction in the rate of rumination at the time of calving and consequently an increased risk of acidosis. Acidosis leads to increased fragility of the corium. Cows should therefore be fed a diet high in long fibre to stimulate rumination. Precalving concentrates should be minimal and concentrates should be built up slowly after calving, reaching a peak at no less than 2-4 weeks after calving. No more than 4.5-5.0kg of concentrate should be given at each feed and the final dietary concentrate:forage ratio should be such that it prevents acidosis. Current interest in the dietary cation-anion balance (DCAB) of the precalving diet could also influence lameness.

Management

Horn absorbs moisture from the environment and damp horn is softer than dry horn. Cows should therefore be managed and housed to keep their feet as dry as possible, something which can be quite difficult in free-stall systems. Regular scraping of the passages, for example twice daily, plus a sprinkling of lime added to the free-stall bed to aid mastitis control, both help to keep hooves dry. Cows should be handled gently: rushing them along tracks means that they are unable to avoid standing on stones and damaging their hooves. The provision of special "cow tracks", with a soft tree-bark surface, is ideal.

Interdigital Necrobacillosis

Colloquially called "foot rot", "foul" and a range of other names, the disease is a bacterial infection of the interdigital skin. The normally smooth skin surface becomes rough and broken and often pus, blood or tissue debris can be seen. Two bacteria are thought to be involved: *Bacteroides melaninogenicus* initially penetrates the interdigital skin and *Fusobacterium necrophorum* then enters to produce a necrotising infection of the dermis. Provided that antibiotic (e.g. ceftiofur) is given promptly, response to treatment is usually good. Recently a new and much more severe form of foul, sometimes known as "Super Foul", has been seen. This seems to be associated with digital dermatitis infection and occasionally with concurrent immunosuppressive diseases such as BVD. The incidence of "foul" may be reduced by the use of a disinfectant footbath.

Digital Dermatitis (Hairy Warts)

Typically, digital dermatitis is seen as a red, raw area of epidermitis, surprisingly painful to the touch, and with a characteristic foul odour. Lesions are most commonly sited on the skin just between the bulbs of the heels, and in fact the reservoir of infection is thought to persist in the interdigital pouch, that is between the claws towards the heel. Lesions may also be seen at other sites such as the skin of the interdigital cleft, at the front of the interdigital cleft and sometimes under-running the sole or even on a sole ulcer. If lesions occur on the anteriorcoronary band they can totally disrupt horn formation and lead to a vertical fissure. Many cows affected in this way never recover. Hairy warts occur in chronic, longstanding, untreated infections because epidermal proliferation is a response to the causative organism.

Digital dermatitis is caused by a spirochaete, most probably a Treponeme. Treatment of individual cases is by topical application of an antibiotic aerosol. Prevention is based on keeping the housing and general environment as clean and dry as possible and by whole herd footbaths through antibiotic solutions. Usually once a month is sufficient, with the most commonly used products being lincomycin, a combination of lincomycin and spectinomycin, erythromycin or oxytetracycline.

Interdigital Skin Hyperplasia

Also known as Tylomas or "Corns", these are large overgrowths of skin between the claws. There is a genetic predisposition, seen especially in some heavy breeds of cattle, while other cases are thought to be the result of chronic irritation of the interdigital skin, for example following a low-grade foul or digital dermatitis infection. Lameness is caused by the pinching action of the claws on the "corn" as the cow walks. Some "corns" develop a secondary digital dermatitis or foot-rot infection.

Small lesions may be treated by limited paring of the axial hoof wall, thereby removing the traumatic pinching action and allowing spontaneous resolution. More advanced lesions need to be removed surgically.

Slurry Heel

Although not in itself a cause of lameness, erosion of the heel can destabilise the hoof, causing it to rotate

backwards and thereby predisposing to other causes of lameness, especially sole ulcers. The normal smooth, soft and pliable perioplic horn of the heel becomes eroded due to excessive standing in slurry (and possibly on rough concrete) and is therefore primarily a disease of winter housed cows. A dry period at pasture or in straw yards (but preferably not in cubicles) is important to allow regeneration of the heel.

Vertical and Horizontal Hoof Fissures

These occur when there has been a disruption in horn formation. Vertical fissures are often known as sandcracks. An anterior digital dermatitis lesion is becoming an increasingly common cause. The incidence of vertical fissure in beef cows has been markedly reduced by supplementing the feed with biotin.

A total, but temporary, cessation of horn formation, for example following a severe toxic mastitis, leads to a horizontal fissure. The time when the "insult" occurred, which led to the cessation of horn formation, can be estimated from the distance of the fissure from the coronary band, since hoof wall passes down the hoof at 5mm per month.

Acknowledgments

Much of this information has been published in Cattle Lameness and Hoofcare, (Farming Press, Ipswich, Suffolk, UK and Diamond Farm Enterprises, Box 537, Alexandra Bay, NY 13607) and is reprinted with their permission. Readers should consult this book for details of hoof-trimming. Thanks go to Catherine Girdler for typing the manuscript.

Abstract

Simulated airborne spread of Aujeszky's disease and foot-and-mouth disease

J. Casal, J.M. Moreso, E. Planas-Cuchí, J. Casal
Veterinary Record (1997) **140**, 672-676

The atmospheric dispersion of virus was simulated using a computer model which had been developed for predicting the dispersion of toxic gases from chemical engineering plants. The results were compared with data from four outbreaks in which virus was believed to have been transported by air: two outbreaks of foot-and-mouth disease in the United Kingdom in 1967 and outbreaks of Aujeszky's disease in Yorkshire in 1981 to 1982 and Indiana in 1988. There was relatively good agreement with most of these data. The paper shows that the model could be useful in an emergency because the risk of virus spread could be predicted in real time.