Roger Blowey

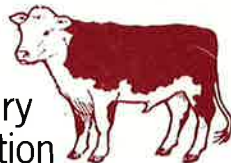# UPDATE IN CATTLE LAMENESS

Proceedings of The VIth International Symposium on Diseases of the Ruminant Digit

Schering-Plough
Animal Health

British
Cattle
Veterinary
Association

Update on Lameness in Cattle

Proceedings of VIth International Symposium

on Diseases of the Ruminant Digit

July   1990

Foreword

Lameness in cattle is a major disease problem in many parts of the world. Within intensive agricultural systems, the economic losses associated with the problem appear to be well- recognised , if poorly quantified; in a more extensive system, lameness may threaten the very life of the animal. The basic veterinary involvement in the disease remains primarily that of treatment, despite growing public pressure for a more critical role to be exercised in the welfare of cattle kept in intensive and semi-intensive systems.

The enthusiasm of Prof. Paul Greenough and Dr. David Leaver for the subject has remained undiminished and without this, the VIth International Symposium on Diseases of theRuminant Digit would never have come about. The British Cattle Veterinary Association and particularly Dr. Bob Ward, responded positively with their unique brand of organisational ability to propose that the event be held in Liverpool University.

Our sponsors, namely Mycopharm, Intervet, Rycovet and Norbrook, have generously supported the programme and I thank them. Bimeda have ensured that delegates will keep Symposium documents all together.

Without Schering-Plough Animal Health and John Millyard, in particular, you would not be reading these Proceedings now; I cannot give them too high praise for their major contribution to this event. Thank you.

To Barbara Hodgkinson (compiler) and Thomas Loughlin, Liverpool (printers), go our thanks for producing the proceedings; a wonderful team.

Richard Murray

CONTENTS

UNINVITED PAPERS

Terminology and Pathogenesis Associated with Laminitis in Cattle

David G.P.

Veterinary Investigation Centre, Kendal Road, Harlescott, Shrewsbury SY1 4HD

In his excellent thesis on bovine laminitis, Nilsson (1963) described five main categories of laminitis; parturition, metastatic, digestive, acetonaemia related and overloading/rheumatic. The thesis also suggested that laminitis predisposed the hoof to other important lameness causing lesions, particularly sole ulcer and white line disease although physical and anatomical factors are considered to be important in the epidemiology of these conditions (David,1988).

Maclean (1971) supported Nilsson's theory regarding the long term effects of laminitis. For the last fifteen years Nilsson's third category, laminitis due to digestive disturbances, has become the centrepiece of all discussion of the epidemiology and aetiology of the major causing lesions with little consideration being given to any of the other categories and other major epidemiological factors. The activities of the agricultural press resulted in lameness becoming synonymous with feeding concentrates. Laminitis caused by digestive disturbances appeared to be an idea which .the modern dairy farmer could understand and accept. Why was this theory so attractive? Firstly, it appeared to fit the evidence available at the time. True, the rise in lameness incidence in dairy herds had paralleled the increased use of concentrates, a large part of the revolution which took place in the dairy industry in the late 1960s, early 1970s, but along with this change came many others - notably herd size, building design, cow management and replacement rate. Secondly, it provided a relatively simple cause that was easily rectified. Changing a feed formulation is much easier than major structural alterations in buildings or radical changes in management. Thirdly it allowed the veterinary profession to wash it's hands of the problem by laying all the blame at the feet of greedy farmers who were feeding too much corn to their cows!

More recently Colam Ainsworth and others (1989), Mgassa and others (1984) and Bazeley and Pinsent (1984) have indicated that perhaps another of Nilsson's original categories, overloading/rheumatic factors, may be playing some part in the epidemiology of laminitis and major lameness causing lesions. Because of this, it is worth reviewing Nilsson's original categories in the light of existing evidence.

Laminitis Associated with Parturition

Nilsson (1969) considered that the majority of his cases of parturition laminitis were associated with metritis and the retention of foetal membranes, postulating the excessive production of histomine by microorganisms in the uterus. A similar mechanism was postulated for the metastatic cases which were principally related to

infectious conditions involving various septi foci. There is little or no follow up research of this although most workers would consider it unlikely that this mechanism was responsible for predisposing to the high incidence of lameness in the U.K. dairy herd.

Rowlands and others (1985) showed an association between bovine lameness and stage of lactation. Could there be another factor operating at parturition which predisposed cow's feet to laminitis? Maclean (1965) suggested that any drop in serum protein occurring at calving could predispose to oedema and be additive to any effects of vasoactive substances such as histamine. Certainly any development of oedema in the corium of hoof would lead to an impairment of circulation and subsequent hypoxic changes characteristic of laminitis. Such changes would certainly be exacerbated by factors causing physical overloading of the claw which are discussed further in the consideration of overloading/rheumatic laminitis.

Laminitis Associated with Nutritional Disturbance

Many of the major feed components have been associated with lameness particularly that caused by laminitis, sole ulcer and white line disease. The hypothetical mechanism postulated by a number of authors involves the production of vasoactive substances or endotoxins absorbed from the rumen as a direct result of abnormal fermentation and acidosis. Symptoms of acute laminitis have been associated with the feeding of rations high in easily fermentable carbohydrates and/or protein. Mouldy feed has also been suggested as a triggering factor. Nilsson (1963) reported more cases in herds fed greater amounts of concentrate rations.

It is clear that acute laminitis can be caused by nutritional factors. What is not clear is whether nutritional disturbances cause an undetectable subclinical laminitis, what factors are responsible for it's development and what proportion of a herd it may affect.

Most of the studies relating nutrition to laminitis are based on herd feeding trials. Very few controlled studies have been performed in individual animals. Mortensen and others (1986) produced very mild signs of laminitis in calves fed a diet containing 30 g/kg barley and oat;. only mild lesions associated with laminitis were seen in autopsy material despite the presence of severe lesions associated with toxaemia such as disseminated intravascular coagulation. It is worth considering that the equivalent feed for an adult 600 kg cow would be in the order of 19.5 kg. No experiments have been performed at lower feeding levels. Andersson (1981) failed to produce laminitis in fistulated cows infused with lactic acid indicating that it was not the acid itself which was a causative factor.

Many of the recent links between nutrition and lameness have centred on high forage/concentrate dry matter ratios. Livesey and Fleming (1984) demonstrated a higher incidence of sole ulcer in heifers fed a 40/60 ration compared to 50/50. Peterse and others (1984) reported a high level of sole ulcer in heifers fed 50/50 ratio compared to 80/20; they considered a 50/50 ration as high. However, what is interesting about this experiment is that the control group of heifers had a 30% incidence of sole ulcer indicating that the feed formulation was not the only factor involved. Manson and Leaver (1988) failed to demonstrate any significant difference in the incidence of clinical lameness principally caused by sole ulcer in a group of cows fed ratios of 56/44 (low concentrate) and 42/58 (high concentrate) although there was differences in locomotion score. Smit and others (1986) failed to show any significant differences in the incidence of sole ulcer or haemorrhagic sole lesions between two groups of cows fed 4 kg and 12 kg of concentrate daily. The author has investigated herds with a high incidence of sole ulcer (David 1989) and found a range of forage/concentrate dry matter ratios from 32/67 to 50/50. Bee (1986) described a high incidence of both sole ulcer and white line disease in Hampshire cattle fed a very low flat rate of concentrate (approximately 80/20 forage to concentrate dry matter ratio).

It is clear much of the published work is confusing. There does not seem to be any particular ratio which constitutes a trigger factor for problems with widespread laminitis or major lameness causing lesions. It has been suggested that the rate of increase and amount of the concentrate ration are related to lameness incidence. Peterse (1986) could demonstrate no difference between the lesion scores (solar haemorrhages thought to be indicative of subclinical laminitis) between two groups, one fed 10 kg of concentrate 3 days after calving and the other building up to 10 kg by 2 weeks after calving. Smit and others (1986) could show no correlation between the incidence of sole ulcer or haemorrhagic lesion scores and rate of increase in concentrate feeding.

The author (David, 1989) has not noted any consistency in the rate or frequency of concentrate feds and herds having a high incidence of sole ulcer. Bazeley and Pinsent (1984) showed no association between laminitis in heifers and peak concentrate feed being achieved within 8 days of calving.

High protein feeds have been suggested as a possible cause of laminitis. Bazeley and Pinsent (1984) reported an association with the feeding of a 25% protein supplement although the total protein content of the ration was not known. Recent studies by Manson and Leaver (1988) reported no significant differences in the incidence of clinical lameness between groups of cows fed rations containing 160 gm/kg and 190 gm/kg, although there were significant differences in locomotion score attributable to an increased duration of lameness in cows fed the higher protein rations.

Increasingly the evidence suggests that laminitis caused by nutritional disturbance may not be the important factor it was once considered to be.

Laminitis Associated with Overloading

Nilsson (1963) described laminitis associated with long marches and sudden introduction to hard floors. He also described a case of laminitis associated with transport which he chose to call rheumatic laminitis. Dewes (1979) described a very similar case involving the transport of heifers by road which resulted in aseptic laminitis including separation of the pedal bone from the laminae with consequent rotation. Mgassa and others (1984) described laminitis in range cattle and attributed

this to walking long distances over hard ground. Colam-Ainsworth and others (1989) related laminitis in heifers to behavioural factors confirming theories previously proposed by the author (David, 1984). Bazeley and Pinsent (1984) considered that housing and introduction to concrete floors played a part in the outbreaks of laminitis that they investigated. Dewes (1978) described discolouration and haemorrhage in the solar horn of lame cows subject to excessive trauma on poor cow tracks.

It is clear that traumatic forces both external and internal, applied to the corium of the hoof can cause laminitis, the predisposition to which would be greater if physiological effects at parturition compromised the circulation of the hoof as already suggested.

If laminitis were due to overloading/trauma it would explain the lack of correlation between solar haemorrhage and different types of concentrate feeding (Peterse 1986, Smit and others 1985). It would also fit in with many of the aspects of the epidemiology of white line disease and sole ulcer reviewed by the author (David 1989).

Conclusion

It is important for the future progress of research into bovine lameness that the term laminitis is used with more precision. The obsession with laminitis caused by nutrition disturbance, with the exclusion of other potential areas of study, has hindered the progress of research. It is becoming clear that there is more to the pathogenesis and epidemiology of laminitis and other major lameness causing lesions than what the cows have to eat. Behavioural, management and genetic factors are increasingly being shown to be of considerable importance.

References

Andersson L (1981). Acta. Vet. Scand. **22**, 140-142.

Bazeley K and Pinsent P J N (1984). Vet. Record **115**, 619-622

Bee D J (1988) Proc. 5th International Symposium on Diseases of the Ruminant Digit. Dublin.

Colam-Ainsworth, P. Lunn, G. A. Thomas, R. C. Eddy, R. G (1989). Vet. Record **125**, 573-575.

David G P (1984). Proc. BCVA 1984-**85**, p 155.

David G P (1989). Proc. Society for Veterinary Epidemiology and Preventive Medicine. Exeter, p149-158.

Dewes H F (1978). New Zealand Veterinary Journal **26**, 147-148.

Dewes H F (1979) New Zealand Veterinary Journal **27**, 45.

Livesey C T & Fleming F L (1984). Veterinary Record **114**, 21, 510-513.

Maclean C W (1971). Vet, Record **89**, 34-37.

Maclean C W (1965). Vet. Record **77**, 662-672.

Mgassa M N , Amaya-Posada G & M Hesselholt (1984). Vet. Record **115**, 413-414.

Manson F J and Leaver J D (1988). Animal Prod. **47**, 185-199.

Mortensen K, Hesselholt M and Basse G (1986). Proc. 14th World Congress on Diseases of Cattle. Dublin. 1025-1030.

Nilsson S A (1963). Acta Vet. Scand. Vol. 4, Suppl. 1.

Peterse D J, Korver S, Oldenbroek J K, Talmon F P (1984). Vet. Record **115**, 629-630.

Rowlands G J, Russell A M & Williams L A (1985). Vet. Record **117**, 576-580.

Smit H, Verbeek B, Peterse D J & Jansen J (1986). Livestock Production Science, **15**, 1-9.

Comparative Aspects of Laminitis - The Horse

S. May

Department of Veterinary Clinical Studies, University of Liverpool, 'Leahurst', Neston, S. Wirral, Cheshire.

A variety of causes of laminitis have ben described in the horse. Most of these are associated with the gastrointestinal tract, and are assumed to lead via the common pathway of endotoxaemia to the disturbances in blood flow to the feet which lead to the laminar pathology. Chief amongst this group of causes is grass laminitis in obese ponies, which tends to have a seasonal incidence with lush grass in Spring and to a lesser extent Autumn, accounting for the majority of cases. Fortunately, grass laminitis differs from all other forms of the disease in having the lowest incidence of pedal bone rotation. Even after several annual "attacks" the chronic sequelae may be minimal. This is in marked contrast to laminitis associated with grain overload, surgical gastrointestinal problems and drugs such as corticosteroids, where pedal bone rotation or shrinking is often rapid and intractable.

Although, in laminitis, there is increased blood flow in the digital artery (Robinson et al 1976), which accounts for the increased digital pulse detected in clinical cases, Hood et al (1978) have demonstrated in experimentally-induced grain over-load laminitis that there is a decrease in blood flow to the foot just prior to the onset of the clinical signs: this decrease is proportional to the severity of the lameness, and the alteration in blood flow appears to be associated with more shunting of blood from the arterial to the venous side, by-passing the capillaries. However, Trout (1987), with the aid of scintigraphic angiography, subsequently demonstrated increased total laminar blood flow in horses with the same type of experimentally-induced laminitis. Recently, Pollitt (1990) has helped to resolve this apparent contradiction with the demonstration of arteriovenous anastamoses at the level of the laminae, and suggests that it is inappropriate dilation of these which results in the paradoxical ischaemia.

If the damage caused to the laminae by the ishcaemic episode is relatively minor, and there is an early return to normal circulation, the position of the pedal bone within the foot may remain unaltered. This is the usual situation in cases of grass laminitis and there may be no chronic sequelae. However, if laminar necrosis is extensive, mechanical forces come into play. The horse's body weight is transmitted from the bony skeleton, via the laminae, to the hoof wall, and thence to the ground. Once this mechanism, which suspends the pedal bone in the foot, is disrupted, the bone starts to move downwards in the hoof (Steven 1981). If the laminae at the toe only become detached, the pedal bone rotates (Coffman et al 1970). If the laminae throughout the foot become detached, the whole pedal bone may sink. At this stage of the disease, there is often a fine balance between the destructive mechanical forces driving the pedal bone through the bottom of the foot, and the repair processes involved in the production of scar tissue which reattaches the pedal bone to the hoof wall. If the pedal bone is stabilised in a different position (rotated or sunk), this together with residual abnormalities in blood flow, which mean that the hoof grows faster at the heel than at the toe, result in the changes typical of chronic laminitis - residual lameness, long heel/curled toe, and divergent growth rings. Such animals have a permanent weakness which may result in reduced athletic potential or no athletic potential.

References

Coffman, J.R., Johnson, J.H., Finocchio, E.J. Guffy, M.M. (1970). J. Am. Vet. Med. Assoc. **156**, 219-221

Hood, D.M., Amoss, M.S., Hightower, D., McDonald, D.R., McGrath, J.P., McMullan, W.C. Scrutchfield, W.L. (1978). J. Eq. Med. Surg. **2**, 439-444.

Pollitt, C.C. Molyneux, G.S. (1990). Equine Vet. J. **22**, 79-87.

Robinson, N.E., Scott, J.B. Dabney, J.M. (1976). Am. J. Vet. Res. **37**, 1171-1176.

Steven, D. (1981). In Practice **3**(4), 22-27.

Trout, D.R. (1987) . Ph.D. Thesis. University of California, Davis.

Laminitis in Sheep and Goats

L.A. Hay

Dept. Clinical Studies, Moredun Research Institute, 408 Gilmerton Road, Edinburgh.

Diseases of the foot are an important source of lameness in small ruminants. Bacterial infections causing footrot, interdigital dermatitis and toe abscesses are the most frequently diagnosed disorders. Laminitis is a relatively uncommon but significant cause of lameness in both sheep and goats.

Epidemiology

Laminitis in these species occurs mainly as a sequel to the overfeeding of a high energy diet. This situation is likely to occur when concentrates rich in carbohydrate are fed intensively to maximise growth rate or milk production. The classes of sheep and goats most at risk are:

Sheep:

:Rams - often overfed prior to showing, selling or tupping.

: Ram lambs - performance tests to assess an individual's genetic superiority are frequently based on comparisons made between the liveweight gain of peers reared and fed under intensive conditions.

: Fattening lambs - when insufficient attention and time is given to changing the diet from predominantly roughage to one with a high proportion of grain e.g. feedlot systems in America.

: Ewes - the more prolific breeds, fed intensively indoors during late pregnancy and early lactation to maximise lamb birth weight and ewe milk production.

Goats:

Dairy breeds - often fed a wide variety of carbohydrate rich feedstuffs by overindulgent owners. Goats are more curious, adventurous and more likely to break into feed stores than sheep.

Fibre breeds - recent trends of inwintering fibre breeds will increase the risk of dietary calamities occurring.

It is difficult to assess the true incidence of laminitis in all it's forms, since subclinical laminitis, where gait and posture are normal but changes in the hooves exist, can often go undetected.

Clinical signs

The clinical signs of acute laminitis are associated with foot pain. The animal is dull, often anorexic, moderately pyrexic, disinclined to stand and remains recumbent for prolonged periods. Heat and tenderness around the coronary band and heel areas are obvious on palpation of all four feet. When standing, the back is arched with all four feet bunched together. Diarrhoea, mastitis or metritis may accompany the development of laminitis. The signs of acute laminitis may be transient and consequently pass unnoticed, particularly during the peripartum period when signs of discomfort may be attributed to events associated with parturition. Some degree of chronic laminitis develops no matter how brief the acute attack has been. Goats in particular are subject to repeated episodes of chronic low grade laminitis. Loosening and separation of the horny wall and sole from the degenerate corium beneath, downward rotation of the pedal bone, overgrowth of the heel and upturning of the toe result in the development of a "rocker" foot and chronically lame goat. Disturbances in keratin synthesis will produce laminitic rings in the horn running parallel to the coronary band.

Aetiology & Pathogenesis

Laminitis may be associated with any toxic condition such as mastitis or metritis, but it usually occurs as a sequel to ruminal acidosis which follows overfeeding on a high energy diet. Ruminal microflora change within a few hours following carbohydrate overload from predominantly gram negative organisms to predominantly gram positive lactic acid producing bacteria.

Acidic conditions arise (pH <5) when the rapid production of lactic acid overwhelms the normal mechanisms operating to preserve ruminal neutrality. Dying and disintegrating gram negative bacteria are a potential source of endotoxins which are rapidly absorbed into the bloodstream through the damaged ruminal mucosa. The resulting lactic acidaemia and endotoxaemia are thought to play a central role in initiating the generalised vascular changes which ultimately lead to a reduction in blood flow to the laminar tissues. These disturbances in the circulation of the foot are reversible initially, and probably cause the exhibited pain. Chronic hypoxia and lack of essential sulphur containing amino acids in the corium results in an insufficient nutrient supply to the keratin producing cells with subsequent synthesis of structurally incompetent keratin. The end result is the production of laminitic rings in mild cases, pedal rotation, or complete separation of the horny and sensitive laminae in very severe cases.

Pathology

The clinical and pathological manifestations of acute laminitis have been induced experimentally in lambs by the intraruminal injection of lactic acid. Within two hours of injection microscopic changes were observed in the stratum vasculosum of the coronary and laminar coria as well as the sensitive laminae. These lesions included oedema, pooling of capillary blood and lymph stasis. Two days later, the laminar region stratum vasculosum remained the site of reaction with lymphocytic

infiltration, lymphatic stasis and venous pooling. The sensitive laminae continued to have dilated capillaries, oedema and lymph stasis. Hydropic degeneration of the nuclei of the stratum germinativum was observed. By 7 days after injection all treated lambs had hydropic degeneration, pyknosis, karyorrhexis, and karyolysis of the nuclei of the stratum germinativum. Separation of the stratum corneum from the stratum germinativum was also observed.

A similar trial performed in East African goats failed to produce either clinical or pathological changes commonly associated with laminitis. When compared with cattle and sheep, it appears that goats can tolerate relatively large amounts of carbohydrate or lactic acid before developing laminitis.

Treatment

Pathological changes will occur within a few hours of the onset of clinical signs, becoming permanent within 24 hours. If irreversible damage to the laminae is to be prevented, treatment must be instituted as soon as possible and the causative or precipitating factors removed.

1. Antibiotics
   broad spectrum parenteral antibiotics are indicated in cases of metritis and mastitis and to prevent secondary infection of the inflamed laminae.
2. Anti-inflammatory analgesics:
   non steroidal anti-inflammatory agents such as flunixin meglumine or phenylbutazone daily will provide some pain relief as well as reduce the severity of pathological changes
3. Diet
   if carbohydrate overload and metabolic acidosis are suspected, remove all concentrate feed and keep on good quality roughage e.g. hay, bran mashes, until the clinical signs resolve.
4. Exercise
   restoration of normal circulation within the hooves is very important. If lameness is not too severe, gentle exercise on soft ground or deep bedding following parenteral analgesia will be beneficial.
5. Foot trimming
   corrective foot trimming may reduce discomfort and improve the gait of those with chronic laminitis. The heel and soles are trimmed in an attempt to restore a more normal pedal bone position, so that it's tip no points down at and presses onto the dropping sole.

Prevention

Laminitis is invariably a disease of management and theoretically should never occur. The feeding regime interacting with other management conditions appear to be the major factors involved. Tailor the carbohydrate content of the diet to closely match production requirements. All dietary changes should be made gradually over a period with no sudden access to highly fermentable carbohydrates. Concentrate rations should be divided and fed in a number of small feeds. Avoid strict confinement during the periparturient period and ensure gentle and hygienic assistance at birth plus parenteral long acting antibiotics if intervention has been necessary.

References
1. Morrow L.L., Tumbelson M.E., Kintner L.D., Pfander W.H., Preston R.L. (1973) Laminitis in lambs injected with lactic acid. American Journal of Veterinary Research Vol. **34**, 1305-1307.
2. Mgasa M.N. Mbassa G.K. (1988) Tolerance of goats to experimental grain engorgement and intra-ruminal lactic acid injection. Veterinary Research Communications **12**, 143-147.

Comparative Views of Laminitis: The Pig

R H C Penny

Nether End Austrey, Nr. Atherstone, Warks. CV9 3EJ

Until the paper by Nilsson (1964) from Sweden, laminitis in the pig was considered not to occur or to be extremely rare, yet laminitis in the horse had been recognised as both a common and serious condition for many years. Hinds (1829) describes the classic symptoms of "founder" in horses, but does stress (mistakenly) that the condition occurs only in the front feet.: this condition was also a case of lameness in cattle (Nilsson, 1963). Before Nilsson's report, the reason for the near absence of the condition in pigs was considered to be the ability of this animal to vomit readily, and so relieve itself of stomach overload.

Nilsson (1964) described 11 cases of laminitis seen over a 10-year period in practice. Eight pigs were females and three males; their age ranged from 10 months to four years, and all were of the Swedish Landrace breed. He attempted to classify his cases (see Table 1); the main clinical features observed were stiffness and reluctance to move or even stand up, and claw pain was evident in eight cases on palpation or the "pincers" test (Table 2). All nine animals treated showed some response to antihistamines and/or corticosteroids.Haematological examination of one case showed basophilia, reported as a sign of laminitis in cattle (Nilsson, 1963). Inflammation of the laminae, haemorrhages into claw horn, changes in claw shape and rotation of the pedal bone, reminiscent of the condition in cattle, were seen at autopsy, and histological findings were recorded.

Nilsson attempted to classify his cases, as was done in the horse, according to their aetiology: one case was considered to be due to toxaemi:, one to recent parturition: three to overfeeding over a period, rather than suddenly. He concluded that laminitis in pigs was by no means exceptional, and cases were probably being overlooked because of difficulties with the differential diagnosis.

Maclean (1968) described 24 cases of acute laminitis, all in sows between one-and-a-half and three-and-a-half years of age. No breed frequency was apparent. Seventeen cases occurred just before or just after parturition, some associated with so-called post-parturient fever, and 14 cases occurred in overweight intensively-housed sows on concrete. Signs in individual animals were not described, but the common findings were arching of the back, a reduced stride and pottering gait, and much lying down for long periods with legs extended. All four feet were invariable involved, but signs were most severe in the fore feet, and the lateral hind claws were least commonly affected. The pulse was elevated in all cases, temperature varied from 37-41.1°C. Heat in all four feet and pulsation in the digital arteries was detected in all cases; this was more difficult to feel than in laminitis cases in cattle, and extremely difficult to detect in normal sows. In all white-clawed sows, an increased pink colouration of the sensitive tissues, particularly of the lamellar area and the sole, could be seen through the overlying horn. Dark areas seen through the horn of the sole and toe prove to be haemorrhagesat autopsy. Haematological studies on 10 cases showed little abnormality that could be related to the laminitis and were similar to those reported in cattle (MacLean, 1965).

Also at autopsy haemorrhages were observed along the abaxial lamellar border and the sole/heel junction. Maclean (1968) also described the histology of the lesions; they were found to be most severe in the lateral fore and the medial hind claws and some anatomical predisposition probably existed as the larger claws were usually affected more severely. (See comment later under anatomy).

Maclean concluded that it was clear that laminitis in pigs was a common lesion resulting from two different types of insult to the corium: one toxaemic and vascular (my interpretation), the other traumatic and associated with overloading, often near to parturition (ie at the time of maximum weight). However, the histological changes were similar in both types of laminitis which indicated that dyskeratosis was a common respone. This could lead to the production of poor quality horn which was subject to infection later in life.

Bollwahn and Hertrampf (1976) from West Germany described laminitis and claw atrophy (rather third phalanx atrophy) in pigs as a sequel to haemorrhagic pododermatitis. This was associated with excessive growth of the wall and the development of corkscrew-claw. The cause was considered to be housing on sharp, abrasive floor surfaces.

Some authors have mentioned laminitis in pigs when reviewing the causes of lameness in this species (Vaughan, 1969; Wells, 1984), while others have failed to mention the condition in their text or list of 38 possible foot and limb conditions causing lameness (Smith, 1988). In a recently published Comprehensive Veterinary Dictionary, laminitis is said to be a disease of horses and rarely cattle (Blood and Studdart, 1988). Does this mean that the diagnosis of a disease similar to that seen in horses is uncommon in pigs? I have to conclude that the answer is yes! However, the explanation for these apparently conflicting opinions could be due, at least in part, to differences in terminology. Let us investigate this possibility.

Anatomy.

The claws of the pig have some similarities with, but important differences from, other cloven-footed animals such as cattle, and a brief description seems pertinent; for a more detailed description see Penny (1968).

A pig's claw is roughly wedge-shaped and has three main surfaces: an abaxial surface (wall) something like that of the ox, an axial surface which opposes its counterpart in the inter-digital cleft, and a volar (weight-bearing) surface. This last-mentioned is divided into two distinct parts; a scimitar-shaped, flat sole of hard horn which joins a softer more bulbous and elastic horn of the heels, at a distinct line. This prominent feature runs from the abaxial area of the heel, forming part of the white line,

and then turns across the volar surface to disappear into the axial groove. The heel is some two to three times the area of the sole.

The medial and lateral claws are of different shape and size, from birth, or at least very soon thereafter. The medial claw is more pointed and its tip tends to turn towards the lateral claw, and, if "pincering" occurs it is this claw that is primarily involved, particularly the medial front. The medial claw is often smaller than the lateral claw, particularly the medial hind; in some populations of Landrace it is difficult to find a pig with near-equal sized hind claws. For many years this discrepancy in claw size has been considered of genetic origin and a potential cause of lameness (Nordby, 1939).

Excess wear reduces the length of the sole, increases the angle at the toe, produces a blocky foot with a fourth surface at the heel, and causes the pig to walk on its toes. Lack of wear leads to the reverse; the sole lengthens and becomes concave, the angle with the volar surface decreases, and the pig is forced to walk on its heels. Of course there could be a possible "chicken and egg" situation here in both of these assumptions.

Bruising/Haemorrhage of Claw Horn:

In an abattoir survey of foot lesions in 3195 finishing pigs at two slaughterhouses the overall frequency of five common claw lesions was 65%. In descending order of frequency they were: heel erosion (30.7%), white-line lesion (25.5%), toe erosion (22.8%), sole erosion (17.9%) and false sand-crack (3.0%). A number of less common lesions were recorded; 25 pigs had one or more markedly overgrown claws, six had corkscrew claws, and four had bruising of the heel/sole junction (Penny and others, 1963). Maclean (1968) considered this lesion to be associated with laminitis.

In further field studies, bruising/haemorrhage at the above site, into the bulbs of the heel, and into the toe region of the sole, were found to be quite common in some units, particularly those with a high frequency of foot lesions associated with lameness (Penny and others, 1965). In two units the frequency of bruising at the heel/sole junction in batches of sucklers was 13% and nil; of weaner/growers in both units nil, and in the 53 sows in the second unit 11%. The volar surfaces of the sows' feet in the first unit were not examined. When sows with foot lesions were walked through a foot-bath and provided with extra bedding, or were turned out onto soft ground, most of the lesions resolved in six to eight weeks. It was concluded that they were caused by environmental trauma from rough and abrasive concrete floors, and new concrete with a very alkaline pH was considered particularly dangerous. Paring down hyperkeratinised horn was a beneficial procedure, but any attempt to pare the haemorrhagic lesions was strongly resisted by the sow and slowed recovery.

Reproduction of Bruising/Haemorrhage:

In attempts to confirm that poor flooring was the main cause of foot lesions, groups of pigs were housed on freshly laid rough and smooth concrete surfaces, from eight weeks to a slaughter weight of 100 kg plus. Bruising of the horn of the volar surface of the claw was more common in the pigs on rough concrete and differences were highly significant, both between treatments and between claws (Table 3). The lateral hind claw, the one least affected with laminitis according to Maclean(1968) was one most commonly involved. Bruising was one of the earliest signs of horn damage to be observed and many bruised areas of horn subsequently developed into erosive lesions. Bruising was most commonly seen on the heel-bulb towards the abaxial wall; the next most common site was the heel/sole junction, and the least common was the sole and toe.

Pincer-Claw:

Moderate and severe lesions of foot-rot were generally more common in pigs on the rough surface and one interesting observation was that pincer-claws were significantly more common in the front claws of pigs on this surface. The medial claw was most frequently affected (Table 4).

Foot-bath Treatment:

Attempts to prevent the development of foot lesions by walking pigs through a foot-bath containing copper sulphate or formalin solution were unsuccessful. However, such treatment did delay the onset of lesions and reduced their severity.

Growth Rate and Feed Conversion:

No adverse effects on either of these production measures could be demonstrated in these experiments (Wright and others, 1972).

Heel/sole Bruising in Neonates:

Observations were then made on the volar surface of the foot of piglets in the first week of life on different farrowing house floor surfaces. Very high frequencies of bruising at this site, up to 93% (Table 5) were recorded (Penny and others, 1971; Penny and Wright, 1972). These lesions could be prevented by laying better concrete, sealing the floors with latex or epoxy-resin-based paints, the use of appropriate bedding or plastic-coated wire-mesh floors. That the floor was the cause of many of the foot and skin lesions seen was conclusively proved when parts of the body (e.g. the knees and abdomen) were protected by bandages. The work of Smith and Mitchell (1976) confirmed many of these findings.

Bruising of the Abaxial Wall:

Haemorrhages into the wall are thought to be an indication of biotin deficiency in pigs (Comben, 1979), but, in a field study in one herd, no differences in frequency were observed over a 12-month period between supplemented and control sows; there was no benefit to other foot lesions. However, replacement gilts were being purchased and when the results for these were analysed separately, there were significant benefits from biotin supplementation in seven of the 44 tests. The frequency of bruising of the heel was decreased but not the frequency of wall haemorrhage or of localised lesions of laminitis in the abaxial wall (Penny and other, 1980). It was concluded that supplementary biotin helped to produce horn that was better able to withstand trauma from the environment. Many of these findings have been confirmed by other workers (viz Simmins and Brooks, 1988).

In attempts to determine what physical and biochemical changes had taken place

in the horn, groups of pigs were supplemented with biotin and others kept as controls. In the conditions of this experiment, no changes in growth rate or rate of wear of claw horn could be demonstrated. Hardness measurements were made at eight sites on the claw and biotin supplementation increased the strength of abaxial wall horn but reduced the hardness of heel-bulb samples. The wall samples were taken from an area of the claw that took a lot of weight during locomotion, whereas the heel samples were from an area that was considered to be cushioning the impact of locomotion (Webb and others, 1984).

Biochemical estimations were made on small numbers of horn samples from biotin supplemented and control animals, and the former had a higher zinc content. This is an interesting finding as supplementary zinc has been used to treat foot lesions in young cattle (Demertzis and Mills, 1973) and increasing the dietary level can help to control outbreaks of exudative epidermitis in swine.

Summary and Conclusions:

Acute laminitis in pigs, similar to the disease seen in horses, has been reported only rarely in the pig. However, bruising/haemorrhage into the heel, heel/sole junction, the sole at the toe and the abaxial wall are common. Local laminitic lesions of the abaxial wall are also common. The question is, are these lesions precipitated by vascular changes from within the claw or are they due to external trauma from the environment? I am forced to conclude the latter, although nutritional and genetic factors may render the claws more susceptible.

for research into laminitis the way forward in the pig could be a three-pronged one;.

a) more knowledge is required on the best floor surfaces for housing pigs of different ages. This work must go hand in hand with better education of pig farmers and stockmen on these matters.

b) we need more basic knowledge of claw horn production and development and any nutritional factors that may influence its production.

c) we need to know more about possible ways of making horn more resistant to the environment, and relatively simple remedial methods for improving unsatisfactory floor surfaces. The obvious danger is that the farmer and his pigs may have to live with such a floor.

Shaw (1978), in concluding a paper entitled Special Materials and Finishes for Floor Surfaces in Animal Housing said, and I quote, "Whether the average farmer will be prepared to pay much of a premium for better floors is most questionable. In the short term best results will be achieved by educating farmers in the most appropriate materials and methods for laying good quality cementitious floors at little extra cost". This statement may have been true and generally acceptable at the time but changes may well be forced on farmers in order to meet modern welfare requirements.

REFERENCES

Blood, DC and Studdart VP. (1988). Bailliere's Comprehensive Veterinary Dictionary, Bailliere Tindall, London. p520.

Bollwahn W. and Hertrampf B. (1976) Proceedings International Pig Veterinary Society Congress, Ames, Iowa, USA. Q3.

Comben, N. (1979) Personal communication.

Demertzis PN, and Mills CF. (1973) Veterinary Record 93: 219.

Hinds J (1829) Veterinary Surgery and Practice of Medicine or Farriery Taught on a New Plan. Whittaker, Treacher & Co., London. p535.

Maclean CW. (1965) Veterinary record. 77: 662.

Maclean CW. (1968) ibid 83: 71.

Nilsson SN. (1963) Acta Veterinaria Scandinavica, 4: Supplementum 1.

Nilsson SN. (1964) Nordisk Veterinaermedicin 16: 128.

Nordby JE. (1939) Journal of Heredity, 30: 307.

Penny RHC, Osborne AD and Wright AI. (1963) Veterinary Record, 75: 1225.

Penny RHC, Osborne AD, Wright AI and Stephens TK. (1965) ibid. 77: 1101.

Penny RHC. (1968) Observations of Some Conditions Affecting the Foot of the Pig. Thesis, Royal College of Veterinary Surgeons, London. p15.

Penny RHC, Edwards MJ and Mulley R. (1971) Australian Veterinary Journal, 47: 529.

Penny RHC and Wright AI. (1972) The Veterinary Annual, Eds Grunsell CSG and Hill FWG, J Wright and Sons, Bristol. p 78.

Penny RHC, Cameron RDA, Johnson S, Kenyon PJ, Smith HA, Bell AWP, Cole JPL and Taylor J (1980) Veterinary Record, 107, 350.

Shaw JDN (1978) Special Materials and Finishes for Floor Surfaces in Animal Housing, Proceedings Cement and Concrete Association Symposium, p133.

Simmins PH and Brooks PH (1988) Veterinary Record, 122: 431.

Smith WJ and Mitchell CD (1976) Farm Building Progress, 43.

Smith WJ (1988) In practice, 10: 113.

Vaughan LC (1969) British Veterinary Journal, 125: 354.

Webb, NG, Penny RHC, and Johnston AM (1984) Veterinary Record, 114: 185.

Wells GAH (1984) In Practice, 6: 43.

Wright AI, Osborne AD, Penny RHC and Gray EM (1972) Veterinary Record, 90: 93.

Table 1

Classification of Laminitis Cases

Type	No. of cases
acute	2
acute to sub-acute	1
sub-acute	6
chronic	2
total	11

Nilsson (1964)

Table 2

The Main Clinical Findings in 11 Cases of Laminitis in the Pig

No	sex	age	timing	posture/gait	arched back	claw pain	treatment	outcome	type of laminitis
1	F	2 yrs	Farrowed 1½ m	Dull, slow to stand	++	++	Antihists. laxative	Killed day2	A
2	F	10 m	Farrowed 2 days	Stiff, would stand		++ & soles	Feed cut Antihists	Recovered day 4	A
3	M	10 m	Serving Sows	Stiff for 10 days walked on knees		++ fore	Corticoids Antihists	Part recovery by day 10	SA
4	F	10 m	Farrowed 1 day	stiff , lying down 3 weeks		+	None	Killed	SA
5	F	2 yrs	To farrow in 1 week	Walked on knees slow to stand stiff	+	++	Antihists Prednisolone	Recovered day 10	A to SA
6	M	4 yrs	Serving sows	Stiff, walked on knees		+		Killed	C
7	M	3yrs	Serving sows	stiff for weeks			Sudden death		C
8	F	3yrs	2 months pregnant	stiff ; worst forelegs		+ fores pincers	Antihists Corticoids	Recovered 1 week	SA
9	F	3 yrs	2 months pregnant	stiff; worst forelegs		+ fores pincers	Antihists Corticoids	Recovered 1 week	SA
10	F	3½	-	stiff for 14 days slow to stand	+	none	Antihists	Recovered 1 week	SA
11	F	3½		stiff for 14 days slow to stand	+	none	Antihists	Recovered 1 week	SA

Laminitis: A=acute, SA=sub-acute, C=chronic.   yrs=years   m=month

Nilsson (1964)

Table 3

Bruising of the Volar Surface of the Feet

| | Number of pigs with bruising of :- | | | | | | | |
| | left front | | right front | | left hind | | right hind | |
floor surface	medial	lateral	medial	lateral	medial	lateral	medial	lateral
Rough concrete No of pigs = 48	8	15	3 **	16 **	5 **	20 **	4 ***	18 ***
Smooth concrete No of pigs = 48	2	8	0	8	0	4	0	11

** p=<0.01   *** p=<0.001
Wright and Others (1972)

Table 4

The Frequency of Pincer -claw

| | Number of pigs with Pincer-claw:- | | | | | | | |
| | left front | | right front | | left hind | | right hind | |
floor surface	medial	lateral	medial	lateral	medial	lateral	medial	lateral
Rough concrete No of pigs = 48	18	6	14	2	4	2	4	18
Smooth concrete No of pigs = 48	3	1	0	1	0	1	0	1

Wright and Others (1972)

Table 5

The Frequency of Brusing of the Claws

| | Herd 1 | | Herd 2 | |
	males	females	males	females
Number of pigs examined	126	117	165	125
Brusing front feet %	92.9	90.6	78.0	80.0
Brusing hind feet %	84.1	87.2	78.0	80.0

Penny and Wright (1972)

Observations on Management and Nutrition in a Holstein Dairy Herd Affected by Subclinical Laminitis

Jos J. Vermunt , Paul R. Greenough

Department of Veterinary Anaesthesiology, Radiology and Surgery, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N OWO

Abstract

The hind claws of ten heifers, ten second-calf cows, and ten mature cows in the University of Saskatchewan dairy herd were examined on four consecutive occasions: four and two months prior to calving, at calving, and two months after calving.

A high prevalence of haemorrhages, associated with subclinical laminitis, was observed in the sole of the claws. The lesions were most prevalent around calving time. After calving, there was a trend to rapid disappearance of haemorrhages from the claws of heifers. Such recovery was not evident in second-calf and mature cows. Haemorrhages were detected in the sole of the claws of heifers as much as four months prior to calving.

Factors associated with nutrition and management were investigated. Growth characteristics from birth to calving and management conditions during first pregnancy were studied. Rapid rearing (average daily weight gain from birth to breeding >800g) and sudden introduction, after confirmed pregnancy, into the dry group of adult, dominant cows housed on concrete in a free-stall system were factors suggested to play a role in the occurrence of haemorrhages in the claws of heifers well before calving.

Introduction

In Europe and Australasia, the economic importance of lameness in dairy cattle is now recognised to have reached a serious level, comparable to other production diseases such as mastitis and reproductive disorders (1,2). As Russell et al (3) noted, "The consistent and occasionally significant negative association between all lameness lesions and milk yield, implying that high milk yields are transmitted with low rates of lameness, may reflect some interference of lameness with lactation". A negative relationship between lameness and reproductive efficiency has been reported (4,5). The observations of Russell et al (3), combined with the observations of others (6), link laminitis to disappointing milk production in heifers. Mortensen and Hesselholt (7) were the first to suggest an epidemiological approach to laminitis, recognising the "multi-factorial" aetiology. A better understanding of the factors involved could lead to recommendations for the control of the condition, as suggested by Weaver (8).

Subclinical laminitis is believed to be the major predisposing cause of lesions such as sole ulcer and white zone lesions which can cause lameness (9,10). Subclinical laminitis has been recognised only recently (9). The characteristic features of the condition are haemorrhages in the sole, and physically softer and yellow horn (9-11). A decline in horn quality could predispose the claw to lesions that cause lameness in intensively managed dairy cattle (10-12). High-energy/low-fibre rations, and a rapid increase in the amount of concentrate fed postpartum are nutritional factors implicated in the aetiology of subclinical laminitis (9,12-14).

Haemorrhages are observed frequently in the sole of claws of heifers soon after calving (6, 10-12) and often precede the appearance of lesions that cause lameness (11). Cattle in the dairy herd at the University of Saskatchewan have a high prevalence of lameness. Sole ulcer is the most frequently diagnosed lesion in cows in this herd (unpublished observations). Soles of the claws of heifers examined at calving and shortly after calving had early signs of sole ulcer and white zone disease. An increase in the incidence of sole ulcer occurred between first and second lactation of heifers calving into this herd (15% and 37% respectively). Within an average of 4.5 months from the start of their first lactation, 34% of the cattle had been culled. Rejection on grounds of below-average production accounted for 22% of all heifers culled. The economic importance of these problems motivated this investigation.

Our objectives were to investigate the frequency distribution of haemorrhages in the soles of heifers and cows from four months prior to calving to two months after calving and to identify factors associated with nutrition and management that might influence the occurrence of these lesions.

Material and Methods

Animals, nutrition

The Greenbrae dairy herd of the University of Saskatchewan was used for this study.The herd comprised approximately 120 Holsteins (not more than 55 in milk at one time). The milk production of this herd was remarkably high, given that, over an extended period of time, the herd has suffered from a high prevalence of lameness (Table 1). The poor condition of the claws of heifers, when examined shortly after calving, had been an ongoing concern. The nutrition programme is outlined in Table 2.

Management

1) Young stock from weaning to breeding: During summer, the calves were confined to a grass paddock. During the winter, they were housed indoors in groups of 4-5 and were penned on a soft manure pack.

2) Breeding group: The heifers were transferred to a breeding group when they were 12-13 months of age. Age and body weight were used as criteria for the time of breeding, but heifers were not bred until they were at least 13 months old. This breeding group was housed in an earth paddock with an open-fronted shelter.

3) Dry group: The heifers joined the group of dry cows when diagnosed pregnant 30-60 days after breeding. This group was housed in a separate area of the dairy barn (free-stall type). Approximately two-thirds of the floor area of the alleyways along the cubicles was slatted. The remainder of the floor was solid

concrete. Rubber mats were used as bedding in the cubicles. There was access to a large earth surfaced paddock during favourable weather conditions. Two weeks before the expected calving date, cows were transferred to individual box stalls.

4) Lactating cows: They were housed in a free-stall barn with concrete alleys and automatic scrapers in the passage area along the cubicles. Rubber mats were used in the cubicles. Weather permitting, the cows were allowed to use an exercise area situated around the freestall barn.

Examination of claws

The soles of the hind claws of three groups of cows and heifers were examined on four occasions at intervals of two months starting four months prior to calving. A layer of approximately 1mm of horn was pared from the weightbearing surface to expose fresh horn. The evaluation included ten replacement heifers, ten second-calf cows and ten cows that had produced two or three calves; selection was random. At each examination, the soles of all hind claws were photographed. The photographs were evaluated according to zone of sole and severity of the haemorrhage or other lesion in each particular zone. "Zone zero" was added to the five zones of the sole described by others (15-18) (Figure 1), because severe haemorrhages and ulcers were observed in this region. Zone zero was the white zone and the weight-bearing surface of the wall at the toe. The haemorrhage observed in each zone was scored to a five point scale (0 = no visible change, 1 = faint discolouration, 2 = trace of haemorrhage, 3 = marked heamorrhage, 4 = exposed corium, presence of fresh or clotted blood, 5 = sole ulcer and associated haemorrhagic reaction). The score for each observation was geometrically adjusted to produce a severity score (i.e. 0 = 0, 1 = 1, 2 = 2, 3 = 4, 4 = 8, 5 = 16), thereby recognising the clinical importance of higher scores. The total severity score for each examination of each animal was calculated by adding the severity scores for each zone for all four claws. The "accumulated scores" were derived by summing the scores for each of the four examinations.

The heifers were weighed every four weeks. Growth curves of all heifers were drawn for the period from birth to first calving. Average daily weight gains for the period from birth to breeding were calculated. The heifers were moved in groups of 2-3 to the dry group when they were confirmed pregnant. This was their first encounter with a free-stall housing system, concrete, slatted flooring, and mature cows. No cubicle training was practised. Heifers still not using the cubicles after calving were taken out of the group and put in a conventional tie-stall for some days. They joined the lactating cows at a later time in the free-stall barn. The herdsmen were interviewed and management practices investigated. Incidents of lameness and production and culling figures were recorded.

Results

The distribution of haemorrhage severities in the three age groups differed relative to the time of calving (Figure 2). The heifers had, on average, more severe haemorrhages than did older cows four months prior to calving. In some instances, the lesions were sufficiently marked to indicate that changes had commenced well in advance of the first examination. In each of the age groups studied, the haemorrhage severities tended to be higher at calving than they were prior to calving. The sole haemorrhage severities at calving appeared to be higher in heifers than in older animals. There was a trend to rapid disappearance of haemorrhages from the claws of heifers after calving. The recovery was not evident in second-calf cows. Mature animals showed no sign of immediate recovery.

Weights at birth, average daily weight gains, and accumulated scores for sole haemorrhage are given in Table 3. Only two of the heifers had an accumulated haemorrhage score of six or less. Average weight at birth was 40.9kg. Three of the four heifers with above average weights at birth had high scores for haemorrhage. However, one heifer with an above average weight at birth had a haemorrhage score of only 6. Average daily weight gain from birth to breeding (first service) was 828g per day. Mean age breeding was 15.5 months and average body weight at breeding was 435kg. The heifers with the lowest body weights at breeding had the lowest scores for haemorrhage. The heifers with the highest scores for haemorrhage were the heaviest at their first service. Most heifers had a marked retardation in their growth rate shortly after they had been confirmed pregnant (Figures 3 & 4). For the four heifers with the most severe haemorrhages, the retarded growth was marked for a period of over 30 days. In each of these heifers the severity of the haemorrhages peaked at or shortly after calving.

Heifers were often noticed standing half-in and half-out of the cubicles for long periods of time and lying down in the alleys on the slats. Confrontation with adult and dominant cows was unavoidable and resulted frequently in accelerated rising or precipitated falling. On average, heifers spent 6.5 months in the dry group before they were moved to individual box stalls.

None of the heifers examined during the six month period of this study were severely lame. However, some heifers were noticed to have characteristic signs of laminitis, such as tender gait and arched back, shortly after calving. Five of the second-calf cows and four of the mature cows had sole ulcers at the postpartum examination.

Two of the three heifers having the most severe haemorrhages were culled for reason of unsatisfactory milk production.

Discussion

In this study, observations were limited to four examinations in the peripartum

period, during which haemorrhages in the sole have been reported previously (6,19,20). On average, heifers had relatively severe sole haemorrhages four months prior to calving. The haemorrhages disappeared from the soles of heifers towards the end of the study, two months postpartum. Haemorrhages in the soles of second-calf cows resolved relatively slowly; mature cows failed to recover at all. Peterse (12) mentioned that scarring of the corium could be an explanation for the slow recovery of older cows. Sole ulcers were not observed in the claws of the heifers but did occur in the older cows. This supports the theory that repetitive insults to the claw corium have a potent long-term effect.

The presence of haemorrhage in the sole four months prior to calving indicated that the problem must have originated at an earlier stage of pregnancy. The most common theory for the cause of laminitis involves a high-energy ration, but the ration fed to the in-calf heifers examined in this study did not appear to be high in energy.

Feeding high-energy rations to dairy cows is essential to maintain high production. The practice of feeding large amounts of concentrate in the absence of roughage (i.e. in the milking parlour), could cause a reduction in rumen pH which is believed to predispose the animal to subclinical laminitis (13,20-23). Feeding practices that may help to raise rumen pH include: grouping and feeding cows according to production level, feeding cows by electronically controlled feed dispensers, frequent trough feeding, and the use of a "complete diet" (24,25). The introduction of one of these methods may reduce the occurrence of nutritionally induced subclinical laminitis.

In addition to nutrition, management may be implicated in the occurrence of sole lesions in heifers. For example, eight of the heifers had an average daily weight gain from birth to service of 800g or more. The animal with the highest score for haemorrhage had an average daily weight gain of 950g. The heifers with the second and third highest scores for haemorrhage grew at an average daily rate of 840g and 850g, respectively. These growth rates exceed those considered by some authorities to be optimal for Holsteins (26,27). The National Research Council does not provide data on nutrient requirements for growing dairy cattle for gains exceeding 800g per day (28). With high daily weight gains (>820g / day), puberty is reached when the heifer is 10 months old (29) and has a body weight of about 275kg. Excessive weight gain in calves in the period from six months to one year (puberty) may lead to the deposition of fat in the juvenile udder, which in turn may inhibit the development of glandular tissue (29,30). Furthermore, a study by Little and Kay (31) indicates that milk yield will be significantly lower in all lactations for dairy heifers that are rapidly reared during the period from 3-10 months of age. Davey (32) came to similar conclusions. On the other hand, Donovan and Braun (33) cite authors to explain that "total lifetime milk yield and gross income will be maximised when the age at first calving is 22 to 24 months, while first lactation production will be maximised at 30 to 32 months". Producers should be encouraged to monitor the growth rates of replacement stock. Moderation in planning growth rate and age at first calving could prove cost effective.

Mortensen and Hesselholt (7) suggested an association between laminitis and disappointing milk production in heifers. Our data provide only circumstantial evidenc that there is a correlation between haemorrhage of the sole and either unsatisfactory milk yield or high daily weight gains.

Haemorrhages in the sole are considered to precede the development of sole ulcers, which occur predominantly in lateral hind claws. The circumstances associated with the appearance of sole ulcers (34) readily applied in this herd, namely:
1. Sudden introduction to concrete surfaces for the first time.
2. Increased activity and behavioural interactions following the introduction of new animals into an established dominance hierarchy (35,36).
3. Decreasing resting time due to unfamiliarity with cubicles in a free-stall system.

A form of overloading laminitis in heifers recently introduced to concrete surfaces has been described (22,37,38). The incidence of laminitis is normally higher in animals kept on slats or solid concrete than in tie stalls or straw yards (39,40). When the non-slatted area in the slatted houses is extensive, the incidence of laminitis-related lameness increases (41). These unfavourable conditions were present in the production unit studied.

Ideally, replacement heifers should be housed separately from mature and dominant cows. It is generally accepted that replacement heifers should be acclimatised to their new environment, including the type of housing and flooring, as early as possible (42). The introduction of new individuals to a group increases aggression within the group (43). Mixing heifers with dry cows as soon as they are determined to be pregnant, as practiced in this herd, adds psychosomatic stress to problems already encountered with nutrition and management. A better understanding of the behavioural needs of dairy cows in free-stall housing can be used to increase resting time, decrease activity and minimise aggression (34).

The presence of severe haemorrhages in the sole may be a good indication that unfavourable nutritional and/or management factors do exist in a herd. This finding could be associated with a disappointing level of production, high incidence of lameness, and losses from early culling. The general health and soundness of replacement heifers may be related to haemorrhage in their claws.

References

1. Whitaker D.A., Kelly J.M., Smith E.J. Incidence of lameness in dairy cows. Vet. Rec. 1983; **113**: 60-62.
2. Harris D.J., Hibburt C.D., Anderson G.A., et al. The incidence, cost and factors associated with foot lameness in dairy cattle in south-western Victoria. Aust. Vet. J. 1988; **6**:171-176.
3, Russel A.M., Bloor A.P., Davies D C. The influence of sire on lameness in cows. Proc. Vth Int. Symp. Ruminant Digit, Dublin, Ireland, 1986: 92-100.
4. Lucy S. Rowlands G. J., Russell A.M. The association between lameness and fertility in dairy cows. Vet. Rec.1986; **118**: 628-631.
5. Collick D. W., Ward W.R., Dobson H. Associations between types of lameness and fertility. Vet. Rec. 1989; **125**: 103-106.
6. Mortensen K., Hesselholt M. The effects of high concentrate diet on the digital health of dairy cows. Proc. Int. Prod. Cong. Belfast, N. Ireland 1986: 67-71.
7. Mortensen K., Hesselholt M. Laminitis in Danish dairy cattle - an epidemiological approach. Proc. IVth Int Symp. Disorders Ruminant Digit, Paris, France 1982.
8. Weaver A.D. The prevention of laminitis in dairy cattle. Bovine Pract. 1979; **14**: 70-72.
9. Peterse D.J. Nutrition as a possible factor in the pathogenesis of ulcers of the sole in cattle. Tijdschr Diergeneeskd 1979; **104**: 966-970.
10. Greenough P.R. The sub-clinical laminitis syndrome. Bovine Pract. 1985; **20**: 144-149.
11. Peterse D.J. De beoordeling van de runderklauw op basis van het optreden van zoollaesies. PhD Thesis, Rijksuniversiteit Utrecht 1980.
12. Peterse D.J. Het optren van zoollaesies bij melkvee. Tijdschr Diergeneesk 1982; **107**: 132-137.
13. Livesey C.T., Fleming F.L. Nutritional influence on laminitis, sole ulcer and bruised sole in Friesan cows. Vet. Rec. 1984; **114**: 510-512.
14. Peterse D. J., Korver S., Oldenbroek J.K., Talmon F.P. Relationship between levels of concentrate feeding and incidence of sole ulcers in dairy cattle. Vet. Rec. 1984; 115: 629-630.
15. Russell A.M., Shaw S.R., Weaver A.D. The Compton lameness survey 1977: A preliminary report. A.R.C. Institute for Research on Animal Diseases, Compton, Newbury, Berkshire, U.K.
16. Greenough P.R. The nomenclature of anatomical features of the bovine digits. Report IInd Symp. Bov. Digital Dis., Skara, Sweden, 1978.
17. Weaver A.D., Andersson L. De Laistre Banting A., et al. Review of disorders of the ruminant digit with proposals for anatomical and pathological terminology and recording. Vet. Rec. 1981; **108**: 117-120.
18. Mills L.L., Leach D.H., Smart M.E., Greenough P.R. A system for the recording of clinical data as an aid in the diagnosis of bovine clinical disease. Can. Vet. J. 1987; **8**: 293-300.
19. Nilsson S.A. Clinical, morphological and experimental studies of laminitis in cattle. Acta. Vet. Scand. (Supp) 1963 ;**4** : 124-139
20. Peterse D.J., Van Vuuren A.M., Ossent P. The effects of daily concentrate increase on the incidence of sole lesions in cattle. Proc. Vth Symp. Disorders Ruminant Digit, Dublin, Ireland 1986: 39-46
21. Peterse D.J., Van Vuuren A.M. The influence of a slow or rapid concentrate increase on the incidence of foot lesions in freshly calved heifers. Proc. EAAP Cong., the Hague, Netherlands 1984.
22. Bazeley K, Pinsent P.J.N. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Vet. Rec. 1984; **115**: 619-622.
23. Moser E.A., Divers T.J. Laminitis and decreased milk production in first-lactation cows improperly fed a dairy ration. JAVMA 1987; 190: 1575-1576.
24. Owen J.B. Complete-diet feeding of dairy cows. In: Haresign W., Cole D.J.A., eds. Recent Developments in Ruminant Nutrition. Toronto: Butterworths, 1981: 312-324.
25. Broster W.H., Sutton J.A. Concentrate: forage ratios for high-yielding dairy cows. In: Haresign W.,Cole D.J.A.,eds. Recent Developments in Ruminant Nutrition. Toronto: Butterworths 1981: 325-352.
26. Agriculture Canada. Dairy husbandry in cattle, Agriculture Canada publication no. 1439/E. Table A-6. 1981:89.
27. Heinrichs A.J. Feeding the dairy heifer. University Park, PA: Pennsylvania State University Extension Service.
28. National Research Council. Nutrient requirements of dairy cattle, 6th ed. National Academy of Sciences, Washington DC table 1. 1988.
29. Swanson E.W. Future research on problems of increasing meat production by early calving. In: Tayler J.C., ed. The early Calving of Heifers and its Impact on Beef Production. Brussels: Commission of the European Communities, 1975: 281-288.
30. Jurgens M.H. Animal Feeding and Nutrition. 5th ed. Dubge, Iowa: Kendall/Hunt Publishing Co., 1982: 320-323.
31. Little W. Kay R.M. The effects of rapid rearing and early calving on the subsequent performance of dairy heifers. Anim. Prod. 1979; **29**:131-142.
32. Davey A.W.F. Feeding and responses of young stock. Proceedings of a course in dairy cattle medicine 1983. Foundation for continuing education, Massey University, Palmerston North, New Zealand. 97-101.
33. Donovan G.A., Braun R.K. Evaluation of dairy heifer replacement-rearing programs. Compend Contin. Educ. Pract. Vet 1987; **4**: F133-F139.
34. David G. The influence of animal building interaction on the incidence of lameness in dairy cattle. Proc. Vth Int. Symp. Disorders Ruminant Digit, Dublin, Ireland 1986:79-86.

i. Kondo S. Hurnik J.F. How can you tell if your cows are upset? Highlights 1988;**11**:3-5.

. Potter M.J., Broom D.M. The behaviour and welfare of cows in relation to cubicle house design. In: Wierenga H.K., Peterse D.J., eds. Cattle Housing Systems and Behaviour. Boston: Martinus Nijhoff Publishers, 1987: 129-147.

. Nilsson S.A. Recent opinions about cause of ulceration of the hoof in cattle. Nord. Vet. Med 1966; **18**: 241-244.

38. Colam-Ainsworth P., Lunn G.A., Thomas R.C., Eddy R.G. Behaviour of cows in cubicles and its possible relationship with laminitis in replacement dairy heifers. Vet. Rec. 1989; **125**: 573-575.

39. Brochart M. Foot lameness of the cow, a multifactorial disease. In: Wierenga H.K., Peterse D.J., eds. Cattle Housing Systems, Lameness and Behaviour. Boston: Matinus Nijhoff Publishers 1987: 159-165.

40. Russell A.M., Rowlands G.J., Shaw S.R., Weaver A.D. Survey of lameness in British dairy cattle. Vet. Rec. 1982: **111**: 155-160.

41. Murphy P.A., Hannan J., Monaghan M. A survey of lameness in beef cattle housed on slats and on straw. In: Wierenga H.K., Peterse D.J., eds. Cattle Housing Systems, Lameness and Behaviour. Boston: Martinus Nijhoff Publishers 1987: 67-72.

42. Schlichting M.C. Adaption of cattle to different floor types. In: Wierenga H.K., Peterse D.J., eds. Cattle Housing Systems and Behaviour. Boston: Martinus Nijhoff Publishers 1987:87-97.

43. Hafez E.S.E., Bouissou M.F. The behaviour of cattle. In: Hafez E.S.E., ed. The Behaviour of Domestic Animals, 3rd ed. London: Balliere Tindall 1975: 203-245.

Table 1.
Percentages of cows lame from determined causes of claw lameness other than interdigital phlegmon, and 305 days production records of the Greenbrae dairy herd, Department of Animal and Poultry Science, University of Saskatchewan.

Percentage of milking herd lame					Production records Herd averages		
Year	Sole ulcer	White zone lesions	Clinical laminitis	All causes	Milk (kg)	Fat (kg)	Comp. BCA[a]
1986	30	2	5	37	8,762	314	194.0
1987	13	0	4	17	8,847	327	197.5
1988	27	8	5	40	8,743	331	197.0

[a]The composite breed class average (BCA) index corrects records of milk, fat and protein for the effects of age at calving (24).

Table 2.
Nutrition programme of the Greenbrae dairy herd of the University of Saskatchewan for 1985 - 1987.

Animal group	Forage Type	Forage quantity kg/day	Concentrate Type	Amount kg/day	TDN[a] %	CP[b] %
Birth-90kg	Alfalfa hay	ad lib.	calf starter	1.5	72.0	17.5
90-120kg	"	"	grower	2.0	71.5	15.0
160-275kg	"	"	"	2.5	"	"
275kg to conception	"	"	"	2.0	"	"
conception to calving	barley silage + alfalfa	19 / 5	dry cow ration	1.0	n/a	n/a
Lactating <200 days	barley silage + alfalfa	to 1.7% av. body weight	dairy ration	8.0[c]	70.5	18.5
Lactating >200days	"	"	"	4.0[c]	"	"

[a] TDN = total digestible nutrients.

[b] CP = crude protein.

[c] Lactating cows received additional concentrate in the milking parlour of up to 8.0 kg/daily according to NRC feeding guide (26).

Table 3.
Weights at birth, average daily weight gains from birth to breeding, accumulated haemorrhage scores, and first lactation, 305 days milk production (1986/1987) for the heifers.

Heifer no	Birth weight (kg)	ADG[a] (g) birth to breeding	Accumulated haemorrhage score[b]	Production to 305 days during first lactation (kg)
420	48.1	950	88	9,583
419[c]	44.5	850	50	
422[d]	42.6	840	81	7,130
418[e]	36.3	830	28	
414	37.2	830	28	7,530
413	36.3	820	44	7,816
417	41.7	810	20	7,561
421	39.0	800	6	7,517
416	39.0	790	28	7,135
415	44.5	760	6	7,639

[a] ADG = average daily weight gain.

[b] Accumulated haemorrhage score = the total of all scores for all six zones of the "weight-bearing" surface of all four hind claws for all four examinations.

[c] Culled for below herd average production in first lactation.

[d] Culled in second lactation for below herd average production.

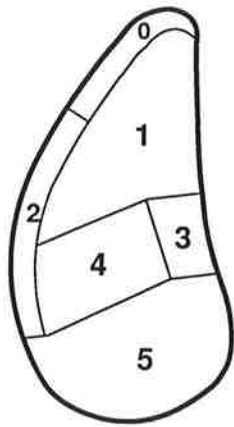[e] Culled in first lactation for severe teat trauma.

Figure 1
Zones of the sole amended to include zone 0.
zone 0 = white zone at the toe
zone 1 = angle of the sole
zone 2 = abaxial white zone
zone 3 = axial groove
zone 4 = junction of sole and bulb
zone 5 = bulb

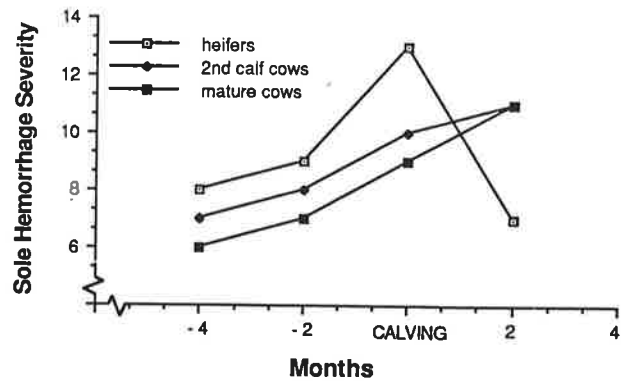**Figure 2**
Mean hemorrhage severities for all six zones of all four hind claws in the three groups of animals studied.
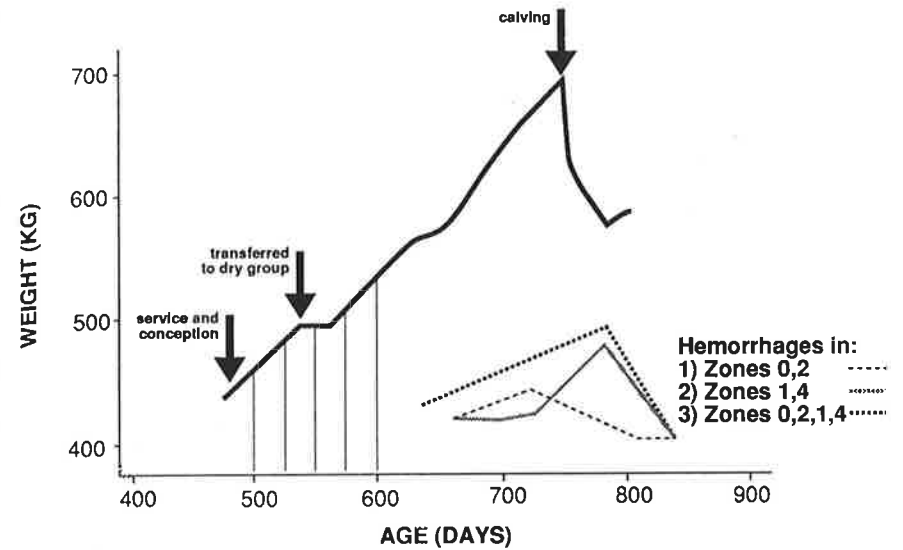

Figure 3
The typical growth pattern from just before first service to calving in the heifers studied.
Superimposed on the graph are the hemorrhage scores for the zones of the soles of the heifers.
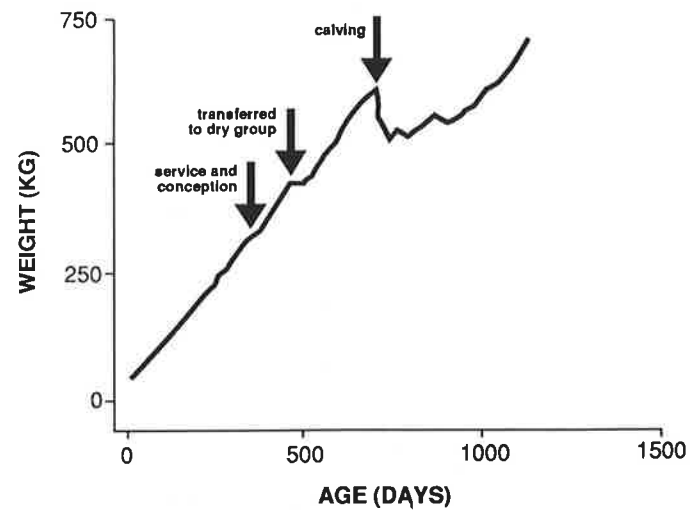

Figure 4
Typical growth pattern from birth to calving in the heifers studied.

Management and Rearing Factors Associated with Subclinical Laminitis in Dairy Heifers

Jos. J. Vermunt

Department of Veterinary Anaesthesiology, Radiology, and Surgery, Western College of Veterinary Medicine, University of Saskatchewan Saskatoon, Sask. S7N OWO , Canada

Introduction

The economic importance and animal welfare implications of lameness in cattle are substantial. The growing awareness of these facts stimulates the research in this field. In the dairy industry, the economic significance of a disorder is often determined by the percentage of animals which are culled for that particular condition. The significance of lameness tends to be underestimated because culling rates of 1.5-2% are relatively low when compared to culling figures for production, reproduction, and udder health (1,2). The economic importance of lameness is better illustrated by the percentage of cows that go lame or are treated for lameness each year; figures of approximately 25% have been reported (1,3). To assess accurately the economic losses, caused by lameness, is impossible when expressed in total cost. In addition to the costs of treatment, milk withdrawal, and extra labour, intangible losses such as decreased reproductive efficiency, milk production, and bodyweight, premature culling, and replacement costs also occur. An estimation of up to £12 per cow per year has been made (1).

A shift in the causes of lameness and understanding of those causes has occurred over the past two decades. Management, housing, and feeding systems have changed to accommodate the increasing herd size and production potential of the modern dairy cow. These changes have been accompanied by an increase in the incidence of lameness in cattle (3,4,5).

(Subclinical) laminitis plays an important role in the etiology of bovine digital disease and is regarded as a predisposing factor in lameness caused by claw disorders (6-10). Laminitis is a multifactorial problem with nutrition, management, and genetic predisposition playing integrated roles (11, 12). Season, age of the animal, environmental factors, stage of lactation, and claw conformation all contribute to the incidence of laminitis.

For a better understanding of claw problems, characteristics of claw diseases and claw quality have to be defined. Most studies on disorders and characteristics of the bovine claw have been carried out on mature animals and slaughterhouse material. Dewes (13) reported that lameness was rare in pre-calving heifers. Peterse (8, 14) used young animals in his studies and noticed few claw lesions prior to first calving. In a recent study in the United Kingdom, sole haemorrhages associated with subclinical

laminitis were found in claws of heifers as young as 5 months (15). Damage to the claws of young cattle may already occur before they reach maturity. Therefore, future research should be directed towards the study of the pathogenesis of laminitis in this group of animals.

In studies at the University of Saskatchewan, lesions were found in the soles of young heifers shortly after they were bred. The lesions increased in severity several months prior to calving and peaked at calving (9, 16). These observations motivated an extensive study to investigate the effect of management and rearing on claw characteristics and the occurrence of lesions in the claws of dairy heifers.

A brief summary of the materials and methods of this study will be given here as this work is the subject of a MSc thesis to be published shortly under the title: "Morphological and physical characteristics and lesions in the claws of dairy heifers raised under two management systems".

Materials and methods

The study was conducted from January 1989 to April 1990 inclusive.

Animals

The dairy heifer replacements of the Greenbrae dairy herd, located on campus of the University of Saskatchewan, were used for this study.

Nutrition

The nutrition program of the dairy herd was similar to the program outlined in the paper "Observations on management...." presented in these proceedings (13) with the following exceptions:

a) The feeding of additional concentrate (dairy ration) in the milking parlor had been abandoned. This concentrate was now fed through computerized feeding stalls. Cows had access to the computer feeders until 90-120 days of lactation, depending on the availability of transponders. At each milking, cows received 0.5 kg of fresh cow concentrate as a top dressing in the parlor. Some high producers might have had a total concentrate intake greater than 16 kg per day, depending upon the amount of mixed ration consumed in the free-stall.

b) As from mid August 1989, the amount of alfalfa hay fed to the confirmed in-calf heifers, dry cows and milking cows was increased to 6 kg / head / day because of high nitrate levels in the new silage.

c) Percentages of Total Digestible Nutrients (TDN) and Crude Protein (CP) varied slightly depending on the feed composition and ingredients used.

Management

Management of young stock was identical to the management described in "Observations on management....." (13)

Study groups:

Two management groups were established. Whenconfirmed to be pregnant, heifers were allocated randomly to one of the groups as they became available until a total of 15 heifers had been allotted to each group.

Heifers in Group 1 ("dry") joined the animals in the dry group and were managed and fed as described before (13). This group was housed in a separate area of the free-stall barn and separated from the milking herd by gates across the passageways. The design of the cubicle partitions was identical for lactating cows and dry stock. The effective length of the cubicle bases measured 2.5 m, whereas the clear width varied from 0.93 - 1.22 m. Rubber mats, together with a limited amount of cut straw during the winter months only, were used as bedding in the cubicles. Approximately two-thirds of the floor area of the alleyways along the cubicles was slatted. The width of the slats varied from 12.5 - 20.5 cm. The gap between the slats measured from 40 - 50 mm. The edges of many of the concrete slats were jagged and rough. The remainder of the floor was solid concrete. During favourable weather conditions, animals had access to a large earth grass paddock. On occasion, the surface of this outside area was very rough (e.g. after heavy rain, during spring thaw).

Heifers in Group 2 ("outside") were housed together for the remainder of their pregnancy and maintained outdoors. They received the same nutrition on a per head basis as animals in the dry group, but were managed differently.

No mature, dry cows were present in this group and the heifers were confined to an earth and gravel surfaced corral. Although the group composition changed, all entrants to this group were familiar with existing group members because they had been maintained together in the breeding group. Straw was used as bedding in an open-fronted shelter. Adequate feed bunk space was available. In the winter, the ground was frozen, icy, and rough in patches. The location of the outside group and the number of heifers in this group were different from the breeding group. However, the environmental variables such as ground surface and underfoot conditions, relative size, shelter, and climate were indistinguishable one from the other.

Barley silage with high nitrate levels was fed to all heifers in both groups during the period from August 1st of 1989 to April 30th 1990. The range of the nitrate level in the silage was from 0.54 - 1.91% (mean 1.16%) on a DM basis.

Heifers from both groups were transferred to individual box stalls 14 days prior to their expected calving date. From then on, nutrition and management were as described for lactating cows (13). The milking herd was divided into a high and a regular group according to level of production and stage of lactation and was housed in a free-stall system with tubular steel cubicle divisions and grooved, concrete alleys. Cubicles were arranged in two parallel lines which extended along the long axis of the barn. Dimensions of the cubicle bases differed between the two production groups; the effective length was in all cases 2.5 m, whereas the clear width was 1.10 m and 1.22 m for the regular and high group, respectively. Rubber mats were used as bedding in the cubicles. During the winter, a limited amount of chopped straw (0.5 bale per 40 cows per day) was put on top of the mats. The feed bunk was situated centrally along the long axis of the barn. The passageways separating the cubicles from the feed bunk were 2.5 m wide and were cleaned every hour with automatic scrapers. There was always sufficient cubicle and feeding space for all cattle to rest and feed together. Weather permitting, cows were allowed to use a dirt surfaced exercise area situated around the barn. Other wise, the animals were confined totally on concrete.

Evaluation of claw characteristics

Heifers in the study groups were examined routinely every 4 weeks from approximately 12 months of age to at least two months after calving. Partial records were collected when heifers were due to calve after April 30th 1990.

Data were collected and analyzed for the following:

a) Rates of horn growth and wear of the dorsal and abaxial wall of the hind lateral claw.

b) Hardness of claw horn measured at the apex of the sole and abaxial wall of the hind lateral claw.

c) Claw conformation (dorsal angle, length of the dorsal border, heel height, toe/heel ratio, and claw length of hind and front lateral claws, and width of both lateral and medial claws of hind and front claws).

d) Sole haemorrhages, interdigital dermatitis, and heel horn erosion in all eight claws.

In addition, all claws of 10 calves were trimmed and examined once for lesions at approximately 6 - 7 months of age.

Lesion scoring and evaluation

The sole of all eight claws of each animal were lightly trimmed at each examination. A sliver of horn was pared from the weight-bearing surface to expose fresh horn. Photographs were taken of the trimmed plantar / palmar aspect of the claws. The presence of interdigital dermatitis and heel horn erosion was determined by gross examination of the plantar/ palmar area of the interdigital skin and the bulb area of all claws and recorded separately for hind and front claws.

The severity of interdigital dermatitis lesions was rated on a scale of 0 to 3+ :0 = no signs of interdigital dermatitis: 1+ = slight swelling of the skin with a rough, thickened, and moist appearance;:2+ = swelling of the skin with greyish exudate and signs of hyperkeratosis; dermis still intact: 3+ = dermis no longer intact; moist eczema and hyperkeratotic lesions.

Heel horn erosion was also rated on a scale of 0 to 3+ :0 = no defects: 1+ = slight pock marking and / or superficial horn defects in the axial surface of the bulb: 2+ = some big fissures in the horn of the bulb and / or sole, not extending to the corium: 3+=disappearance of heel horn and / or deep defects in the horn of bulb or sole, extending to the corium.

Photographs were used for evaluation of haemorrhages in the sole. Lesions were localized as to zone of the sole, numbered 0 through 5, and graded as to severity (9, 13). Zone 0 was amended to include the axial white zone at the toe area. Lesions

graded 1 to 3 consisted of blood stained horn, sometimes of abnormal consistency but in all cases the abnormal horn could be pared to expose normal horn beneath. A sole lesion was defined as a sole ulcer if the corium was exposed or if, following the removal of overlying horn, the lesion was continuous with the corium and normal horn had been replaced by soft haemorrhagic material. The severity grade was used in the calculation of the "severity score". After geometrical adjustment, the severity score represented twice the severity of the previous level. The reason for this adjusting or weighting was to recognize the clinical significance of severe lesions, indicating major tissue damage. Lesions with lower scores could be either transitory or reversible.

Animal performance

Average daily weight gains during the periods from birth to breeding, from birth to calving, and from breeding to calving were calculated.

Also, the amount of precipitation (rainfall and snowfall) between examinations was recorded.

Results

Only preliminary results are available at present and are not reported in full. The final results will be presented in detail elsewhere.

References
1. Whitaker DA, Kelly, JM, Smith, EJ.(1983) Incidence of lameness in dairy cows. Veterinary Record ; **113**: 60 - 62.
2. Dijkhuizen AA. (1983) Economic aspects of diseases and disease control among dairy cattle. PhD. Thesis, Rijksuniversiteit Utrecht .
3. Arkins S. (1981) Lameness in dairy cows. Part I & II. Irish Vet J ; **35:** 135 - 140 & 163 - 170.
4. Eddy RG, Scott CP. (1980) Some observations on the incidence of lameness in dairy Verinary Record **113**: 140 - 144.
5. Russell AM, Rowlands GJ, Shaw SR, Weaver AD. (1982) Survey of lameness in British dairy cattle. Veterinary Record; **111**: 155 - 160.
6. Nilsson SA. (1963) Clinical, morphological and experimental studies of laminitis in cattle. Acta Vet Scand (Suppl) **4**: 9 - 304.
7. Maclean CW. (1971) The long-term effects of laminitis in dairy cows. Veterinary Record ; **89**: 34 - 37.
8. Peterse DJ. (1979) Nutrition as a possible factor in the pathogenesis of ulcers of the sole in cattle. Tijdschr Diergeneesk ; **104**: 966 - 970.
9. Greenough PR Vermunt JJ. (1990) Evaluating subclinical laminitis and associated lesions in dairy cattle. Proceedings VIth International Symposium on Disorders Ruminant Digit, Liverpool, England,.
10. Smart M. E. (1986) Relationship of sub-clinical laminitis and nutrition in dairy cattle: a Canadian experience. Proceedings Vth International Symposium on Disorders Ruminant Digit, Dublin, Ireland, : 51 - 62.
11. Bazeley K, Pinsent PJN. (1984) Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Veterinary Record; **115**: 619 - 622
12. Colam-Ainsworth P, Lunn GA, Thomas RC, Eddy RG. (1989) Behaviour of cows in cubicles and its possible relationship with laminitis in replacement dairy heifers. Veterinary Record ; **125**: 573 - 575.
13. Dewes HF. (1978) Some aspects of lameness in dairy herds. New Zealand Veterinary Journal ; **26**: 147 - 148 & 157 - 159.
14. Peterse DJ. (1980) De beoordeling van de runderklauw op basis van het optreden van zoollaesies. PhD Thesis, Rijksuniversiteit Utrecht, .
15. Bradly HK, Shannon D, Neilson DR.(1989) Subclinical laminitis in dairy heifers. Veterinary Record **125**: 177 - 179.
16. Vermunt JJ, Greenough PR. (1990) Observations on management and nutrition in a Holstein dairy herd affected by subclinical laminitis. Proceedings VIth International Symposium Disorders Ruminant Digit, Liverpool, England, 1990.

Applied Techniques For Diagnosis of Lameness In Cattle

U. Bargai

Koret School of Veterinary Medicine, Hebrew University of Jerusalem, P.O. Box 12, Rehovot, Israel

Introduction

Bovine lameness is of important economic consequence to every cattle owner. Loss of milk yield, loss of weight and early culling are the major results of lameness. Unlike lameness in companion animals or in the horse, cases of bovine lameness are brought to medical attention quite late in the course of the disease process. Due to the nature of the cattle industry - dairy or beef - a lame cow or heifer will only be noticed when the clinical condition becomes very obvious to the herdsman. At that stage, the process responsible for the lameness is usually quite advanced. Because of this, early and accurate diagnosis is vital in cases of bovine lameness. The chances of restoring the productivity of the animal, its milk yield or weight gain, depend largely on rapid medical or surgical treatment, the course of which relates directly to accurate diagnosis. In addition to the clinical examination of the affected leg, two major ancillary means of diagnosis should be used by the attending veterinarian in order to achieve a definite diagnosis. These are synovial fluid examination and radiology.

A) Synovial fluid examination.

Synovial fluid collection is a very practical technique accessible to every practitioner because it does not require sophisticated equipment; examination is indicated in all cases where joint swelling or tendon sheath enlargement seem to be the origin of the pain.

The physical qualities of the fluid in various conditions of joint involvement are highly correlated with the nature of the lesion. Changes in colour, turbidity, clotting time, and contents could be well evaluated at the cow's side. With the help of the synovial evaluation one can safely differentiate between septic arthritis, osteoarthritis or joint traumatic effusion. Additional examination of the synovial fluid in the laboratory can determine cell count, protein concentration, and the mucin content, all of which are sensitive criteria for inflammatory processes.

Needless to say that clipping, shaving and sterile equipment is essential whenever synovial fluid is collected, if one wants to prevent external contamination of the joint and the sample.

However, synovial fluid is not able to assist the practitioner in establishing a diagnosis in most cases of lameness. Lameness caused by septic arthritis, periostitis, degenerative joint disease, fractures or sequestra are more common than joint affections; these conditions can only be accurately diagnosed by other ancillary diagnostic means - radiology.

B) The use of radiology in bovine lameness.

i) **Foot:**
Foot lesions are usually clinically similar, is swollen, painful and hot. Only radiology can differentiate between digital cushion abscess, early necrobacillosis (or paranychia as it may be called), septic arthritis of the distal phalangeal joint, or fracture of second or third phalanx
In order to establish drainage to a phalangeal septic joint, several surgical techniques have been described, none of which can be applied if one is unable to determine which joint is infected, or if the infection has spread to more than one joint. Only radiology can determine the level at which drainage should be established, or whether amputation should be resorted to.
Needless to say that foreign objects often lodge in the feet of cattle. Nails, screws, telephone wires in the feet of beef cattle may all be buried in the granulation tissue of a chronic inflammatory lesion and cannot be traced without radiology.

ii) **Fetlock:**
In the fetlock region, fractures of the first phalanx and growth-plate trauma are often similar in clinical appearance. All kinds of Salter-Harris epiphyseal fractures occur in the legs of young cattle at the fetlock. Only radiology can differentiate between septic arthritis of the fetlock joint and any of the epiphyseal fractures. Presence or absence of osteomyelitis in the adjacent bones affects the prognosis and the course of treatment, and this also can only be determined by radiology.

iii) **Metacarpus / metatarsus:**
The metatarsal and metacarpal bones are the most common sites of sequestra in cattle, due to the vulnerability of these bones to trauma and to the lack of muscule surrounding them. Only surgical removal of these sequestra will result in healing. Radiology is necessary to establish the fact that the sequestrum is the source of the fistula and to ascertain where the sequestrum is located in the bone.

iv) **Tarsus:**
Tarsal lesions are quite common in dairy cattle when the animals are housed on concrete floors. These can be either infectious or degenerative, and each type must be treated differently. Diagnosis of the infected, swollen tarsus must be differentiated to either articular or peri-articular tarsitis, each of which should be treated differently.

Beef bulls often fracture their tarsi. Radiology of the tarsus will differentiate between the various types of fracture and will be able to determine between those cases which have a guarded prognosis and those which have a terminal prognosis.

Dairy bulls suffer from degenerative processes in the tarsus more than in any other joint of the animal body. Only radiology can determine the degree of the change and the prognosis for the bull.

v) **Stifle:**

The stifle joint, the largest joint of the body, is the site of one of the most common causes for lameness in dairy cattle. Various conditions affect the stifle: cruciate ligament rupture, epiphysiolysis of the tibial crest in young cattle, septic tarsitis, and tarsal synovitis are just a few examples of such processes. In adult cattle degenerative joint disease may be incapacitating and radiology will establish the accurate scientific basis for deciding the prognosis for the cow, and whether to reinseminate her or not.

vi) **Pelvis and spine:**

Last, but not least, is lameness originating in the pelvis or spine. In the past these anatomical sites required very powerful X-ray equipment available only in veterinary hospitals. Modern mobile condenser-discharge X-ray machines, coupled with the new rare-earth intensifying screens, are able to produce diagnostic radiographs of these anatomical sites even on the farm. Conditions such as hip dysplasia, rupture of ligamentum teres, coxofemoral luxation or sub-luxation, spinal abscess or even spinal disc disease can now be diagnosed on the farm by radiology.

Final remarks

It is safe to state that radiology is indispensable for accurate and prompt diagnosis of bovine lameness. Radiology is within the reach of practitioners and group practices, and its use should be a routine procedure in the examination of most bovine lameness.

The various lesions, their clinical appearance and radiographic findings will be demonstrated at the lecture with the use of coloured slides.

Evaluation of Subclinical Laminitis and Associated Lesions in Dairy Cattle

Paul R. Greenough, Jos J. Vermunt

Department of Veterinary Anesthesiology, Radiology and Surgery, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan S7N OWO

Abstract

A high prevalence of haemorrhages, associated with subclinical laminitis, was observed in the soles of the hind claws of a herd of 120 dairy cattle. In order to objectively measure the severity of haemorrhages in the sole which occur during the peripartum period, a scoring system was developed and used to study the hind claws of this dairy herd. The soles of the claws of a sample of the herd were examined on four consecutive occasions, at two month intervals starting four months pre-partum. The lesions observed in cows and heifers were most prevalent around calving time.

The different scores of sole haemorrhage are demonstrated and linked with sole ulcer (pododermatitis circumscripta), toe ulcer, white zone lesions, and heel erosion.

Introduction

Haemorrhages in the sole are believed to be an important sign of laminitis (1-3). Subclinical laminitis in turn is believed to be the major predisposing cause of lesions such as sole ulcer and white zone lesions which can cause lameness (4-6). Haemorrhage is also associated with false sole and certain types of heel erosion. Sole haemorrhage, therefore, can be considered to be an important stage of (subclinical) laminitis, indicating claw disease.

Haemorrhage occurs at the level of the papillary pegs; red blood cells pass into the tubules of the claw horn causing the horn to be stained from pink to dark blue. The staining may be circumscribed or diffuse and may have a solid or striated appearance. The yellow discolouration of the sole or white zone that is often observed may result from the extravasation of serum from damaged vessels (7, 8). Both signs are regarded as characteristic signs of subclinical laminitis and are frequently associated with softening of the horn (4, 5, 8, 9).

Peterse stated that haemorrhages are frequently seen after calving and precede the appearance of lesions that cause lameness (8). Mortensen and Hesselholt confirmed these observations and concluded that an epidemiological approach to the control of subclincial laminitis is appropriate (10, 11). In Europe, the economic importance of lameness is now recognized to have reached a serious level. One worker estimated that the average annual financial loss due to claw problems in the United Kingdom then amounted to {£1175 - $2,000 (Canadian)} for every 100 cows (12). Other workers in the U.K. have estimated that lameness costs £15,000,000 annually

(13, 14). Digital disease in dairy cattle in Quebec is estimated to cost $10,000,000 per annum (15).

A method is required to measure, objectively, the severity of sole haemorrhages. Such measurements would enable a practitioner to assess the susceptibility of the individual animal to future lameness or to evaluate the severity of subclinical laminitis in a herd of animals. Our objective in the present study was to develop and validate a system for objectively evaluating the severity of sole haemorrhages and ulcers as observed in 30 dairy cattle. We investigated the frequency distribution of sole haemorrhages during the period from four months prior to calving until two months after calving. Differences between first calf heifers and mature cows were also examined.

Materials and Methods

Animals

The Greenbrae dairy herd located on campus of the University of Saskatchewan was used for this study. The herd comprised approximately 120 registered Holsteins (not more than 55 in milk at one time). All animals were raised on the farm. Over an extended period of time, this herd suffered from a high prevalence of lameness; sole ulcers being particularly troublesome (Table 1). A further concern has been the poor condition of the claws of heifers when examined shortly after calving.

Table 1:
Percentages of cows lame from determined causes other than foot rot (Phlegmona interdigitalis), in the Greenbrae dairy herd, Department of Animal and Poultry Science, University of Saskatchewan.

	Percentage of milking herd lame			
Year	sole ulcer	White zone lesions	Clinical laminitis	All causes
1986	30	2	5	37
1987	13	0	4	17
1988	27	8	5	40

A. Evaluation of the claws

The soles of the claws of the pelvic limbs of three groups of cows and heifers were examined on four occasions at intervals of two months starting four months prior to calving. The evaluation included ten heifers, ten second calf cows, and ten cows that had produced two or three calves. Selection was according to calving date.

A layer of approximately 1 mm of horn was pared from the soles of all four claws of the pelvic limbs. The exposed surfaces of fresh sole horn were photographed. Using the photographic records, the claws of the 30 animals were examined on two separate occasions by both authors, who independently recorded the sole haemorrhage scores. The average scores were used for the evaluation.

B. The variables for evaluation

Zone zero was added to the five zones of the sole described by several workers (16 - 18) (Figure 1). The addition of this zone proved to be necessary because severe haemorrhages and so called "ulcers" were observed in this region.

FIGURE 1 Zones of the sole amended to include zone 0.

zone 0 = white zone at the toe   zone 1 = apex of the sole
zone 2 = abaxial white zone   zone 3 = axial groove
zone 4 = junction sole and bulb   zone 5 = bulb
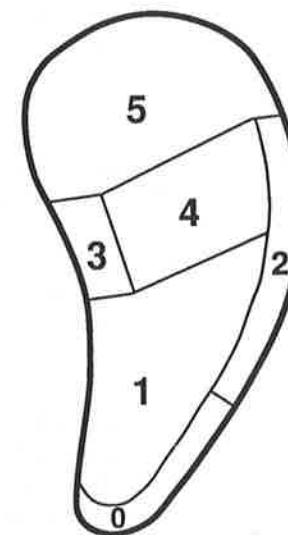
The observed haemorrhages or lesions were scored on a scale of 0 to 5. The score of the haemorrhage or lesion was converted to a weighted severity score by making geometrical adjustments that would cause each score level to represent twice the severity of the previous level (Table 2).

Table 2:
Scale of haemorrhage scores and corresponding, geometrically adjusted severities.

Observed score	Weighted severity	Observations
0	0	No visible change
1	1	Faint discolouration or blush
2	2	Trace, light-red or brown discolouration, often diffuse.
3	4	Obvious, clear red discolouration.
4	8	Loss of continuity of horn, corium open to the environment, and presence of fresh or clotted blood.
5	16	"Clinical" lesion E.g. sole ulcer and an associated hemorrhagic reaction.

We found, by trial and error, that this weighting was appropriate because lower scores represented slight lesions that could be either transient or reversible whereas higher scores indicated major tissue damage.

C. Statistics

The Kruskal-Wallis k-sample test (19) was used to analyse the differences in sole haemorrhage severities among the three groups of dairy cattle.

Results

A total of 1,920 observations was recorded from the sole of the claws of the pelvic limbs of the animals under study. The weighted sole haemorrhage severities for the white zone (zones 0 + 2) and the sole (zones 1 + 4) in all four hind claws at each examination are presented in Tables 3 to 5. Of the 60 lateral claws, 27.6% had haemorrhagic lesions with a weighted severity of 5 or more in one or more of these zones; only 9.5% of the medial claws were similarly affected.

Table 3:
The cumulative[1], weighted sole haemorrhage severities for ten dairy heifers at four and two months prior to calving, at calving, and two months postpartum.

	Zones 0 + 2[2]				Zones 1 + 4[3]				
Heifer	-4	-2	0	+2	-4	-2	0	+2	sole ulcer
413	4	9	2	1	4	5	17	1	-
414	6	10	0	0	6	2	4	0	-
415	0	0	1	0	1	4	0	0	-
416	1	2	6	1	9	4	3	2	-
417	4	2	2	2	4	2	2	2	-
418	7	2	2	1	7	3	3	3	-
419	4	0	30	0	0	1	14	1	-
420	5	14	14	1	8	6	6	34	-
421	0	1	0	5	0	0	0	0	-
422	1	7	7	1	9	19	19	18	-

Table 4:
The cumulative[1], weighted sole haemorrhage severities for ten second calf dairy cows at four and two months prior to calving, at calving, and two months postpartum.

	Zones 0 + 2[2]				Zones 1 + 4[3]				
Cows	-4	-2	0	+2	-4	-2	0	+2	sole ulcer
381	1	0	1	14	0	3	0	5	+
388	2	0	2	2	25	2	4	4	-
391	1	1	0	0	0	0	0	0	+
393	0	11	11	11	0	6	6	6	+
394	7	0	12	0	7	0	2	2	-
395	0	1	4	0	0	0	2	1	-
396	0	0	2	2	5	2	4	4	-
397	0	26	3	13	2	7	38	38	+
359	1	11	4	0	10	6	3	1	+
376	7	0	2	2	7	7	4	4	-

[1] The cumulative scores is calculated as the sum of the weighted haemorrhage scores for 2 zones, in all 4 hind claws at each examination.

[2] Zones 0 and 2 refer to the white zone of the claw.

[3] Zones 1 and 4 refer to the sole of the claw.

Table 5:
The cumulative, weighted sole haemorrhage severities for ten mature dairy cows at four and two months prior to calving, at calving, and two months postpartum.

Cows	Zones 0 + 2				Zones 1 + 4				sole ulcer
	-4	-2	0	+2	-4	-2	0	+2	
177	0	0	0	13	0	5	12	2	-
286	6	6	8	8	2	2	5	6	+
300	0	0	0	0	0	0	0	0	-
317	3	0	0	8	2	6	12	13	-
324	0	0	0	0	6	6	6	7	+
326	13	13	13	3	6	6	6	0	+
329	8	4	12	28	5	4	1	10	+
352	0	0	0	0	6	6	6	6	-
355	6	6	6	5	1	2	1	0	-
362	0	1	2	2	0	2	2	2	-

Two heifers developed a lesion similar to an early sole ulcer in zone four at two months after calving. Others had lesions in zones zero and two. In the bimonthly observations preceding the development of sole ulcers, haemorrhage was observed at the location at which ulcers ultimately developed. No lesions were observed in zone three. The heifers had, on average, more severe haemorrhages than did older animals four months prior to calving (Figure 2). In some instances, the lesions were sufficiently marked to indicate that the changes had commenced well in advance of the first examination. In each of the age groups studied, the haemorrhage severities tended to be higher at calving time and/or at the two month postpartum examination than they were prior to calving. The sole haemorrhage severities at calving were higher in heifers than in the older animals (Figure 2). The severity of sole haemorrhages differed between the groups at each examination but were not significantly different when considered cumulatively for all four examinations. The distribution of haemorrhage and other lesion severities differed relative to the time of calving between heifers on the one hand and older cows on the other.

Of the 94 heifers calving and introduced into this herd over a period of five years, 15% had sole ulcers during the first lactation and 37% were similarly affected during the second lactation. Every animal in the three groups studied had heel erosions at some time during the six months of study. The depth of the erosions increased either at or two months after calving. In eleven animals with deep erosions, a distinct and diffuse haemorrhage of the sole was present.

Figure 2
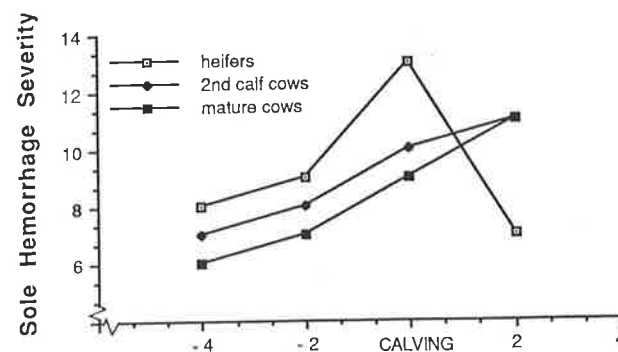
Discussion

In this study, observations were limited to four examinations in the peripartum period, during which haemorrhages of the sole have been reported previously (2, 4, 10). Some of the lesions observed were not severe in themselves but, because they affected more than one zone of several claws of the same animal, indicated extensive involvement of the sole horn. We believe that the presence of haemorrhages in several zones of the sole is an indication of subclinical laminitis which predisposes the claws to further disease. However, conventional scoring on a scale of 0 to 5 could be misleading. For example, a cumulative score for five slight haemorrhages could rank equal in importance to one severe lesions. In order to recognize the clinical significance of severe lesions, we have adopted a weighted score which is based on geometric progression. This scoring system worked well, for us, in this study. In the groups studied, 69% of the animals accumulated a sole haemorrhage severity of greater than five for all hind digits in at least one of the four examinations. We suggest that if an individual animal has a cumulative, weighted haemorrhage score of five or more, it probably has subclincial laminitis. From a practical point of view, five weighted scores of one or one weighted score of five may be regarded as the threshold of disturbed claw health. A practitioner may wish to increase this threshold to suit his method of epidemiological investigation.

There was a trend to rapid disappearance of haemorrhages from the claws of heifers after the first calf (Figure 2). The recovery was not evident after the second calf. Mature animals showed no sign of immediate recovery. These observations would support the concept proposed by Peterse (8) that scar tissue in the corium may cause slower recovery in older animals. It must be noted that sole ulcers were less commonly observed during the first lactation than during the second or third

(Tables 3-5). Our results indicate that, because sole haemorrhages in heifers are present four months prior to calving, it would follow that the causative factors must be present prior to mid-pregnancy. Subclincial laminitis in heifers is an increasing concern for practitioners and has been addressed by several workers (6, 20-23).

It has been reported that lameness-causing lesions occur in the lateral claws of more than 80% of all cases of pelvic limb lameness (15, 24-26). The results of our study demonstrate that the sole haemorrhage severity was three times greater in lateral than in medial claws.

Erosion of the heel is defined as an irregular loss of heel horn in the form of multiple irregular depressions or deep oblique grooves (27). This condition is found to affect the heels of more dairy cattle kept under intensive conditions. The clinical importance of heel erosion is associated with the loss of function of the heel as a primary shock absorber. Advanced heel erosions will cause mechanical stress to be transferred to other areas of the claw. Therefore, loss of heel horn can be a precipitating cause of sole ulcer or white zone lesions.The aetiology of the disorder has been poorly understood and commonly regarded as being associated with unhygienic conditions. Toussaint-Raven associates the phenomenon with interdigital dermatitis caused by Bacteroides nodosus (1). Our observations lead us to believe that there is a third explanation for the aetiology of heel erosion. The condition appears to be related to the haemorrhagic events associated with subclinical laminitis. Disturbance of the microvasculature of the corium results in the escape of blood components into the tubules of the horn of the sole and bulb. As recovery occurs and sound horn is produced, the event is recorded as a blood-stained stratum. As new horn is generated, the haemorrhagic strata move towards the surface. During wear (or if horn is pared) the haemorrhagic stratum appears in the substance of the horn until it too is worn away. A haemorrhagic stratum often terminated as a groove may be deep. Usually, deep grooves become contaminated and disintegration of the heel horn may occur. The pink or dark red staining of the sole and white zone precede, by several weeks, the appearance of heel lesion that are of clinical significance.

The haemorrhages observed in the white zone of the abaxial wall and toe regions were not severe in the animals studied. However, we believe that these lesions can lead to separation and disintegration of the white zone. When avulsion of the wall from the sole occurs, the space becomes readily impacted with foreign material and infection can result.

Acknowledgments

References
1. Toussaint-Raven E. Lameness in cattle and foot care. Tijdschr Diergeneeskd 1971; 96: 1244-1264.
2. Nilsson SA. Clinical, morphological and experimental studies of laminitis in cattle. Acta Vet Scand (Supp) 1963; 4: 124-139.
3. Andersson L, Bergman A. Pathology of bovine laminitis especially as regards vascular lesions. Acta Vet Scand 1980; 21: 559-566.
4. Peterse DJ. Incidence of lesion of the sole. Tijdschr Diergeneesk 1982; 107: 132-137.
5. Greenough PR. The sub-clinical laminitis syndrome. Bovine Pract 1985; 20: 14-149.
6. Bradley HK, Shannon D, Neilson DR. Subclinical laminitis in dairy heifers. Vet Rec 1989; 125: 177-179.
7. Smedegaard HH. Contusion of the sole in cattle. Veterinarian 1964; 2: 119-132.
8. Peterse DJ. Judgement of bovine claws by the occurrence of sole lesions. PhD Thesis, Rijksuniversiteit Utrecht 1980.
9. Peterse DJ. Nutrition as a possible factor in the pathology of ulcers of the sole in cattle. Tijdschr Diergeneesk 1979; 104: 966-970.
10. Mortensen K, Hesselholt M. Laminitis in Danish dairy cattle - an epidemiological approach. Proc IVth Symp Disorders Ruminant Digit Paris, France, 1982.
11. Mortensen K, Hesselholt M. The effects of high concentrate diet on the digital health of dairy cows. Proc Int Prod Cong Belfast, N. Ireland, 1986.
12. Whitaker DA, Kelly JM, Smith EJ,. Incidence of lameness in dairy cows. Vet Rec 1983; 113: 60-62.
13. Pinsent PJN. The management and husbandry aspects of foot lameness in dairy cattle. Bovine Pract 1981; 16: 61-64.
14. Baggott DG, Russell AM. Lameness in cattle. Br Vet J 1981; 137: 113-132.
15. Vhoquette-Levy L, Baril M, St-Pierre H. A study of foot disease of dairy cattle in Quebec. Can Vet J 1985; 26: 278-281.
16. Greenough PR. The nomenclature of anatomical features of the bovine digits. Report IInd Symp Bov Digital Dis, Skara, Sweden, 1978.
17. Weaver AD, Andersson L, De Laistre Banting A, et al. A review of disorders of the ruminant digit with proposals for anatomical and pathological terminology and recording. Vet Rec 1981; 108: 117-120.
18. Mill LL, Leach DH, Smart ME, Greenough PR. A system for the recording of clinical data as an aid in the diagnosis of bovine clinical disease. Can Vet J 1987; 8: 293-300.
19. Steel RGD, Torrie JH. Principles and Procedures of Statistics: A Biometrical Approach. New York: Mcgraw-Hill, 1980: 544-545.
20. Bergsten C. Sole bruising as an indicator of laminitis in cattle. A field study. Proc XVth World Buiatrics Cong, Palma de Mallorca, Spain, 1988: 1072-1076.
21. Bergsten C, Andersson L, Wiktorson L. Effect of feeding intensity at calving on

the prevalence of subclinical laminitis. Proc Vth Symp Disorders Ruminant Digit, Dublin, Ireland, 1986: 33-37.

22. Bazeley K, Pinsent PJN. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Vet Rec 1984; **115**: 619-622.

23. Colam-Ainsworth P, Lunn GA, Thomas RC, Eddy RG. Behaviour of cows in cubicles and its possible relationship with laminitis in replacement dairy heifers. Vet Rec 1989; **125**: 573-575.

24. Arkins S. Lameness in dairy cows. Irish Vet J 1981; **35**: 163-170.

25. Russell AM, Rowlands GJ, Shaw SR, Weaver AD. A survey of lameness in British dairy cattle. Vet Rec 1982; **106**: 155-160.

26: Eddy RG, Scott CP. Some observations on the incidence of lameness in dairy cattle in Somerset. Vet Rec 1980; **106**: 140-144.

27. Espinasse J, Savey M, Thorley CM, Toussaint Raven E, Weaver AD. Colour atlas on disorders of cattle and sheep digit, Editions du Point Veterinaire, 1984.

Description and Diagnosis of Superficial Digital Lesions in Dairy Cattle

R.W. Blowey

124, Stroud Road, Gloucester.

Introduction

The bovine digit is affected by a range of superficial lesions. These may be of genetic, traumatic, environmental or infectious origin. Interdigital skin hyperplasia is commonly seen in Hereford cattle, although certain lines of British Friesian and other beef breeds may also be affected. Persistent dry or sandy conditions may lead to loss of perioplic horn and the development of vertical fissures (sandcracks), especially following damage to the hairless skin at the coronary band. Infected fissures, especially those with proliferation of granulation tissue, produce severe lameness. Acute disease, for example feveror toxaemia (e.g. toxic mastitis), which causes a temporary cessation of horn formation, may lead to the development of horizontal fissures. Many are not observed clinically until months after the event. Broken toes may be secondary to horizontal fissures or traumatic in origin, for example an overgrown claw which becomes lodged between the slats of a slatted floor. Heel necrosis, or slurry heel, is an erosive process which occurs in housed cattle, often those kept under unhygienic conditions. Initial stages are seen as small pits, depressions and fissures in the normally smooth soft horn covering the heel bulb. In more advanced cases the horn may be totally eroded. This destabilises the normal weight-bearing surface of the claw. With the heel no longer acting as a full weight-bearing surface, excess weight is taken on the sole, the toe may deviate dorsally, the pedal bone may rotate and sole ulcers may result. In severe cases under-run solar horn extends from the sole ulcer to the remaining heel horn. Digital dermatitis has only recently been reported in Britain (Blowey & Sharp 1988), although its occurrence is widespread in Europe (Cheli & Mortellaro 1986, Peterse 1986) and a similar condition has been reported in North America (Rebhun et al 1980; Weaver 1989 personal communication). This paper gives a detailed description of the lesions as seen in the United Kingdom and discusses control measures currently in use.

Clinical Features

Lameness is the most pronounced clinical sign. This may be sudden in onset, or develop slowly over a few days. Cows with mild lesions only show discomfort, moving from one foot to another, or shaking the affected foot when in the parlour, thus interfering with milking. Acutely lame cows with more advanced lesions on the plantar aspect of the foot tend to walk on their toes. Neglected cases which walk in this way may totally erode the toe, leading to exposure of the sensitive laminae and severe

lameness.

Typically the lesion occurs on the skin at the plantar aspect of the foot, mid-way between the heel bulbs and adjacent to, but not initially involving, the soft perioplic horn covering the heels (Fig. 1). Early lesions produce matting of the hairs, which stand erect in a thick, light brown exudate, which has a characteristic pungent odour. Hair loss occurs later. When superficial detritis has been removed, the lesion is seen as a circular moist, red, raw and granulating area. It is extremely sensitive to the touch, much more than would normally be expected from a superficial lesion with no associated soft tissue reaction. More advanced cases may damage the perioplic horn of the heel, leading to an under-run heel and eventually an under-run sole. Many of the latter cases have a concurrent sole ulcer. This may be coincidental, although the loss of important weight-bearing heel tissue will lead to excess pressure on the solar laminae and predispose to solar ulceration.

Lesions may also occur on the anterior aspect of the foot. They often involve both the perioplic horn and coronary band of one claw only and are less commonly seen in the mid-line than the plantar lesions. Occasional cases may involve the lateral aspect of the claw at the coronet. A deep erosive area is produced, causing a severe lameness which is often more difficult to treat, probably because of the coronary band damage.

Interdigital dermatitis produces a similar superficial inflammation of the skin in the interdigital cleft. Lesions are commonly seen on the surface of interdigital skin hyperplasia, but may also occur in an otherwise normal interdigital cleft. The lesion is very painful when touched, although the pungent purulent exudate of digital dermititis is not a feature. Proliferative forms have been described (Cheli and Mortellaro 1986) but have not been reported in the U.K.

Histopathology

Superficial suppurative inflammation and thickening of the epidermis is typically seen, with areas of necrosis, micro-abscessation and infiltration with neutrophils and mononuclear cells (Blowey & Sharp 1988; Bassett et al 1990). Deeper dermal changes of mononuclear perivascular cuffing have been reported (Rebhun et al 1980; Blowey & Sharp 1988), but others consider this to be a normal feature (Bassett et al 1990).

Incidence

Lesions may occur in all four feet, although the hind feet are more commonly affected than the fore feet. Typically only one foot is affected (Blowey 1988), but multiple cases may occur in the same animal (Peterse et al 1982). Digital dermatitis is more commonly seen when cows are housed, although a lower incidence of cases may be seen at pasture. Lesions are more common in heifers (Blowey & Sharp 1988), or in cows recently introduced into an infected herd (Nutter and Moffitt 1990). Concurrent thickened soles and heel necrosis are common (Blowey & Sharp 1988).

Aetiology

A wide range of aetiological agents has been suggested, including bacterial, viral and mycotic infections, nutritional deficiencies and poor foot environment (Cheli & Mortellaro 1986). The rapid spread and wide distribution of lesions within some herds suggests infectious causes and the rapid response to topical antibiotic and gentian violet spray would suggest a bacterial. Bacteroides capillosus and B. fragilis have been isolated (Blowey & Sharp 1988). Both are anaerobes associated with human soft tissue infections. B. nodosus is a common cause of interdigital dermatitis in the Netherlands (Peterse 1986) but repeated attempts have failed to isolate the organism from affected cattle in Britain. (Blowey, unpublished data).

Differential Diagnoses

Digital dermatitis is a distinct entity, unlikely to be confused with other conditions. Interdigital necrobacillosis ('foul', 'foot-rot') is an infection of the dermal tissues caused by Bacteroides necrophorus. Typically there is an extensive soft tissue swelling forcing the claws apart and there is good response to parenteral antibiotic therapy. The skin in the interdigital cleft may be split, in advanced cases producing deep necrosis and a caseopurulent exudate. Mud fever is a superficial inflammation and thickening of the skin extending from the heel to the fetlock, with the plantar aspect of the pastern being the primary site. Dermatophilus has been proposed as a cause. Lesions occur under cold, wet conditions, typically when cattle have to walk or stand in muddy areas following turnout from housing. Response to treatment is slow, with housing under dry conditions being the most important therapy.

Treatment

Individual cases of digital dermatitis are treated by cleaning the surface of the lesion (often simply scraping with a hoof-knife) and applying a topical gentian violet plus 2.5% w/w (chlor)tetracycline aerosol. Gentian violet alone is effective, but less so. A 2.0% w/w gentian violet plus 6.5% w/w dichlorophen aerosol has given good results in a limited number of herds. Herd outbreaks are controlled by use of a footbath. Most authors consider that formalin is not effective (Cheli & Mortellaro 1986; Blowey & Sharp 1988; Blowey 1990; Nutter & Moffitt 1990), although some report good control following its use (Clark 1990). Footbaths containing 2-4g per litre tetracycline (Peterse 1986; Blowey & Sharp 1988), 0.5-1.0g per litre dimetridazole (Blowey 1988) or 20% zinc sulphate (Nutter & Moffitt 1990) are more commonly used. The frequency of foot-bathing required varies with the severity of the outbreak, the nature and concentration of the active ingredient in use and the degree of faecal soiling of the feet prior to the use of the foot-bath. In typical cases, good control is obtained by running a herd of 100-150 cows through a 2g/litre tetracycline footbath prepared fresh twice daily for two days, that is a total of four passages. After the first passage cows may be seen shaking affected feet, due to irritation. Attempts to use only the citric acid carrier (the base for tetracycline) at 2g/litre in the footbath have not
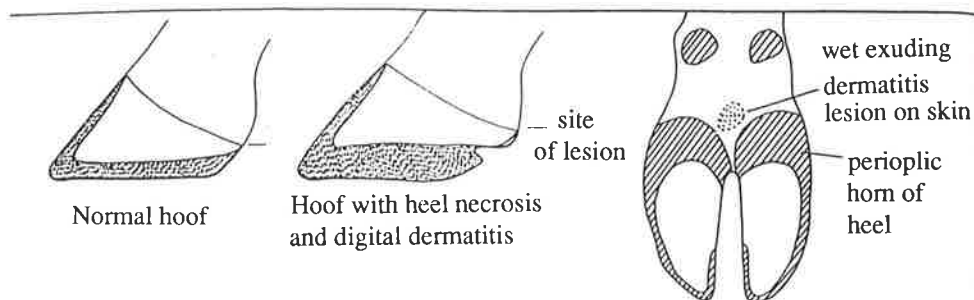
proved successful (Blowey 1990).

Figure 1. The typical position of digital dermatitis lesions. Lesions may also
occur on the anterior aspect of the foot.
(Diagram reprinted from the Veterinary Record)

References
Bassett H.F., Monaghan M.L., Lenhan P., Doherty M.L. & Carter M.E. (1990) Vet.
    Rec. 126 164 Bovine Digital Dermatitis.
Blowey R.W. & Sharp M.W. (1988) Vet.Rec. 122 505-508 Digital Dermatitis in Dairy
    Cattle.
Blowey R.W. (1988) Proc. Brit. Cattle Veterinary Association 1988-89, p.1 Bovine
    Digital Dermatitis.
Blowey R.W. (1990) Vet.Rec. 126 120 Digital Dermatitis Control.
Cheli R. & Mortellaro C.M. (1986) Proc. 5th Int. Symp. on Disorders of the Ruminant
    Digit pp.8-15. Digital Dermatitis Today and Tomorrow.
Clark P.J. (1990) Vet. Rec. 126 200 Digital Dermatitis Control.
Nutter W.T. & Moffitt J.A. (1990) Vet. Rec. 126 200 Digital Dermatitis Control.
Peterse D.J., Cornelisse J.L. & Toussaint Raven E. (1982) Proceedings 12th Int. Conf.
    on Diseases of Cattle, Amsterdam (1982), pp 830-832.
Peterse D.J. (1986) Proc. 14th World Congress of diseases of Cattle, Dublin 1986 vol.
    2 pp1015-1023
Rebhun W.C., Payne R.M., Kings J.M., Wolfe M. & Begg S.N. (1980) Journal of the
    American Veterinary Assoc. 177 437 Interdigital Papillomatosis in Dairy Cattle.

Aspects of bovine endotoxaemia of possible relevance to lesions in the ruminant digit

Pia Haubro Andersen, DVM, Ph.D.

*Dept. Clinical Studies, Royal Veterinary and Agricultural
University, DK–1870 Frederiksberg, Denmark.*

INTRODUCTION
Endotoxins are defined as "lipopolysaccharide–protein complexes contained in
the cell walls of Gram–negative bacteria including non–infectious Gram–negatives"
(Merck Index 1983). Endotoxins are responsible for many of the clinical signs of
patients infected with Gram–negatives. Clinical symptoms of endotoxicosis are dose–
dependent and appear in cattle a few minutes after intravenous administration of
endotoxin. Respiration rate increases 3 –4 times, the animal starts to salivate, urinate
and defecate and pulse frequency increases to up to twice the initial rate. There is an
initial endocrine–metabolic stress response, characterized by hyperglucosaemia,
hyperlactaemia and increases in plasma cortisol concentration. The onset of
endotoxicosis and the severity of the symptoms – fever, leukopenia,
thrombocytopenia, hypoglycaemia and hypozincaemia – are dose–dependent (1,2).
The animal will die in circulatory shock if the endotoxin dose is large (we have
injected 25 ug of an E. coli O55:B5 endotoxin per kg b.w. without lethality) or if
the animal is otherwise debilitated (we have unfortunately experienced a cow that
died after injection of 0.1 ug of the same endotoxin per kg b.w.) However, clinical
disease is elicited by much lower doses.
It is often stated that the development of endotoxaemia is of importance to the
pathogenesis of various clinical conditions, from "sudden death syndrome" in feed–
lot cattle to E. coli mastitis and laminitis in dairy cattle. At the same time there is
little decisive evidence of the presence of endotoxaemia in these cases.
Circumstantial evidence is on the other hand strong since the symptomatology of
bovine endotoxicosis is well documentated, both clinically and clinical–chemically.
One major cause of the diffulty in substantiating the diagnosis of endotoxaemia
by demonstrating the presence of free endotoxin may be that the endotoxin
concentration in blood of animals suffering from endotoxaemia is extremely low
(picograms per ml). The only endotoxin analysis methods with sensitivities high
enough to meet these demands are based on the Limulus Amoebocyte Lysate
principle, which unfortunately does not distinguish between "genuine endotoxins"
and contaminants, which are everpresent (3). Contamination of blood samples,
glassware and testing equibment with environmental endotoxins can easily occur. We
believe that the detection of endotoxins in healthy animals, e.g. in pre–experimental
periods, is a result of contamination and that such experiments are strongly biased.
Indeed very many such experiments fail to establish proper negative controls.

The contrary problem; failure to detect endotoxins in the blood of obviously sick animals, has been a challenge to our team in Copenhagen and we have questioned the presence of endotoxaemia as a necessity for the development of clinical disease. We are presently studying three main areas: firstly, the rumen is thought to be the main source of endotoxins in feeding induced endotoxaemia. How do different feeding regimens influence rumen endotoxin ? Secondly, if endotoxin is introduced into the circulatory system, what happens to it when followed using the Limulus Amoebocyte Lysate test ? And thirdly is it possible to establish a relation between the extent of endotoxicosis and the release of inflammation mediators, as it has been shown for other species ? In the following, a brief discussion of the experimental work performed in Copenhagen in these areas will be given.

PRODUCTION DISEASES AND ENDOTOXAEMIA

Epidemiological investigations have linked high concentrate feeding regimens with production diseases such as laminitis, abomasal displacement, fatty liver syndrome and "sudden death" of feed-lot steers (4,5,6,7). The almost unnaturally high production of milk and meat in modern production systems is only maintained by rations containing large amounts of concentrates, which are likely to produce an acidotic rumen environment (8). The combination of rumen acidosis and high carbohydrate feeding has often been proposed to play a central role in the pathogenesis of production diseases as high carbohydrate feeding results in an increased number of Gram-negative bacteria. This again has led to the hypothesis that rumen acidosis is associated with a dramatic increase in ruminal endotoxin concentration (9, 10, 11, 12, 13).

TOXICITY OF RUMEN FLUID

Toxicity of rumen fluid is well described and the acidotic rumen environment has been investigated by several authors in attempts to determine one or more factors responsible for the toxicity. Particular attention has been paid to the accumulation of D(–) lactic acid, ethanol, tyramine, histamine and endotoxin (14, 15, 16, 17, 18, 19, 20). Of these single factors, only the experimental administration of endotoxin seems to elicit all the typical clinical signs of acute rumen acidosis. Intravenous administration of rumen bacterial endotoxin also induces the classic symptoms of endotoxicosis (21) and the possible absorption of endotoxin from the gastrointestinal tract is therefore of great interest.

QUANTIFICATION OF RUMEN ENDOTOXIN

The normal rumen environment is inhabited by many Gram-negative bacteria and endotoxin is released when the bacteria grow or die. Up to 60% of the total bacterial endotoxin may be released during growth and the presence of free endotoxin in rumen fluid is probably due to a combination of lysis, caused by for instance low pH, and growth.

The rumen endotoxin concentration is thought to berelated to the total number of Gram-negative bacteria in the rumen contents. The number of Gram-negative in relation to Gram-postitive bacteria is larger in hay-fed cattle than in grain-fed cattle (22), but the total number of bacteria is greater in grain-fed cattle and consequently the total number of Gram-negative bacteria is greater in grain-fed cattle (10). Endotoxin extracted from the rumen bacteria causes gelation of Limulus amoebocyte lysate (LAL) but until now LAL has not been used to determine endotoxin concentrations in rumen fluid. Based on the lethal effect on mice of actinomycin D potentiated rumen fluid, Nagaraja et al. (10) demonstrated that the free endotoxin concentrations in the rumen fluid of steers fed on high concentrate rations were considerably higher than in steers fed solely on hay. We have used the LAL test to investigate the rumen endotoxin concentrations during the adaptation from all hay rations to high concentrate rations and we were able to confirm the results obtained by Nagaraja et al., though the increases observed in our study were 3 – 4 times larger (unpublished). This discrepancy may be ascribed to the less sensitive method employed by Nagaraja et al. What we found more interesting was that the endotoxin concentration did not change during the first days after the grain supplement. The endotoxin levels started to increase after three days of feeding with concentrates and reached a steady level approximately three times higher than the base-line values after six days. When the cows were fed concentrate ad libitum, the endotoxin concentrations increased by up to eight times the base-line values. The results indicate that high rumen endotoxin concentrations are closely related to a rumen environment previously adapted to concentrate feeding.

ENDOTOXIN AND RUMEN ACIDOSIS

Acute rumen acidosis does not develop under normal intensive feeding conditions, but is frequently diagnosed after over-eating or accidental changes in rations. In any case, acute rumen acidosis is suitable as an experimental model for the more commonly occurring subclinical types of rumen acidosis. Subclinical rumen acidosis is characterized by the low buffering capacity of the ruminal contents and low pH values may be expected temporarily and/or locally. Various degrees of damage to the ruminal barrier are therefore common in both types of acidosis, ranging from local spots with superficial lesions to extensive areas with profound destruction of the ruminal epithelium (23, 24, 8).

Most experimental rumen acidosis models assume that optimal experimental conditions are achieved when grain-engorgement is induced suddenly in animals not previously adapted to grain. However, the pattern of feeding induced changes in endotoxin concentrations described above indicate that this may not be true. Therefore, we induced grain-engorgement in animals adapted to grain and animals fed on a hay regimen both grain adapted and hay maintained animals. Two animals were maintained on an all-hay ration for 3 weeks prior to engorgement, and the other two were allowed concentrates and hay ad libitum. Experimental grain engorgement was induced in four cows with app. 70 g barley pr kg b.w. given through a rumen fistula (9). The clinical and the clinical-chemical picture obtained in the present study is in accordance with the diagnostic criteria of acute rumenacidosis (8, 18). A severe rumen acidosis developed in all the experimental animals, but the rumen endotoxin concentrations did not change or even decreased in the two hay-fed cows, while the concentrations rose to up to 50 – 60 times the baseline values in the two cows fed on concentrates ad libitum. These results indicate that adaptation of the rumen environment to grain feeding provides a greater

potential for the release or production of free endotoxin in the rumen fluid. The rumen endotoxin load in grain–engorged feed–lot cattle or high yielding dairy cattle must therefore be expected to be much heavier than in hay–fed cattle.

If ruminal endotoxin is involved in the pathogenesis of the disease which follows grain–engorgement, it seems as though high yielding cattle are more sensitive to grain overload than cattle on low intensity feeding.

ABSORBTION OF RUMEN ENDOTOXIN

There has been considerable controversy concerning the possible absorbtion of gastrointestinal endotoxin. Many, especially older studies, have demonstrated that endotoxin does not escape from the normal gastrointestinal tract (25, 26), but the general opinion nowadays is that small amounts of endotoxin cross the epithelial barriers and enters at least the portal circulation (27, 28, 29) even in normal individuals. We have previously described the presence of portal endotoxaemia in healthy ruminants and ruminants with experimentally induced rumen acidosis (13), and we are now working on the possible relationship between rumen epithelial damage and endotoxin concentration in the portal blood.

The escape of endotoxins from the portal blood to the peripheral circulation is somewhat more doubtful and the discussion has to takeinto consideration the endotoxin clearance capacity of the liver. Yet it is generally agreed that endotoxins are not contaminants of systemic blood in healthy individuals and that an impairment of liver function may result in systemic endotoxaemia in otherwise healthy humans.

While Dougherty et al. (9) found evidence of the appearance of endotoxin in the systemic circulation after grain–engorgement, we did not detect endotoxin concentrations above the detection limit of 0.03 EU/ml plasma in the peripheral blood at any time during the acidosis experiments mentioned above. If endotoxins were present below this level, they may be considered to be of no biological significance. Another possibility is that a significant endotoxemia was present for a short period between two sampling times. This possibility cannot be ruled out as plasma endotoxin clearance seems to be extremely rapid (30). However, when endotoxins escape from the rumen they should be expected to do so in a continuous stream as the epithelial damage persists for many days. We therefore believe that when endotoxins do escape to the portal circulation, an immediate clearance in the normal liver will occur, preventing the endotoxins from entering the peripheral circulation. Some of the clinical–chemical results obtained in the same study are similar to those observed in endotoxicosis such as thrombocytopenia and hypozincemia (1,2 30). We therefore suggest that the portal endotoxaemia is of significance for the development of the clinical picture in rumen acidosis. The way that this occur could be thatinflammation mediators are synthesized either in the damaged ruminal wall, in the endotoxin triggered prehepatic endothelial cells and thrombocytes, or in the endotoxin clearing cells of the liver.

OTHER SOURCES OF ENDOTOXIN

Other bovine disease complexes than the directly rumen related diseases have been associated with endotoxaemia, such as Gram–negative mastitis, laminitis and uterine infections (31, 32, 33). The common pathogenetic factor responsible for the development of the common symptomatology of these diseases is thought to be the systemic entrance of endotoxins from the inflammated organ(s) but the relationship between the clinical symptoms of endotoxicosis and the presence of endotoxaemia is not very well described.

Attempts to detect endotoxins in the peripheral blood of cattle with experimental mastitis induced with endotoxin have failed, although the clinical–chemical findings reported in these experiments were in accordance with those of the classic endotoxicosis (31). However, we have investigated the occurence of endotoxaemia in cases of naturally occuring E. coli mastitis in a practice in Jutland. One study comprised 12 cows suspected of suffering from severe E. coli mastitis and 12 healthy control cows. Blood samples for endotoxin determination were drawn from the milk vein and milk samples were obtained from the infected quarter in order to determine the number of colony–forming units and to verify the clinical diagnosis. Only 9 cases turned out to be E. coli mastitis. Endotoxin was detected and quantified in 5 of these, whereas no endotoxin was detectable in either the control cows or the cows with other types of mastitis (34). The milk vein drains the udder and does not pass the liver on its way from the udder to the entrance in the caval vein. It is therefore uncertain if for instance jugular vein blood of the endotoxin postive cases would contain endotoxin. The role of the liver in endotoxin mediated diseases is discussed in the following.

ENDOTOXIN CLEARANCE

There have been very few investigations into endotoxin plasma disappearance in cattle, but it appears to be rapid according to data obtained from experiments with radiolabelled endotoxins (35). We determined endotoxin plasma disappearance times with a modified LAL technique after intravenous administration of a bolus of 0.025 mg E. coli O55:B5 endotoxin pr kg b.w. to Jersey cows. Group 1 comprised 6 clinically healthy cows, and the endotoxin bolus was almost cleared from the plasma within 15 minutes. The endotoxin doses were very large and the clinical situation obtained by the injection of a bolus is probably far removed from real life, where the endotoxin is likely to enter the blood in a slower and more continuous stream. Yet we have measured similar endotoxin concentrations in the blood of naturally occurring cases of E. coli mastitis (34) and we believe that our model reflects a true image of the clearance capacity. All the cows in this experiment developed serious endotoxicosis. The symptoms worsened for 6 – 8 hours after the challenge and clinical recovery took at least 24 hours, biochemical recovery took several days.

The endotoxin plasma disappearance times determined in healthy cattle with the modified LAL technique are apparentlyin accordance with the findings of Maxie et al. (35), who studied the clearance of 3H–labelled endotoxin from the blood of healthy calves. The use of the LAL technique for endotoxin detection in plasma is preferable to the use of radio–labelled isotopes, as LAL detects the biologically active principle of the endotoxin, while with the use of radio–labelled endotoxin it is possible that biologically insignificant products of degtadation may be measured. However, Maxie et al. (36) failed to detect the endotoxin with the LAL technique, probably because no extraction procedures were performed on the plasma before testing. The method used in our laboratory for the extraction of endotoxin from

potent
rumer
must

If
grain-
grain

ABS

gastr
endo
gener
barri
indiv
healt
and
dama

some
endo
are
of li

syste
conc
bloo
were
signi
short
plasr
endo
strea
wher
norr
circu
simil
hypc
signi
that
dam
throl

OTF

been
uteri

plasma is a combination of dilution and heating which has proven to be effective (37). Furthermore, the combination of the regular LAL technique with rocket immunoelectrophoresis has eliminated the problem of subjective visual reading, which is especially important when yellowish specimens containing proteins and lipids are tested (3).

THE LIVER AND MODULATION OF ENDOTOXICOSIS

The incidence of suspected endotoxin related diseases increases in the early post partum period, when hepatic lipidosis is frequent (38). As endotoxin clearance is highly dependent on the liver (39), we determined the plasma disappearance times in four cows with spontaneously developed hepatic lipidosis. Two of these cows were unable to clear the injected endotoxin dose at all, while the others had plasma disappearance times which were 14 – 16 times longer than the healthy group. The endotoxicosis induced was lethal in all cases. The results of this limited experiment suggest that hepatic lipidosis inhibits the endotoxin detoxification in the liver. This finding is of major clinical significance, since severe hepatic lipidosis develop in one third of high yielding dairy cows after calving and is associated with higher incidence of infectious and metabolic diseases, including laminitis (38).

The clearance capacity of a normal liver is thought to exceed the amounts of endotoxins contaminating the portal vein blood (40). Impairment of the liver function in humans due to cirrhosis, hepatitis or hepatic lipidosis results in a spillover effect, where endotoxins of gastro–intestinal origin may enter the systemic circulation (41, 42). The results therefore support the general opinion held by practitioners that cows suffering from hepatic lipidosis are far more susceptible to endotoxin–related diseases than otherwise healthy cows. Also, these results again underline the importance of hepatic lipidosis as a central problem in the discussion of the production disease complex, including laminitis.

ENDOTOXIN INDUCED MEDIATORS AND FREE OXYGEN RADICALS

The role of inflammation mediators in the development of endotoxicosis, especially the prostaglandins, is now widely recognised. Investigations on experimental endotoxaemia in horses, pigs, dogs, primates and cattle have shown the pathophysiological role of the arachidonic acid metabolites thromboxane A2 (TXA2), prostacyclin (PGI2) and prostaglandin E2 (PGE2) and illustrated their relationship to the development of the clinical syndrome endotoxicosis (43, 44,45, 46, 47).

The finding that a very high endotoxin bolus results in endotoxaemia for approximately 15 minutes, while the symptoms of endotoxicosis persist for at least 6 hours, supports the theory, that momentarily released endogenous mediators play a major role in the induction of endotoxicosis in healthy cattle. The administration of endotoxin to cattle induced increased synthesis of TXA2, PGI2 and PGE2, and we found that the increases in plasma concentration of these inflammation mediators persisted for several hours after the endotoxin was cleared from the systemic circulation (30, 47). The biological half–lives of the arachidonic acid metabolites are short and therefore synthesis must have continued after the challenge was cleared from the blood. One reason for the pparently continuing synthesis of arachidonic acid metabolites may be the formation of free oxygen–derived radicals. Whenever the arachidonic acid cascade is activated, free oxygen radicals (O2–) are generated as a "by–product" (48). They are extremely reactive and implicated in the pathophysiology of increased endothelial permeability and cytotoxity. They increase the local prostaglandin synthesis which again favors platelet aggregation and microvascular stasis and may bring the tissue into a vicious circle of hypoxia and cellular damage (49).

We detected only minor increases in plasma TXB2 and bicyclic PGE2 (stable metabolites of TXA2 and PGE2) during our preliminary acidosis experiments. Systemic endotoxemia was not detected and we think that many of the clinical signs observed in rumen acidosis may be attributed to the release of local inflammation mediators in the hepatic and pre–hepatic tissues and circulatory system.

The detection of the prostaglandin metabolites may be just the tip of the iceberg of endotoxin induced mediators. Several other mediators are implicated in endotoxicosis and platelet aggregation, e.g. platelet activating factor (PAF) and tumor necrosis factor (TNF) (50, 51) and still need to be investigated in relation to bovine acidosis and laminitis.

While the nature and occurrence of endotoxins seems to be increasingly clarified, the pathophysiology of endotoxicosis seems to be more and more complex as new inflammation mediators and modulators are discovered faster than clinical experiments are able to provide an understanding of their significance.

STRESS

We have observed conspicuous differences in the sensitivities of cows to experimental grain–engorgement, in spite of the fact that the cows were maintained on identical rations prior to the experiments. Under identical experimental conditions, for example one cow came down with severe acidosis after 11 hours, while at the same time the other was only lightly affected.

Plasma cortisol measurements in such cows showed that the more resistant cows had early increases in cortisol concentrations compared to the "sensitive" cows where the plasma cortisole level remained unchanged much longer. This difference could be due to an apparently better stress "defense" to the disease in the first cow. The natural blocking action of cortisol on the development of the arachidonic acid cascade has been described previously (52)and these findings underline the significance of the endocrine metabolic stress response in limiting the effects of the disease. Chronic stress due to disease, poor housing and management conditions etc., with a depletion of adrenal function is therefore likely to aggravate not only rumen acidosis but any other condition associated with the release of prostaglandin inflammation mediators, including laminitis.

REFERENCES

1 Griel LC, Zarkower A and RJ Eberhardt. Clinical and clinico-pathological effects of Escherichia coli endotoxin in mature cattle. Can J Comp Med 1975 **39**: 1–6

2 Luthman J, Kindahl H, Jacobson SO and Thunberg L. Local and General effects of Salmonella typhimurium Endotoxin in Calves. J Vet Med A 1988 **35**: 586–595

3 Baek L: New, sensitive rocket immunoelectrophoretic assay for measurement of the reaction between endotoxin and Limulus amoebocyte lysate. J Clin Microbiol 1983 **17**: 1013 –1020

4 Coppock CE : Effect of Forage – Concetrate Ratio in Complete Feeds Fed ad Libitum on Feed Intake Prepartum and the Occurrence of Abomasal Displacement in Dairy Cows. 1972 J Dairy Sci **55**, 983 – 933

5 Dougherty RW : Problems Associated with Feeding Farm Livestock under Intensive Systems. 1976 Wld Rev Nutr Diet **25**: 249 – 275

6 Hesselholt M, J Grymer, P Willeberg and P Haubro Andersen Left Abomasal Displacement: Some Aspects of the Ethiology and Treatment. Proc. XIIth World Congress on Diseases of Cattle, Amsterdam 1982 **2**: 729 – 734

7 Gerloff BJ, TH Herdt and RS Emery : Relationship of hepatic lipidosis to health and performance in dairy cattle. 1986 J A V M A. **188**: 845 – 850

8 Dirksen G : Acidosis. Physiology and Metabolism in the Ruminant. A.T. Phillipson 1970, 612 – 629

9 Dougherty RW, KS Coburn, HM Cook and MJ Allison: Preliminary Study of Appearance of Endotoxin in the Circulatory System of Sheep and Cattle after Induced Grain Engorgement. 1975 Am J Vet Res **36**: 831 – 832

10 Nagaraja TG, Fina LR, Bartley EE and HD Anthony. Endotoxic activity of cell–free rumen fluid from cattle fed hay or grain. Can J Microbiol 1978 **24**:1253–1261

11 Nagaraja TG, EE Bartley, LR Fina and HD Anthony: Relationship of Rumen Gram–Negative Bacteria and Free Endotoxin to Lactic Acidosis in Cattle. 1978 J Anim Sci **47**: 1329 – 1336

12 Andersen P Haubro : Portal Endotoxemia in Cattle – anexperimental investigation. Ph.D. thesis, Dept. of Surgery, The Royal Veterinary and Agricultural University, Copenhagen 1985

13 Dougherty RW and RM Cello: A Preliminary Report on Toxic Factors in the Rumen Ingesta of Cows and Sheep. 1949 Cornell Vet **39**:403 – 413

14 Turner AW and VE Hodgetts: Buffer Systems in the Rumen of the Sheep, I and II. 1955 Aust J Agri Res **6**: 115 – 144

15 Mullenax CH, RF Keeler and MJ Allison: Physiological Responses of Ruminantsto Toxic Factors Extracted from Rumen Bacteria and Rumen Fluid. 1966 Am J Vet Res **27**: 857 – 868

16 Ahrens FA : Histamine, Lactic Acid and Hypertonicity as Factors in the Development of Ruminitis in Cattle. 1967 Am J Vet Res **28**: 1335 – 1342

17 Dunlop RH : Pathogenesis of Ruminant Lactic Acidosis. 1972 Adv Vet Sci Comp Med **16**: 259 – 302

18 Koers WC, TJ Britton and WR Woods : Ruminal Histamine, Lactate and Animal Performance. 1976 J Anim Sci **43**: 684 – 691

19 McManus WR, DJ Cottle, GJ Lee, GDF Jackson and J Black : Observations on toxicity to mice of rumen fluid from sheep fed roughage or concentrate diets. 1978 Res Vet Sci **24**: 388 – 389

20 Nagaraja TG, EE Bartley, LR Fina, HD Anthony, SM Dennis and RM Bechtle : Quantitation of endotoxin in cell–free rumen fluid of cattle. 1978 J Anim Sci **46**: 1759 –1767

21 Nagaraja TG, EE Bartley, HD Anthony, HW Leipold and LR Fina: Endotoxin shock in calves from intravenous injection of rumen bacterial endotoxin. 1979 J Anim Sci **49**: 567 – 582

22 Krogh, N: Studies on Alterations in the Rumen Fluid of Sheep, Especially Concerning the Microbial Composition When Readily Available Carbohydrates are Added to the Food. 1961 Acta Vet Scand **2**: 357 – 374

23 Kay M, BF Fell and R Boyne : The Relationship Between the Acidity of the Rumen Contens and Ruminitis in Calves fed on Barley. 1969 Res Vet Sci **10**: 181 – 187

24 McManus WR, GJ Lee and VNE Robinson: Microlesions on rumen papillae of sheep fed diets of wheat grain. 1977 Res Vet Sci **22**: 135 – 137

25 Berczi I, L Bertok, K Baintner and B Veress: Failure ooral E. coli endotoxin to induce either specific tolerance or toxic symptoms in the rat. 1968 J Pathol Bact **96**:481 –1049

26 Sanford, J and HE Noyes: Studies on the absorbtion of Escherichia coli endotoxin from the gastrointesTinal tract of dogs in the pathogenesis of "irreversible" hemorrhagic shock.1958 J Clin Invest **37**:1425 – 1435

27 Gans H and K Matsumoto : The escape of endotoxins from the intestine. 1974 Surg Gynecol Obstr **139**:395 – 402

28 Jacob AI, Goldberg PK, Bloom N, Degenshen GA and PJ Kozinn Endotoxin and bacteria in portal blood. Gastroenterology 1977 **72**:1268 –1270

29 Nolan JP, KH Hare, JJ McDevitt and A Vilayat: In vitro studies of intestinal endotoxin absorbtion. 1. Kinetics of absorbtion in the isolated everted gut sac. 1977 Gastroenterology **72**:434 – 439

30 Andersen P Haubro, N Jarløv and M Hesselholt : Pathophysiology of experimental bovine endotoxicosis I; Endotoxin in vivo clearance in healthy and hepatic lipidotic cattle. Proceedings XVth World Buiatrics Congress, 1988 **2**:1410 –1415

31 Verheijden JHM, van Miert ASJPAM, Schotman AJH and van Duin CTM. Pathophysiological aspects of E. coli mastitis in ruminants. 1983 Vet Res Comm 7:29–236

32 Mortensen K, M Hesselholt and A Basse: Pathogenesis of bovine laminitis (diffuse aseptic pododermatitis). Experimental models. Proc. 14th World Congr. Diseases of Cattle 1986 **2**: 1025 – 1030

33 Fredriksson, G: Some reproductive and clinical aspects of endotoxins in cow with special emphasis on the role of prostaglandins. 1984 Acta Vet Scand **25**:365 – 377

34 Katholm, J and P. Haubro Andersen: Acute coliform mastitis: A field study on endotoxin and biochemical changes in plasma and colony–forming units (CFU) in milk. 1990 Abstr. Proc. International Conf. Mastitis: Pathology or physiology ? Gent, Belgium

35 Maxie MG, Valli VEO and JH Lumsden: Studies with radioactive endotoxin II. Clearance of 3H–labelled endotoxin from the blood of calves. 1974 Can J Comp Med **38**:367 – 382

36 Maxie MG and VEO Valli: Studies with Radioactive Endotoxin III. Localization of 3H–Labelled Endotoxin in the formed Elements of the Blood and Detection of Endotoxin in Calf Blood with the Limulus Amebocyte Lysate. 1974 Can J Comp Med **38**:383 – 390

37 DuBose DA, Lamaire M, Basamania K and J Rowlands: Comparison of plasma extraction techniques in preparation for samples for endotoxin testing by Limulus amoebocyte lysate test. 1980 J Clin Microbiol **11**:68 – 72

38 Reid IM and CJ Roberts: Subclinical fatty liver in dairy cows. 1983 Irish Vet J **37**:104–110

39 Rutenburg S, R Skarnes R, C Palmerio and J Fine: Detoxification of endotoxins by perfusion of liver and spleen. 1967 Proc Soc Exp Biol Med **125**:455 – 459

40 Yamaguchi Y, K Yamaguchi, JL Babb and H Gans: In vivo quantitation of the rat liver's ability to eliminate endotoxin from portal blood. 1982 J Reticuloendothelial Soc; **32**:409 – 422

41 Prytz, H, J Holst–Christensen, B Korner B and H Liehr: Venous and Systemic Endotoxeamia in Patients without Liver Diseases and Systemic Endotoxaemia in Patients with Cirrhosis. 1976 Scand J Gastroenterol **11**:857 –863

42 Ravin HA, D Rowley, C Jenkins and J Fine: On the absorbtion of bacterial endotoxin from the gastrointestinal tract of the normal and shocked animal. 1960 J Exp Med **112**: 783 – 792

43 Bottoms GD, CB Templeton, JF Fessler, MA Johnson, OF Roesel, KM Evert and SB Adams : Thromboxane, prostaglandin I2 (epoprostenol), and the hemodynamic changes in equine endotoxin shock. 1982 Am J Vet Res **43**:999 – 1002

44 Bottoms GD, MA Johnson and OF Roesel: Endotoxin–induced hemodynamic changes in dogs : Role of thromboxane and prostaglandin I2. 1983 Am J Vet Res **44**:1497 – 1500

45 Harris RH, M Zmudka, Y Maddox, PW Ramwell and JR Fletcher: Relationships of TXB2 and 6–keto PGF 1 alpha to the hemodynamic changes during baboon endotoxic shock. 1980 Adv in PG and TX Res **7**:843 – 849

46 Schrauwen E, G Vandeplassche, G Laekemannn and A Houvenaghel : Endotoxin shock in the pig : Release of prostaglandins and beneficial effects of Flurbiprofen. 1983 Arch Int Pharmacodyn **262**:332 – 334

47 Jarløv N, P Haubro Andersen and M Hesselholt: Pathophysiology of experimental bovin endotoxicosis II; Investigations on endotoxin–induced synthesis of thromboxane A2, prostacycline and prostaglandine E2. 1988 Proceedings XVth World Buiatrics Congress **2**:1416 – 1421

48 Higgins AJ: The biology, pathophysiology, and control of eicosanoids in inflammation. 1985 J Vet Pharmacol Therapeut **8**: 1 – 18

49 Shlafer M, PF Kane, VY Wiggins and MJ Kirsch: Possible role for cytotoxic oxygen metabolites in the pathogenesis of cardiac ischemic injury. 1982 Circulation **66(suppl. 1)**:85 –92

50 Old, LJ: Tumour necrosis factor. Another chapter in the long history of endotoxin. 1987 Nature **330**:602 – 603

51 May SA, P Lees, AJ Higgins and AD Sedgwick: Inflammation: A clinical perspective. 1987 Vet Rec **120**: 514 – 517

52 Breazile J : Physiolgic basis and consequences of distress in animals. 1987
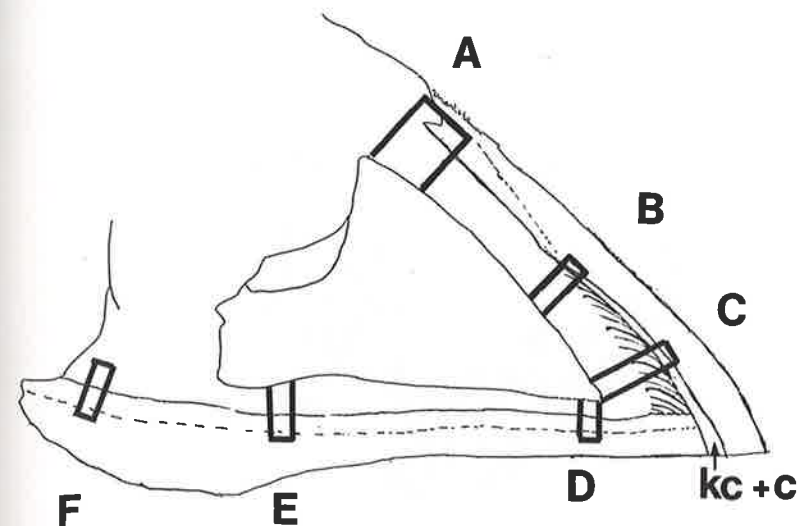J A V M A **10**:1212 – 1215

Fig. 1. Schematic illustration of sagittal section of bovine digit. A–F : Samples for histological examination (cf. Andersson & Bergman 1980) . A: Periople and coronet. B & C: Proximal and distal part of lamellated area, resp. D: Sole; point of pedal bone. E: Bulb; typical site of sole ulcer (pododermatitis circumscripta). F: Bulb; non–supportive area . **kc + c**: Stratum internum of the horny hoof wall; at section C, cap horn **kc** interdigitate with the basal parts of the horny lamellae **c** (cf. Dirks 1985 and fig. 3b). The dotted line indicates position of changes associated with an attack of laminitis approx. 14 days previously.

Labels used in this and the following illustrations are alike:
d: dermal lamella. **e**: epidermal lamella. **c**: keratinized/horny lamella; part of the epidermal lamella but separated here, for the sake of the morphological description. **kc**: cap horn; located between the horny lamellae in the distal half of the wall. **ke**: epidermal cap. **ca**: vessels of dermal lamellae.
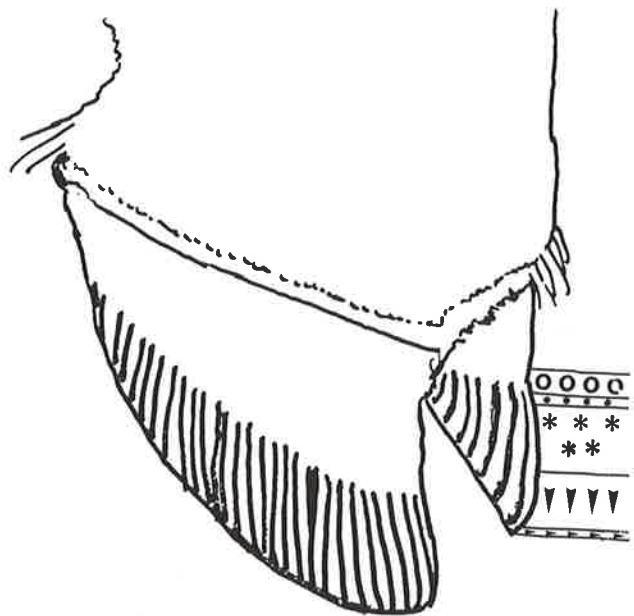
Fig. 2. Schematic illustration of an exungulated bovine foot showing areas of the wall and distal part of the coronet where the cellular growth and differentiation occurs at different rates (cf. <u>Dirks</u> 1985):

O O O Area 1 & 2: The origin of the epidermal lamellae (**e**).

• • • Area 3: The area where the horny lamellae (**c**) start and where a fast increase in height takes place.

* * * Area 4: The area with only scanty increase in the heigth of the horny lamellae (**c**).

▼ ▼ ▼ Area 5: The area with almost no increase in height of the horny lamellae (**c**) and progressive synthesis of cap horn (**kc**).

▬ ▬ ▬ Area 6: The area near the wall–sole junction with horny tubules and large strings of cap horn (**kc**).
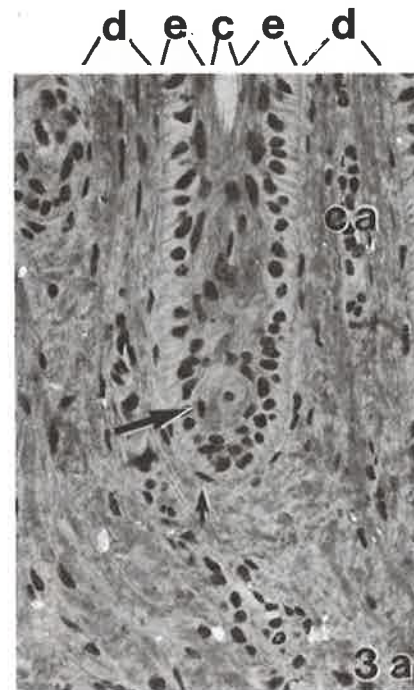
Fig. 3a. Normal keratinization at section C. Labels are explained in fig. 1. The stratum basale and spinosum of the epidermal lamella (**e**) appears regular and no vacuoles can be seen. A striation is seen in the basal parts of the basal cells. The large arrow points at a not fully keratinized horn iseland in the apical part of an epidermal lamella (**e**). The small arrow points at one of several cells, presumably fibroblasts, lying in the subbasement membrane zone. Bull, 11 month, left front medial digit (HE, historesin embedded, 4µ, 450 x).

The orientation of all the histological pictures are identical; the top of the pictures points dorsally and the bottom points toward the pedal bone.
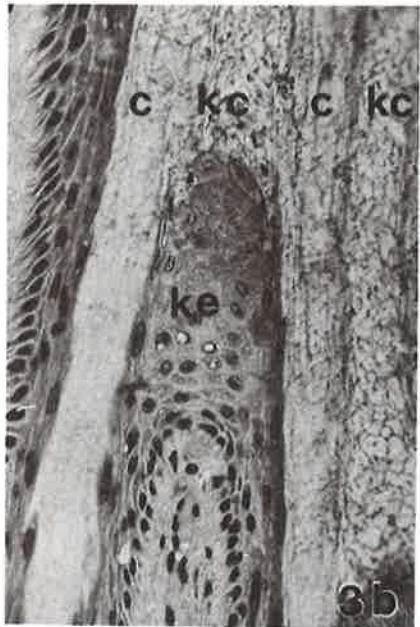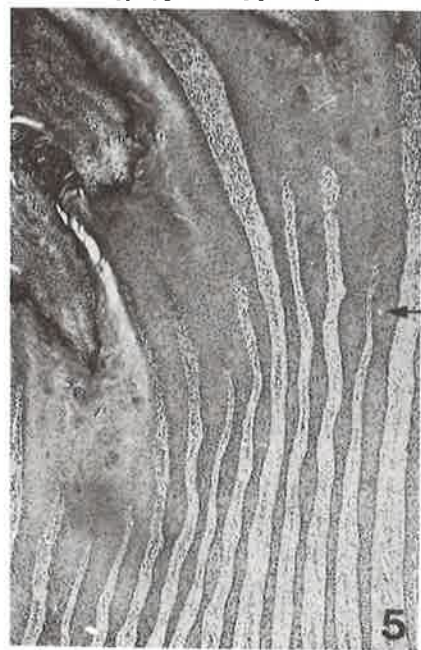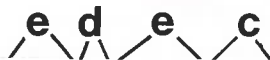
Figure 3b

Figure 4a
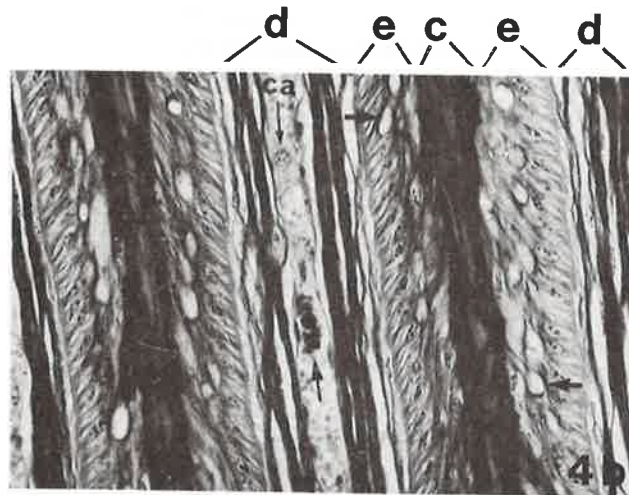
Figure 4b

Figure 5

Studies of Hoof Horn

A. M. Johnston

Royal Veterinary College, University of London

Investigation of disease(s) affecting the digit has for many years relied on subjective assessment. This follows cleaning of the digit with water and a variable amount of trimming with a hoof knife to reveal any defects or lesions present. As early as 1921, Smith discussed methods of assessing expansion and compression of the horse hoof wall. The former was measured with an apparatus which was said to register from 1/350th to 1/25th of an inch, and the latter by cutting a piece of the wall away which would accept a penny when the foot was off the ground but not with the foot weight-bearing. Many authors report on studies of lameness in dairy herds over varying periods of time (Eddy and Scott,1980) and assessments of claw disorders related to claw measurement, including the shape of the claw (Smit and others, 1986). Measurements of hoof angle and length, heel depth and area of hoof base were described by Hahn and his co-workers (1984a,b) from a study of Holstein cattle. These and measurements using force impression plate and video tape methods (Webb and Clarke, 1981; Applegate and others, 1988) have given basic information on hoof shape, interaction between foot and floor and effect on incidence of lesions and defects.

Following reports of the reduction of foot problems in sows and gilts (Brooks and Simmons, 1980; Penny and others, 1980) by supplementing diets with biotin, replicate trials were started at the Royal Veterinary College to study the effect on the hoof of the growing pig from 20 kg to pork and heavy hog weights. During these trials, despite a reduction in the defects and lesions in the biotin-supplemented group no evidence was found of a difference in growth and wear rate (Johnson and Penny, 1989). A significant finding was the relationship of weight to incidence of defects and lesions, with a significantly greater increase in the unsupplemented group from 70kg bodyweight upwards. By the end of each trial the wall of the unsupplemented group appeared to splay more than in the supplemented group and a significant difference was found when measurement of the sole of each claw was made. By pork weight the mean surface area of the sole of the biotin-supplemented group was 16.42cm^2 compared to 19.03cm^2 (p < 0.02) in the unsupplemented group.

Measurement of Horn Hardness

The spreading out of the distal wall, with widening of the white line, cannot be entirely explained by the wall being less hard than that of the biotin-supplemented group. Webb and others (1984) used a Shore D meter, an instrument used in industry to measure hardness of various substances such as rubber, leather and plastics, to determine the hardness of the hoof wall horn. The Shore D meter is suitable for use as a hand-held instrument on the hoof of the pig,cow and horse but is only suitable for use on hoof wall which is reasonably smooth, with minimal convexity and free from the vertical lines often seen on horses' hoof horn. With very poor hoof horn it is not possible to obtain reliable values as the wall crumbles under the meter probe. Using a Shore D meter, the effect of supplementary biotin on hoof hardness of the lactating dairy cow was studied over a period of five months of supplementation. During the first three months of the trial when the cows were loose-housed in cubicles, without the use of foot baths, there was an increase in hardness (unpublished findings).

Light and Electron Microscopy

In initial studies, when biopsy samples of pig hoof horn from the mid-wall were examined under light microscopy, the findings were unremarkable. When the wall sample included obvious defects they were merely observed in greater detail on microscopy. However, systematic study of the hoof produced more interesting results.

For the purpose of the study, the digit was divided into regions, with the proximal third which included the coronary band called 'distal + 2', the middle third which included the laminar corium was called 'distal +1' and the distal third incorporating part of the sole referred to as the 'distal region'.

On reaching pork weight the pigs were slaughtered and the hooves removed, after slaughter but before insertion in the scald tank, by sawing at the mid-pastern region. Two vertical cuts were made, 1cm apart, with a vibrating circular saw, from the hairy skin proximal to the coronary band to the palmar surface. Two equidistant horizontal cuts were then made to provide three blocks of tissue incorporating the full length of the wall. These blocks were carefully removed from the digit with a scalpel leaving as much of the underlying soft tissue as possible attached to the horn, and processed for light or electron microscopy.

On light microscopy a plastic section of hoof wall from a pig on the unsupplemented ration, stained with toluidine blue showed fragmented horn, lack of organisation within the epidermis and a different staining reaction in the horn. A plastic section from the hoof of a pig which had been given supplementary biotin, again stained with toluidine blue, showed well-organised structures within the horn and a more uniform staining of the horn. In sections from the distal +2 region, "holes" were found within the epidermis. In sections from the pigs fed the normal ration few holes were found within the epidermis and the horn looked ragged. When compared to sections from the pigs which had received the biotin supplement, the holes were very obvious within the epidermis, with strong horn and well organised structures.

With the scanning electron microscope using the same magnification the difference between the groups was more marked. When supplementary biotin had been given, large spherical areas with a distinct boundary, were seen within the epidermis along with smaller holes, well-defined papillae ,and structured horn: when no biotin supplement was given the horn was clearly less well organised with a few small holes.

The "holes" are circular to oval in shape, increasing in number towards the cornified layer, of two sizes: one of 9.5 μm diameter, spherical in cross-section with a fibrous-like band surrounding the named "spherical bodies": the other of 1.4 μm diameter arranged in rows linked by strands of tissue. The spherical bodies and holes in the biotin-supplemented group were more numerous, more distinct, more clearly-defined structures as was the fibrous band.

The tubular horn of the biotin-supplemented group had a greater number of tubules of more cohesive and better defined structure. It was calculated that the mean number of tubules in the biotin group was 144 per sq mm and the mean of the control group was 96 per sq mm. The part of the laminar horn produced by the epidermis devoid of tubules was relatively small, being 100 μm wide in the biotin group compared to   144 μm wide in the unsupplemented group and was seen as not so tightly packed and cohesive as in the horn produced by the pigs receiving the biotin supplement.

Using transmission electron microscopy, differences between the groups were also found. The basal layers of the epidermis of horn produced by the pigs given supplementary biotin had better defined papillae with active nuclei, columnar shaped basal cells and obvious amounts of the perinuclear cytoplasm necessary for good fibre and matrix production. The process of degeneration of epidermal cell organelles was erratic and less organised during the process of keratinisation in the unsupplemented pigs. When biotin was added, the resulting horn showed large numbers of circular areas bounded by keratin fibres which at high power showed the boundary to be lined with electron-dense material plus keratin fibres enclosing electron-translucent material with scattered particulate matter.

Transmission electron micrographs of the horn adjacent to the epidermis showed loose packing of the keratin fibres and lack of complete degeneration of cell organelles. The area adjacent to the epidermis in the biotin-supplemented group had the keratin fibres tightly packed together and the squames very cohesive. This tighter, more cohesive matrix most certainly accounts for the differences between the groups of width of non-tubular horn and number of tubules per sq. mm. These findings help to explain why the wall is harder in terms of Shore D units following biotin supplementation and there were significant differences in the area of the sole ($p < 0.02$) between the groups as a result of the wall splaying outwards.

Subsequent studies of hooves from pig units where a problem with poor hoof horn had been established showed a more marked variation in findings than described above. In samples of horn from the pigs with "bad" hooves the inter-squame membranes were separated by large intercellular spaces containing membranous material with the adjacent squame membranes only closely apposed in the region of the desmosomal remnants. The keratin fibres were not as well organised or as dense as had been found in samples from good hoof horn where the squames contained densely packed bundles of keratin fibres. Where there was a horn matrix of poor integrity with the squames not closely aligned, accumulations of red blood cells and also anaerobic bacteria have been found. A similar picture has been found on samples from cow hoof horn.

The stresses and strains on the hoof wall during locomotion or when standing tend to spread the wall. Indeed, some flexibility of the wall is essential fof goog hoof function particularly towards the heel. The mechanical attributes of keratinised tissue are determined by the properties, proportions, arrangements and cross-linking of the constituent proteins in the keratins and squames and by cell arrangement and adhesion. The presence of a larger number of well defined "spherical bodies" indicates a more active and organised epidermis. This is reflected in the structure of the adjacent horn which presented a well-differentiated, dense structure in samples of good hoof horn. The remnants of spherical bodies in the samples from hooves of poor horn indicate inadequate keratinisation had occurred. The possible biological significance is that the incorporation of this matrix tends to distribute any applied stress evenly over the filaments, preventing the propagation of cracks. This will all lead to softer, weaker horn and goes some way to explaining the differences in defects and lesions between populations with good hoof horn and those with poorer horn.

References

Applegate A.L., Curtis S.E., Groppel J.L., Macfarlane J.N. Widowski T.M. (1988) Journal of Animal Science **66**, 334-341.

Brooks P.H. Simmins P.H. (1980) Veterinary Record 107, 430.

Eddy R.G. Scott C.P. (1980) Veterinary Record **106**, 140-144.

Hahn M.V., McDaniel B.T. Wilk J.C. (1984a) Journal of Dairy Science **67**, 29-236.

Hahn, M.V., McDaniel, B. T. Wilk, J.C. (1984b) Journal of dairy science **67** 2986-2998

Johnston, A. M.., Penny, R. H. C. (1989) Veterinary Record **125** 130-132

Penny R.H.C., Cameron R.D.A., Johnson S., Kenyon P.J., Smith H.A., Bell A.W.P., Cole J.P.L. Taylor J. (1980) Veterinary Record **107**, 350.

Smit H., Perbeek B., Peterse D.J., Janson J., McDaniel B.T. and Politiek R.D. (1986) Livestock Production Science **15**, 205-217.

Smith F. (1921) In: A Manual of Veterinary Physiology. 5th edition, Balliere, Tindall and Cox.

Webb N.G. Clarke M. (1981) Farm Building Progress **66**, 23-26.

Webb N.G., Penny R.H.C. Johnston A.M. (1984) Veterinary Record **114**, 185.

Behaviour and Welfare Aspects of Cattle Lameness in Relation to Building Design

M. J. Potter* and D.M. Broom**

* *RSPCA, Causeway, Horsham, West Sussex, RH12 1HG*
** *Department of Clinical Veterinary Medicine, University of Cambridge, Madingley Road, Cambridge, CB3 OES.*

Abstract

Lameness as a welfare problem is discussed in terms of pain, longer term effects on physiology and behaviour. Referring to a previous detailed study by the authors of the behaviour of Friesian cows housed in a commercial farm's cubicle system and other available literature, the importance of careful attention to both building design and to social factors within the herd are stressed. By considering lameness within the context of the five freedoms regarded as essential for the well being of an animal, it is concluded that lameness can have widespread adverse effects on the welfare of that animal.

Introduction

The major part of this symposium concentrates on causation, clinical aspects and economic considerations of lameness. Although this paper is mainly devoted to the importance of building design and behavioural factors, it also considers the question of the magnitude of the effects of lameness on the welfare of the individual animal. It is widely accepted that lameness is a major problem in housed dairy cows. How serious is it for the individual animal? In what ways will it affect the well being of that animal? If welfare is defined as the state of the animal as regards it's attempts to cope with it's environment, how is it best assessed? (Broom 1988).

An animal that is lame might be experiencing severe pain; longer term physiological responses, walking and other movements might be seriously affected; and less obvious but equally important, other behaviours might be disrupted.

Pain

That pain is an integral factor associated with lameness would appear to be self evident truth. However, the assessment of pain is far from simple since as Fraser and Broom (1990) point out, in certain situations it might be wholly disadvantageous for an animal to display signs of pain. For lame cattle, indications of pain are obvious in the changed gait of the animal, and it seems reasonable to assume that the greater this disruption to normal movement is, the more intense the pain is likely to be. The degree of pain, however, remains largely unknown. Various techniques such as measuring the effects of administering analgesics, or even direct recording such as from the sensory nerves from pain receptors, might provide some information about pain level. Morton and Griffiths (1985) propose that various bodily and behavioural signs can usefully be given scores so that an accumulated score of pain level can be obtained.

Whether such information would help resolve the practical problems of preventing lameness is doubtful, but in the absence of such information, we should err on the side of caution and assume that pain might be severe even when behavioural and clinical signs are limited.

Long Term Indicators of Poor Welfare.

When adverse conditions last for an extended period, other responses occur which necessitate the use of different measures. Poor growth, inadequate reproductive function, poor lactation or maternal behaviour, and high incidence of disease can all indicate poor welfare. Elevated glucocorticoid production, although difficult to interpret, has been shown to be linked with stress in cattle (Ladewig 1984; Dantzer et al, 1983)

.Increased adrenal activity can also have a secondary effect on animals in suppressing the functioning of certain aspects of the immune system (Fraser and Broom 1990). Lameness, therefore, might directly result in an individual being less good at combating disease.

Effects on Behaviour

The most obvious indicator of lameness is clearly its effect on walking. Manson and Leaver (1988) used a system for scoring locomotion as a sensitive method of assessing the prevalence, severity and duration of lameness. However, it is the secondary effects of a reduced ability to walk that are important in welfare terms. These will depend on the environment in which the individual lives. Both the social structure within the group and building design will be important factors to consider.

Importance of Building Design

a) Number of feeding places

In a study of the behaviour of Friesian cows housed in a commercial farm's cubicle system, Potter and Broom (1987) found that many high ranking animals demonstrated a strong preference to fed at a particular section of the feed barrier. Since these preferred sections were often at the far ends of the barrier, it was suggested that this might indicate a mutual repulsion of the most dominant animals. It is possible that all cows might have a preferred feeding area, but higher ranks are better able to maintain their position. Data on individual feeding times in this study showed that low ranking cows spent a slight, but not significantly lower proportion of time feeding. It was concluded that a long feed barrier which allows all cows to fed simultaneously is a feature of good building design.

Metz (1983) reported a dramatic increase in chasing when the number of feed places was reduced. Therefore, if there are insufficient feeding places, due to the highly synchronised behaviour of dairy cows (Benham, 1982; Wierenga, 1983; Potter and Broom, 1987) there are considerable effects on the cows. It is likely,

therefore, that the welfare of cows is poor when they are unable to get to a feeding place because their herd-mates are feeding.

b) Design of passageways

The dimensions of various parts of cubicle systems have been shown to dramatically influence the freedom of movement of cows. For example, Konggaard (1983) saw more contact yielding, turning and waiting if passageways were 1.2m wide than if they were 2m wide. Potter and Broom (1987) demonstrated that when housing system design allows animals to synchronise their activities, the total amount of cattle movement can be very low and also results in a tendency for a one-way flow of cows through passageways. For lame individuals, this means that the total amount of walking is low and the risk of head to head confrontations is reduced.

c) Number of cubicles

An inadequate number of cubicles, such that not all cows can lie down at once, leads to more aggressive interactions and low-ranking animals having to lie in passageways where conditions are dirty and likelihood of disease and injury is high (Kaiser and Lippitz, 1974; Friend et al., 1977; Wierenga, 1987). Potter and Broom (1987) also found that low ranking individuals spent a greater proportion of their total cubicle time standing, and suggested that this demonstrated a dual function for cubicles, (i) as a lying place, (ii) as a zone where effective personal distance is increased by the bars of the cubicle. This was supported by the finding that low ranking cows spent almost three times as much standing in the cubicle passage with head and front legs in a cubicle than did other cows. If space in a housing system is limited, it might be beneficial to increase the number of cubicles at the expense of floor space to provide these safety zones for low ranking animals.

d) Social factors

The effects of house design will not only have profound effects on social behaviour but will interact with factors such as the age structure of the group. and the frequency of movements of animals into the group. Eddy (1989) suggests that the incidence of sole ulcers is particularly high among bought-in heifers because such animals spend more time standing and do not feel confident enough to lie with the rest of the herd. Social mixing has considerable effects on behaviour and milk production (Arave et al, 1974). It can also influence the incidence of lameness. Many injuries to the feet are the result of falls, slips and abrasion on concrete or slatted floors. Such mechanical damage might frequently result from competitive social interactions in which animals engage in pushing contests or when one animal moves rapidly to avoid an aggressor. Considerate herd management and careful housing design therefore, can reduce levels of social stress and competitive interaction. Stress itself can also influence lameness directly, for example, by increasing the susceptibility of cattle to metabolic disorders of the foot (Peterse, 1987).

82

Conclusion

Welfare must be considered in terms of the state of the individual, and a useful framework when considering it is provided by the five freedoms defined by the Farm Animal Welfare Council (1983). These are, freedom from: hunger and malnutrition, thermal and physical discomfort, injury and disease, suppression of 'normal behaviour', and fear and stress.

Lameness cannot be thought of only in terms of injury and disease. It can influence each of these five freedoms and therefore can have very serious welfare implications for the individual. Attention to both building design and social factors within the herd is vital in both the control of lameness and in reducing its impact on the individual.

References

Arave, C.W., Albright, J.L. Sinclair, C.L. (1974). Behaviour, milk yield and leucocytes of dairy cows in reduced space and isolation. J. Dairy Sci., 57, 1497-1501.

Benham, P.F.J. (1982). Synchronisation of behaviour in grazing cattle. Appl. Anim. Ethol., 8, 403-404.

Broom, D.M. (988.) The Scientific assessment of animal welfare. Appl. Anim. Behav. Sci., 20, 5-19.

Dantzer, R., Mormede, P., Bluthe, R.M. Soissons, T.(1983) The effect of different housing conditions on behavioural and adrenocortical reactions in veal calves. Reprod. Nutr. Develop., 23, 67-74.

Eddy, R.(1989). In: 'Lameness', Farmers Weekly, 26th May 1989, 58-59.

Farm Animal Welfare Council. 1983. Code of recommendations for the welfare of cattle. HMSO, London.

Fraser, A.F. Broom, D.M. (1990). Farm Animal Behaviour and Welfare. London Balliere Tindall .

Friend, T.H., Polan, C.E. McGilliard, M.L. (1977). Free stall and free bunk requirements relative to behaviour, production and individual feed intake in dairy cows. J. Dairy Sci., 60, 108-116.

Kaiser, R, Lippitz, O. (1974). Untersuchungen zum Verhalten von Milchkuhen im Boxen laufstall bei unterschiedlichem Tier-Liegeplatz-Verhaltniss und standig freiem Zugang zur reduzierten Krippe. Tierzucht, 28, 187-189.

Konggaard, S.P.(1983). Feeding conditions in relation to welfare for dairy cows in loose-housed conditions. In: Farm Animal Housing and Welfare (Ed. S.H.Baxter, M.R. Baxter J.A.C.MacCormack. Curr. Top. vet. Med. Anim. Sci., 24, 272-278. The Hague Martinus Nijhoff:

Ladewig, J. (1984). The effect of behavioural stress on the episodic release and circadian variation of cortisol in bulls. In Proc. Int. Cong. Appl. Ethol. Farm

83

Anim. ed. J. Unshelm, G. van Putten and K. Zeeb. 339-342 Darmstadt: K.T.B.L..

Manson, F.J. Leaver, J.D. (1988). The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Anim. Prod., **47**, 185-190.

Metz, J.H.M. (1983.) Food competition in cattle. In: Farm Animal Housing and Welfare (Ed. S.H. Baxter, M.R. Baxter and J.A.C.MacCormack) Curr. Top. vet. Med. Anim. Sci., **24**, 164-170. (Martinus Nijhoff: The Hague).

Morton, D.B. Griffiths, P.H.M. (1985). Guidelines on the recognition of pain, distress and discomfort in experimental animals and an hypothesis for assessment. Vet. Rec., **116**, 431-436.

Peterse, D.J. (1987). Aetiology of claw disorders in dairy cattle. In: Cattle Housing Systems, Lameness and Behaviour (Ed. H.K. Wierenga and D.J. Peterse). Curr. Top. vet. Med. Anim. Sci., **40**, 3-13.

Potter, M.J. Broom, D.M. (1987). The behaviour and welfare of cows in relation to cubicle house design. In: Cattle Housing Systems, Lameness and Behaviour Ed. H.K. Wierenga and D.J. Peterse. Curr. Top. vet. Med. Anim. Sci., **40**, 129-147. (Martinus Nijhoff: Dordrecht).

Wierenga, H.K. (1983) The influence of space for walking and lying in a cubicle system on the behaviour of dairy cattle. In: Farm Animal Housing and Welfare Ed. S.H. Baxter, M.R. Baxter and J.A.C. MacCormack. Curr. Top. vet. Med. Anim. Sci., **24**, 171-180. The Hague Martinus Nijhoff:

Note on Welfare of Dairy Cows with Reference to Spatial and Comfort Aspects of Design of Cubicles

Jan Cermak

ADAS Welsh Office Agriculture Department, Gabalfa, Cardiff

Background

Housed dairy cows require space for resting, feeding, drinking and exercise. In dairy buildings, the space which is available to animals is often arranged to primarily aid technology of handling feed and waste products and to streamline movement of animals. This often results in the linear layout of resting and feeding areas. In pursuit of acceptable costs of buildings, the building designers derive layout solutions which often promote rather than minimise competition for space. In particular the space is usually constricted around prime locations in the unit, for example clear ways between rows of cubicles, water troughs, mangers and access to collecting yards and milking parlour.

Underlying level of social aggression, promoted by social hierarchy of animals and the competitive use of space can often result in sudden avoiding actions, when animals slip and fall on slurry-covered surfaces.(1). In small cubicles, overloading of joints can occur as animals struggle to get up or lie down. Poorly designed cubicle units can be particularly damaging to cows.

This note attempts to highlight that adequate provision of space as well as provision of a dry lying area for animals is an essential part of any therapeutic remedy which may be prescribed in prevention and control of bovine foot lameness. No account of dimensional analysis of cows' requirements for space in cubicles, and in particular its relationship with the body weight is presented as it has been adequately covered elsewhere (2).

The hypothesis is that should cows lie longer in cubicles, their exposure to slurry deposited in passages will be subsequently shorter and this in turn should reduce the environmental challenge to the foot and also any likelihood of falls on slippery concrete surfaces should be reduced.

Cow Space Envelope - Zoocentric Approach

Casual observation of cow rising behaviour may not clearly highlight a series of body actions which the animal needs to execute in order to complete rising or lying movements. Figure 1 shows an outline of the number of steps, comprising a shift of the centre of gravity during the rising movement. It also indicates the forward space demand which is within the range of 0.7 to 1.1 metre for an 600 kg Friesian dairy cow.

Figure 1 : Forward space demand of rising movement (600 kg Friesian dairy cow)
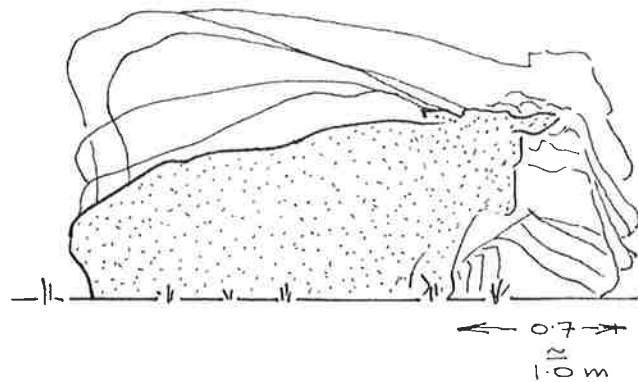
It is important that the length of the cubicle base should allow the cow to find enough forward space to accomplish the rising movement. To some extent, when provision of "space-sharing" partitions (i.e. those designs where the cow can insert the head and the neck through the partition and thus share space with the neighbouring cubicle) are installed, the overall length of the cubicle bed could be reduced by 80-120 mm over the recommended dimensions given in Table 1 (2) Figure 2 shows this arrangement.

Table 1 : Relationship between chest girth, body weight, diagonal body length and the cubicle length

Cow Body weight (Kg)	Chest Girth (m)	Body Length (m)	Cubical Length (m)
375	1.68	1.36	2.00
425	1.75	1.41	2.04
475	1.81	1.46	2.08
525	1.87	1.50	2.12
575	1.93	1.54	2.16
625	1.98	1.58	2.20
675	2.04	1.62	2.24
725	2.09	1.65	2.28
775	2.14	1.68	2.30
825	2.18	1.72	2.33

Practical application of this arrangement, particularly in re-furbished dairy units when the space is often a limiting factor, has been confirmed on a number of dairy units in practice.

Figure 2 : Benefit of a space-sharing partition.
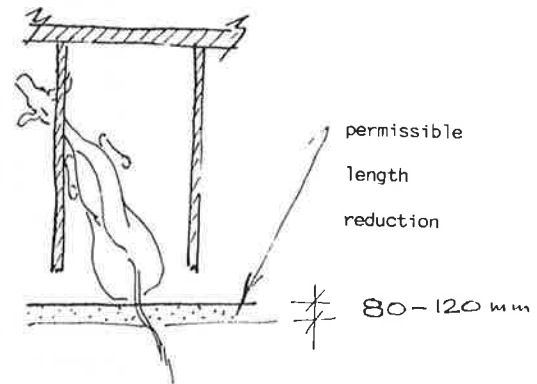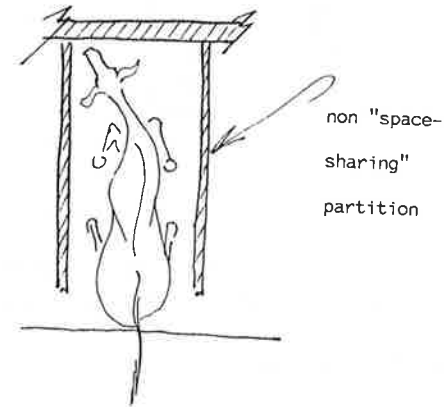
The "space-sharing" cubicle partition should provide three zones of free space. Figure 3 gives an outline of the head, ribcage and pelvis zone around the animal's body which should be free from obstruction.

Figure 3 : Outline of free space required to meet cows' lateral spatial needs
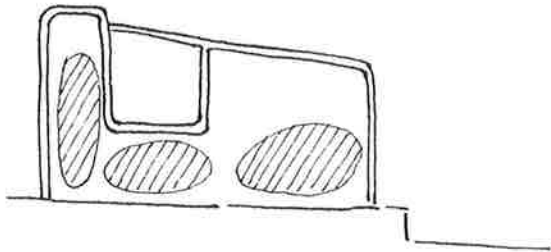
As shown in figure 4, the cow will occupy a given cubicle space in a more "fluid fashion" than would otherwise be allowed by a solid wall, "non-permeable" partition.

Figure 4 : Space sharing between partitions
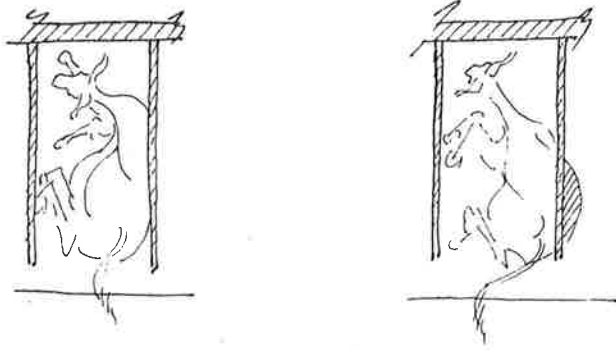
There are a number of cubicle partitions on the market. The selection should therefore be based on to what extent various types meet this design criterion. It is therefore of no consequence whether the partition is constructed from steel, timber or plastic.

Comfortable Cubicle Base

Cows prefer dry a resilient lying area to hard surfaces, for example bare concrete cubicle bases. There are several types of rubber or plastic-based cow mattresses which can be placed on top of the concrete beds. It has been found that the provision of mattresses does lead to increased lying time. Only some mattresses however would

equal the cow lying time (approximately 14 hours) associated with the provision of some 50-80 cm of chopped straw over a gently sloped concrete cubicle bed (75 mm height difference). A novel approach to provision of dry resilient cubicle beds has been successfully tested on many Michigan dairy unit in the U.S.A. and also over the last two housing seasons on the new dairy unit at Sonning Farm, University of Reading. (3) Figure 5 shows a diagrammatic sequence of operations of construction of a straw-filled cow mattress using a geo-textile mat (Geolon 40 and 80 by Nicolon Ltd). The cost of materials for the complete installation amounts to £5.60 to £8.00 per cubicle, depending on the type of geo-textile mat used (1988 prices). Albeit, the mattress is slightly more difficult to clean, its porous properties provide a drier surface than some other types. Like all mattresses and cubicle beds, it is necessary to remove dung pads daily.

Figure 5 : Construction sequence of geo-textile cow mattress.

Geo-textile mat   straw
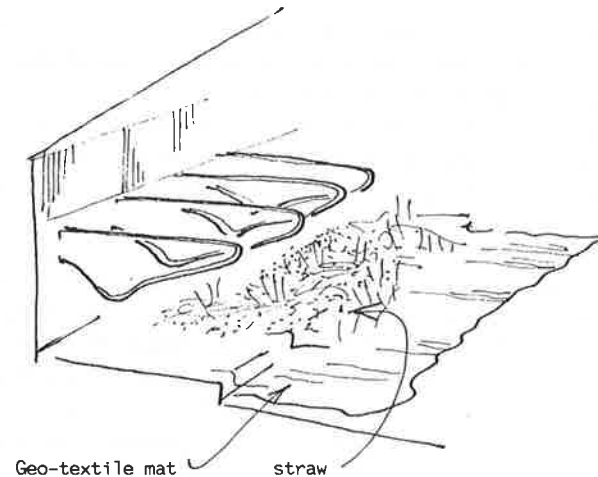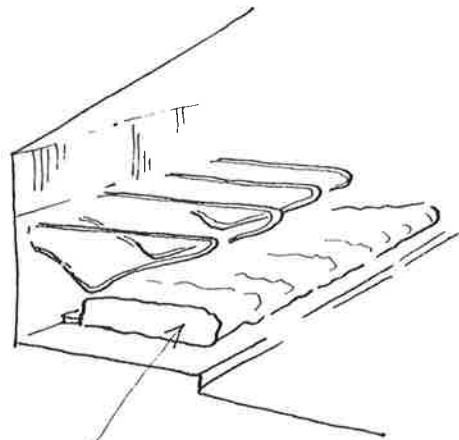
mat rolled over

Conclusion

Welfare of dairy cows housed in cubicle units, particularly with reference to the risk of lameness can be enhanced by the provision of ample space in cubicles and by provision of a comfortable resilient dry lying area.

An outline of principles governing the use of space in cubicles and the need for "space-sharing" designs of cubicle partitions is presented. A reference is made to a novel approach to the construction of a low-cost cow mattress for use in cubicles.

References

1) Potter M.J. and Broom D.M. (1987). The behaviour and welfare of cows in relation to cubicle house design. In : Cattle Housing Systems, Lameness and Behaviour (Ed Wierenza H.K. and Peterse D.J.), Martius Nijhoff Publishers, 129-147.
2) Cermak J.P. et al. Design and management of cubicles for dairy cows. Ministry of Agriculture, Fisheries and Food Booklet, 1983.
3) Dr. W. Bickert. Michigan State University U.S.A., Personal communication.

Housing, Lameness and Etho-pathological Studies in Cattle

H.K. Wierenga*, E. ter Wee*, A.C. Smits** and S.A. Goedegebuure***

* Research Institute for Animal Production "Schoonoord", P.O. Box 501, 3700 AM Zeist, The Netherlands.
** Institute for Agricultural Engineering, P.O. Box 43, 6700 AA Wageningen, The Netherlands.
*** Department of Veterinary Pathology, University of Utrecht, P.O. Box80.157, 3508 TD Utrecht, The Netherlands.

Abstract

The relation between housing design, construction of floors and lameness in cattle is discussed, particularly the behavioural interaction with housing systems.

In fattening bulls and veal calves osteochondrosis (damage of the cartilage) of the carpal joints is mainly due to the fast growth of the animals, but housing on a hard and slippery floor has an additional effect. Recently investigations have been started on the effect of providing more comfortable floors, behaviour patterns and incidence of osteochondrosis.

In dairy cattle lameness, claw and leg disorders may be caused by hard and slippery floors, uneven surfaces (inc. slats), and restricted freedom of movement when standing up or lying down. Investigations on these topics should be initiated, and more detailed studies into the relation between housing system, floor and lameness are needed. In these studies, behavioural and veterinary investigations - etho-pathology- should be combined.

Introduction

Lameness in cattle is the result of an interaction between housing design, farm management and the animals (Wierenga and Peterse, 1987). The aim of this paper is to discuss briefly the role of housing design and, in particular, the design and construction of the floor with the occurrence of lameness in fattening bulls, veal calves and dairy cows. In particular the importance of behavioural studies will be discussed. Only a limited amount of our own experimental data will be mentioned; mainly we will refer to available literature.

Lameness in fattening bulls

In the Netherlands and in several other European countries, fattening bulls are kept in small groups on a fully slatted floor. The fast growth of these animals and their being housed on a hard and slippery floor results in osteochondrosis (Dammrich, 1974; Seibel et al., 1973). Osteochondrosis - which is a damage of the cartilage of the joints and the growth plates of the bones - occurs mainly in the fore legs (Siebel et al., 1973) and is presumed to cause animals pain (Vries et al., 1986). A slippery floor

causes animals to move carefully. Ming (1984) has shown that animals on a slatted floor regularly slip, particularly when they perform comfort behaviour. The hard floor makes the animals reluctant to lie down and they try to lie down carefully (Andreae 1979). Dammrich (1987) suggested that careful movements to prevent slipping or during lying down or standing up, could result in an unequal loading or an overloading of the joints, and contribute to the development of osteochondrosis.

When we used a scoring system for osteochondrosis, valued 0 (no lesions) to 3 (severe lesions), we found a mean score of between 1.9 and 2.0 for six month old fattening bulls kept on a half concrete half wooden slatted floor, and on a fully concrete slatted floor (Table 1).

Table 1 Mean score for damage of the carpal joints of 6 months old group-housed fattening bulls.

		mean score (0-3)
¹/₂ concrete, ¹/₂ wooden slatted floor (n=12)	left carpus	2.0
	right carpus	1.9
fully wooden slatted floor (n=12)	left carpus	1.9
	right carpus	2.0

A preliminary experiment was carried out which a plain concrete slatted floor was compared with a concrete slatted floor covered with rubber. Fattening bulls were kept on these floors from 6 - 16 months of age. At 16 months of age the animals were slaughtered and the carpal joints examined. Results showed (Table 2) that those animals which were kept on concrete up to 16 months of age had more severe lesions (mean score 2.7) than those animals at 6 months of age. Furthermore, these first results suggest that the rubber on top of the concrete slatted floor (less slippery and softer, deformable) resulted in less severe damages (mean score 2.1: Table 2).

Table 2 Mean score for damage of the carpal joints of 16 months old group-housed fattening bulls kept from an age of 6 months on a concrete slatted floor with or without rubber.

		mean score (0-3)
concrete (n=6)	left carpus	2.7
	right carpus	2.7
concrete + rubber (n=6)	left carpus	2.1
	right carpus	2.1

Between 7 months of age and slaughter, it was observed that bulls gradually developed abnormal behaviour when attempting to stand up (Wierenga 1987). This may be a direct effect of the hard floor and the reluctance of the bulls to lie down, partly; it may also be caused by developing osteochondrosis. It seems possible, that abnormal behaviour in lying down is less painful compared to lying down normally on the knees.

The results from various preliminary experiments have suggested an influence of the floor on the occurrence of osteochondrosis. Based on these results we started recently a large experiment with fattening bulls kept in groups. Bulls reared from birth/six days of age until slaughter at 16 months old on a hard and slippery floor (at first wood and later concrete) will be compared with bulls kept on a soft and less slippery slatted floor with rubber on top. A third experimental group of animals will be kept first on a wooden (0 - 3 months) and then on a concrete slatted floor (4 - 6 months) and only in the fattening period (7 - 16 months) will the concrete slatted floor be rubber covered. From rearing until slaughtering regular behavioural observations will be carried out. In particular, the slipping of the animals when performing comfort behaviour or when walking will be recorded. The carpal joints will be inspected after slaughter.

Veal calves and lameness

Almost no information is available about lameness in veal calves. Because veal calves are also fast growing animals and are often kept on wooden slatted floors (hard, slippery) , osteochondrosis might be expected. Indeed, inspections of the carpal joints of veal calves kept for various experiments on the experimental farm of the Research Institute for Animal Production showed damages comparable to fattening bulls of the same age (Table 3). No differences in lesions were found between calves fed only milk replacer and calves fed additional roughage, or only roughage. A group of calves kept on a slatted floor with rubber covering did not show a lower score for carpal joint

damage. No difference was recorded between calves kept individually (group 5b) and those kept in groups.

Table 3 Mean scores of damages to the cartilage of the carpal joints in veal calves at slaugether.

exper.	number of animals	age at slaughter (wks)	mean score (0-3)	remarks about housing or feeding
1	43	31	2.1	fed milk replacer and/or roughage
2	42	34	2.1	fed only roughage
3	69	26	2.1	idem group 1
4	15	26	2.3	idem group 1, but kept on concrete slatted floor covered with rubber
5a	20	24	2.1	out of groups of 30 calves
5b	20	24	2.1	kept individually in crates

Recently we have started an experiment comparing group-housed veal calves kept i) on a wooden slatted floor, ii) a concrete slatted floor with a rubber covering, iii) a synthetic slatted floor or iv) on two types of rubber mats simply placed on the wooden slatted floor. The behaviour of the calves, in particular slipping during comfort behaviour and walking, and standing up and lying down, will be recorded during the fattening period. The lesions of the carpal joints of the calves will be inspected at slaughter.

Lameness in dairy cattle affected by design and construction of the floor.

For dairy cows kept in cubicle houses, a direct interaction between the house and the animals may result in traumatic claw disorders and lesions of the integument (Wee et al., 1989); a wet floor in particular may result in infectious claw disorders. This later type of claw disorder - and also metabolic claw disorders - will be left out of our discussions.

It can be imagined that the floor is the most important factor in traumatic claw disorders. with slatted floors it is best to have narrow slats to prevent too high loading of the claws (Pfadler 1981; Boxberger 1982). Generally speaking an uneven floor surface could be expected to cause claw disorders, although no detailed information is available. Slippery hard floors are found in many modern dairy cattle houses,.as for the fattening bulls and the veal calves,this could cause problems for dairy cows. Sommer (1985) showed differences in the way that dairy cows walked on different floor types. It is not known if slippery floors also cause damage to the joints of dairy cattle, nor if such floors cause claw disorders. Investigations in this area are needed.
Besides the floor of the walking area in the cubicle house - or with dairy cattle housing in general - the lying area may also affect lameness. It is known (Kammer and Schnitzer 1975) that it is essential for cattle to have enough freedom of movement when they stand up or lie down. Cows which are tethered and cows which have to lie down in a cubicle, are in fact always hindered in their movements during lying down or standing up. When freedom of movement is restricted too much this could directly result in lameness.

There is little information available concerning lameness in dairy cattle as related to the design of the housing system. Detailed studies are needed in which behavioural and veterinary investigations (etho-pathology) should be carried out together.

Literature

Andreae U. (1979). Zur Aktivitatsfrequenz von Mastbullen bei Spaltenbodenhaltung. Landbauforschung Volkenrode, Sonderheft, **48**:89-94.

Boxberger J.(1982). Wichtige Verhaltensparameter von Kuhen als Grundlage zur Verbesserung der Stalleinrichtung. Forschungsbericht Agrartechnik, 80. TU, Weihenstephan.

Dammrich K. (1974). Adaptationskrankheiten des Bewegungsapparates bei Masttieren. Fortschritte der Veterinarmedizin, **20**: 69-80.

Dammricht K.,(1987). The reaction of the legs (bone; joints) to loading and the consequences for lameness. In : H.K. Wierenga and D.J. Peterse (eds.), Cattle housing systems, lameness and behaviour. Martinus Nijhoff Publ., Dordrecht, pp. 50-55.

Kammer P. und Schnitzer U., (1975). Die Stallbeurteilung am Beispiel des Ausruheverhaltens von Milchkuhen. KTBL- Schrift, Darmstadt.
Ming (1984). Untersuchungen uber die Eignung von Loch-und Spaltenboden in der Rindviehmast . EDTH, Zurich, Diplomarbeit.

Pfadler W (1981). Ermittlung optimaler Funktionsmasse von Spaltenboden in Milchviehlaufstallen. Forschungsbericht Agrartechnik,58. TU, Weihenstephan.

Seibel S ., Dammrich K. und Andreae U. (1973). Uberbelastungsschaden an der distalen Epiphysenfugenscheibe des Radius bei Mastbullen aus Verschiedenen Aufstallungsformen. Berl. Munchn. tierarztl. Wschr., 86:441-447.

Sommer T. (1985). Untersuchungen zur Tiergerechtheit praxisublichel Gestaltung von Laufflachen fur Milchvieh im Boxenlaufstal. Ethologische Station Hasli, Zoologisch Institut, Universitat Bern.

Vries F.P.W. de Wierenga H.K. en Goedegebuure S.A.(1986). Een orienterend onderzoek naar het voorkomen van afwijkingen aan het carpaal gewricht bij vleesstieren en naar het verband met de wijze van opstaan en gaan liggen. IVO - rapport B-278, 50p. (with English summary).

Wee .E. ter, Wierenga, H.K., Smits, A.C. and Smits, M.J.C., (1989). Claw and leg disorders in cattle in relation to the design and construction of floors. IVO "Schoonoord", Zeist, Report B-345.

Wierenga H.K. (1987). Behavioural problems in fattening bulls. In: M.C. Schlichting and D. Schmidt (eds), Welfare aspects of housing systems for veal calves and fattening bulls. Commission of European Communities, Brussels (EUR 10777-en), pp. 105-122.

Wierenga H.K. and Peterse D.J. (1987). Cattle housing systems, lameness and behaviour. Martinus Nijhoff Publ., Dordrecht.

Feeding Factors Associated with Lameness : a Preliminary Report from an Epidemiological Survey.

Manson F.J. and Merritt J.B.

University of Liverpool, Department of Veterinary Clinical Science, Leahurst, Neston, South Wirral. L64 7TE

Introduction

There is evidence from experimental work that lameness is a major predisposing cause of lameness. The feeding of large amounts of concentrate and in particular readily fermentable carbohydrates is thought to be one of the main risk factors of laminitis and laminitic related problems (Garner et al., 1975; Manson and Leaver, 1988). The concentrate to forage ratio of the diet is also important; work has shown that high concentrate to forage ratios give rise to a greater incidence of laminitis and solar ulcers (Livesey and Fleming, 1984) and also leads to the formation of poorer quality horn, (as measured by hoof hardness tests), which would make the hoof more susceptible to trauma (Manson and Leaver, 1989).

Feeding around calving may be of particular importance. Work by Peterse (1979) has shown that "steaming up" around calving increases the risk of sole lesions occurring. However, there is some speculation as to whether the most important factor is the level of concentrate fed or the length of time over which changes in feeding around calving are introduced. Trimberger et al (1972) found that rapid changes from a forage to a concentrate based diet led to an increased incidence of laminitis, whereas Peterse and van Vuuren (1984) showed that the rate of change from a low to a high concentrate ration had little effect on the incidence of solar lesions.

A high level of protein has been implicated as having a role in the development of lameness (Manson and Leaver 1988), although there are indications that protein source is of lesser importance (Logue et al., 1989).

Some workers have also suggested that laminitis is due to an allergic reaction to protein (Nilsson, 1966; Chew, 1972). Toxins of proteinaceous origin are thought to induce acute laminitis through their effects on the keratinous material of the hoof horn (Urmas, 1968). A high percentage of free ammonia in silage has also been found to be a feature of farms where a high incidence of lameness was observed (Bazeley and Pinsent, 1984). However, although experimental studies have shown a link between nutrition and lameness, survey work using "on - farm" situations has not been done to establish which of the nutritional influences known to be involved in the etiology of lameness are the most important. As part of an epidemiological survey being undertaken at the University of Liverpool to determine the risk factors involved in dairy cow lameness, feeding is one factor which is being monitored. In the final analysis a case - comparison model and a multiple regression analysis will be used, and high and low incidence herds will be compared as will cows with high and low incidences within farms. In this preliminary report some of the feeding factors will be discussed.

Materials and Methods

The survey is being conducted on 38 farms, 9 in the Wirral (North West England), 9 in Cheshire (North West England), 11 in Somerset (South West England) and 9 in Dyfed (Mid West Wales). All the cows are of Friesian-Holstein type, with the exception of a herd of Guernseys, and all cows are cubicle housed.

To monitor lameness the herdsperson, farmer or vet are asked to complete a foot recording form whenever a cow's foot is examined either for trimming or for treatment for lameness. On these forms the farm, cow, foot or limb involved, person examining the cow and date are identified. The site and extent and type of lesion are recorded pictorially on a diagram of the solar and side (wall) views. The person treating the cow is also asked to give an assessment of the severity of lameness (slight, moderate or severe), and of the condition of the claws (normal, overgrown, unequal size or corkscrew). The method of treatment and any other comments are also recorded. Additionally all the cows on 27 of the farms are locomotion scored monthly using the locomotion scoring system of Manson and Leaver (1989) as indicated elsewhere in this Congress (Clarkson, 1990) to give an independent assessment of lameness.

This paper will discuss feeding aspects, but environmental factors (concrete, slurry, cubicles, bedding and trackways) and breeding, milk production, fertility and stockmanship factors are also being studied in this survey.

Open questions rather than a standard questionaire format or closed questions are used. This approach is used so that answers simply consisting of ambiguous and often wrongly used terms do not occur. The researchers, using standard definitions then categorize the farmer's feeding policy into "flat rate" etc. Questions are asked to ascertain the following: amounts fed (per litre, to maintenance), rate of change of concentrate use over lactation, types of straights used and how they are fed (mixed diet, on top of silage etc.), frequency of feeding, types of forage used and how fed (self feed, easy feed etc.) and amounts of forage fed of known. At each monthly visit during the housing period follow up questions are asked to check if any changes in feeding have occurred. Additionally, the farmer is also asked to record on a form any deliveries of bought in feeds, and any changes in feeding.

Results

The result presented below were based on 36 farms.

The mean number of treated feet per 100 cows (subsequently referred to as incidence) over the feeding period considered in this paper (October 1989 to February 1990) was 23.4%, ranging between 2% and 100%: the means for the Wirral, Cheshire, Somerset and Dyfed areas were 22.7%, 30.8%, 26.8% and 12.3%, respectively. Low and high incidence herds were compared in relation to some of the different feeding methods. The herds were split into low incidence herds (less than 24%) and high

incidence herds (more than or equal to 24%).

The majority that is 23 herds (64%) were fed according to yield or a slight variation of that method, e.g. according to peak yield, and the other 13 (36%) were fed on a flat rate basis or on a 2 or 3 stepped system. There was no association between system of concentrate feeding and feet treatment incidence (X^2 test, p>0.05), although there was a slight tendency for herds fed according to yield to have a lower incidence:18 (78%) herds fed to yield were classed as low incidence, whereas only 7 (54%) of herds fed flat rate were classed as low incidence.

Sixteen (44%) herds were easy fed only (silage available in troughs), 11 (31%0 herds had access to silage in troughs and at the silage clamp, one herd was self feed only (silage available at silage clamp), 7 (19%) herds were offered a mixed diet (silage and straights mixed together), and one herd was fed a complete diet. The mean incidences for easy feed herds, easy and self feed herds, mixed feed herds and the complete diet herd were 18.2%, 17.4%, 46.1% and 18% respectively. There was no association between method of silage feeding and incidence (X^2, p>0.05).

Only 8 out of the 36 herds were fed all their concentrate ration in the parlour, the other herds either being fed a mixed or complete diet or offered a mid-day feed. The mean incidence for the 8 herds was 20.4% and 24.3% for the other herds. No significant association was found between the 8 herds and the other herds with respect to incidence (X^2 test, p>0.05). Similarly, when the proportion of Metabolisable Energy (ME) supplied by the straights and /or parlour cake fed in the parlour was compared with the proportion fed outside the parlour (less than or equal to 50% ME supplied in the parlour versus more than 50% ME supplied in the parlour) no significant association was found with incidence (X^2 test, p>0.05).

Four herds were fed a sodium bicarbonate supplement and the mean level of feet incidence (5.0%) was considerably less than those herds where no supplement was fed (25.7%).

Types of lesion such as sole ulcers and laminitic lesions, which are thought to be of metabolic origin were also examined in relation to high and low incidence. It was found that solar ulcers and laminitic lesions accounted for on average 38% of all foot lesions over all herds. In the high incidence group they accounted for 43% of all lesions, whereas in the low incidence group they accounted for 34%. No significant association (X^2 test, p>0.05) was found between incidence and the proportion of solar ulcer or laminitic lesions found in a herd (less than 38% lesions of solar ulcer/laminitic type versus more than 38%)

There were no significant associations (X^2 test, p>0.05) between incidence with system of concentrate feeding nor with proportion of ME fed in parlour.

Discussion

The tendency for herds fed to yield to have lower levels of incidence is in contrast to previous work by Webster and Leaver (1983), who observed that lower locomotion scores (i.e. less lameness) were found in cows on a flat rate system of feeding compared to a yield system. However, differences between the two studies in methodology (theirs was conducted under controlled conditions) may have explained any discrepancy.

The association of sodium bicarbonate supplementation with low incidences supports suggestions that the addition of this supplement to the ration stabilises rumen pH (Weaver, 1979) and therefore decreases the likelihood of acidosis from occurring (Kaufmann, 1976), which is related to the laminitis syndrome (Peterse, 1985)

The finding that the percentage of solar ulcers and laminitic lesions (38%) was substantially greater than 14% as previously reported in a major survey conducted in 1977 (Russell et al. 1982) may indicate to some extent the increasing importance of nutrition as regards lameness, since these lesions are thought to be primarily of metabolic origin (Edwards, 1982). However, caution should be taken in comparing data from the two surveys since different lameness recording procedures were used.

The lack of association between incidence and general frequency of concentrate intake, as indicated by proportion of ME fed in the parlour and method of concentrate/straights feeding (in parlour, mid-day feed, mixed diet etc.) may initially seem surprising as there is evidence that a higher feeding frequency of concentrates results in a more constant and lower decrease of pH in the rumen (Kaufmann, 1976), with a resulting decrease in the level of metabolic upsets.

The findings that frequency of concentrate feeding and silage availability did not appear to be risk factors in lameness over the study period reported on (October 1989 to February 1990) may initially appear surprising. However, the relatively short period of 5 months from which the data in this preliminary report was taken may explain the absence of a relationship, since the time taken for the cause (feed intake) to produce an effect (a severity of lameness in a cow that a stockperson would consider worthwhile examining and treating) may take several months.

Another explanation for the apparent lack of a relationship between feeding and lameness is the lack of extreme feeding practices found on commercial farms, and in particular under a quota system, whereas diets fed to cattle under experimental conditions are more likely to be extreme. Additionally, other uncontrolled variables which may affect the incidence of lameness (environmental factors, breeding policy, hoof trimming policy etc.) and which would have altered slightly from farm to farm, would unlikely to have balanced each other out. Thus the feeding factors would probably have been altered in slightly different ways by the other risk factors on each farm.

On completion of this work a multiple regression analysis will be used so that the confounding effects of one or more variables can be removed, and so that the different risk factors can be ranked in order of their influence on lameness. Such a ranking would allow a prioritization of alterations to farm management practices which would reduce lameness; and this exemplifies one of the benefits of a survey type approach to lameness as compared to a purely experimental approach.

References.

Bazeley, K. Pinsent, P.J.N. 1984. Preliminary observations on a series of outbreaks of acute laminitis in cattle. Veterinary Record **115**, 619.

Chew, K.H. (1972). Subacute/chronic laminitis and sole ulceration in a dairy herd. Can. Vet. Jo. **13**, 90.

Garner, H.E., Coffman, J.R., and Hahn, A.W. (1975). Equine laminitis of alimentary origin: an experimental model. Am. J. Vet. Res. **36**, 441.

Edwards, G.B. (1982). Acute and subacute laminitis. In: C.W.G. Grunsell and Hill, F.W.G.. (eds.), The Veterinary Annual, 22nd issue. Scientechnica, Bristol. p.99.

Kaufmann, W. (1976). Influence of the composition of the ration and the feeding frequency on pH regulation in the rumen and on feed intake in ruminants. Livest. Prod. Sci. **3**, 103.

Livesey, C.T. Fleming, F.L. (1984). Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. Vet. Rec. **114**, 510.

Logue, D.N., Lawson, A., Roberts, D.J. Hunter, E.A.(1989) The effect of two different protein sources in the diet upon incidence and prevalence of lameness in dairy cattle. Anim. Prod. **48**, 636 (Abstr.)

Manson, F.J. Leaver, J.D. (1988). The influences of concentrate amount on locomotion and clinical lameness in dairy cattle. Anim. Prod. **47**, 185.

Manson, F.J. Leaver, J.D. (1989). The effect of concentrate : silage ratio and of hoof trimming on lameness in dairy cattle. Anim. Prod. **49**, 15.

Nilsson, S.A.(1966.) Recent opinions about cause of ulceration of the hoof in cattle. Nord. Vet.-Med. **18**, 241.

Peterse, D.J. (1979). Een mogelijke invloed van de voeding op het optreden van zoolzweren bij het rund. Tijdschr. Diergeensk. **24**, 966.

Peterse, D.J. van Vuuren, A.M. (1984) The influence of rate of concentrate increase on the incidence of foot lesions in freshly calved heifers. Procs. 35th Ann. Mtg. European Association of Animal Production, The Hague, The Netherlands.

Peterse, D.J. (1985). Laminitis and interdigital dermatitis and heel horn erosion. Veterinary Clinics of North America: Food Animal Practice 1, 83.

Peterse, D.J. (1987). Aetiology of claw disorders in dairy cattle. In: Cattle Housing Systems, Lameness and Behaviour. Wierenga, H. K. and Peterse, D. J. (eds),Martinus Nijhoff Pubs., Dordrecht. p.3.

Russell, A.M., Rowlands, G.J., Shaw, S.R. Weaver, A.D. (1982). Survey of lameness in British dairy cattle. Vet. Rec. **21**, 155.

Trimberger, G.W., Tyrrell, H.F., Morrow, D.A. et al.(1972). Effects of liberal concentrate feeding on health, reproductive efficiency, economy of milk production, and other related responses of the dairy cow. New York Food and Life Sciences Bulletin, No. 8, 1.

Urmas, P. (1968). A new hypothesis about laminitis. Finsk. Vet. Tidskr.,**74**, 11.

Weaver, A.D.,(1979). The prevention of laminitis in dairy cattle. Bovine Practitioner, **15**, 70.

Webster, D. M Leaver, J.D.(1983.) Assessment of lameness in dairy cattle on different systems of concentrate feeding. West of Scotland Agricultural College, Crichton Royal Farm Report, 1983, 27.

Effects of feed changes around calving on cattle lameness

J.D. Leaver

Wye College, University of London, Wye, Ashford, Kent, TN25 5AH.

Introduction

Lameness in dairy cows is a complex problem. It represents the symptom of one or more underlying clinical problems which might have single or multiple predisposing causes. The results of many experiments on hoof lameness in cattle have been equivocal, mainly due to the adherence to experimental protocols which attempt to correlate particular hoof lesions with management variables (e.g. diet). The recorded incidence of a hoof lesion may not correlate well with the management variable studied due to the subjectivity of the lesion description, and to the time lag between the management insult and the appearance of the lesion. Also, a recorded incident of lameness conveys no information on the severity or duration of the causative lesion.

Thus although there is a strong suspicion by veterinary practitioners and animal nutritionists that diet around calving can be a pre-disposing cause of lameness in dairy cattle, there is little confirmatory evidence available.

In this paper, the nutritional state of the cow around calving will be discussed together with the established nutritional causes of lameness. Consideration will also be given to experimental approaches for studying lameness.

Lameness during lactation

The incidence of hoof lesions producing lameness is greater in the winter than in the summer months (Rowlands et al, 1983). This could be due to the influence of housing and/or to the concentrate/forage winter diet compared with summer grazing. A study (Leaver and Webster, (1983) of lameness measured as locomotion score shown in Figure 1 indicates that both influences are probably involved.

In the 12 month study, two groups of September/October calving cows were grazed during the summer, and fed concentrates and grass silage in the winter. The two groups received the same total amount of concentrates over the winter, but one group was fed according to yield (a greater proportion of concentrates were fed in early lactation and to higher yielders), and one group was fed on a flat rate (all cows fed the same daily amount for 26 weeks). Lameness as indicated by locomotion score was significantly higher (more lameness) in the feeding to yield group indicating that feeding a greater proportion of the concentrates in early lactation was detrimental to lameness. An accelerated decline in lameness occurred after turnout to grass in the spring in both groups, and remained low until housing in October.

It is now well established that concentrates are implicated in lameness resulting from hoof lesions (Livesey and Fleming, 1984; Peterse et al, 1984). Studies of diet and locomotion have also shown that lameness is more prevalent with a high proportion of concentrates in the total diet than with a low proportion (Manson and Leaver, 1989). In these studies high protein concentrates also produced more lameness than low protein (Manson and Leaver, 1988b), and high starch more than low starch concentrates (Kelly and Leaver, 1990).

It was not possible from these experiments to identify the direct effects of early lactation feeding as the treatments were applied from weeks 3-26 of lactation. Those diets producing a high incidence of lameness tended to show an increase in locomotion score throughout the experimental period (Figure 2). This suggests there was a continuous effect of diet on lameness. However, it is possible that the increased locomotion score in early lactation was due to tenderness of the feet arising from laminitis, and late problems were due to bruising and solar ulcers as a sequel to the laminitis (in these experiments solar ulcer was a major lesion). This highlights the need to identify the critical periods when diet influences lameness, and the metabolic pathways associated with the problem.

Nutritional influences around calving

The diet offered pre-calving does not appear to be influential on lameness during lactation. Intakes of feed are generally low in comparison with intakes during lactation, and in practice concentrate inputs are low (few farmers now 'steam up' their cows). Thus Livesey and Fleming (1984) found no difference between grazing, grazing plus concentrates or complete diet feeding offered prior to calving on subsequent lameness.

In the first two or three weeks following parturition the cow goes through a number of changes in its physiological and metabolic state. Involution of the uterus takes place and reproductive cycles are initiated, whilst at the same time milk yield increases at a more rapid pace than voluntary intake resulting in a negative energy balance. Infections may also be present in the reproductive tract and mastitis infections are common. It is not surprising therefore that this is a critical period for the general metabolism of the cow when problems such as lameness might be triggered.

The energy deficit in early lactation is met by mobilisation of adipose tissue reserves. It has been estimated that high yielding cows mobilise about 1kg/day of adipose tissue (Vernon and Flint, 1984). There is also a deficit of protein and 150-300 g/day of tissue protein can be mobilised at that time (Bauman and Elliot, 1983). It is not clear whether these metabolic changes have an influence on lameness.

The cow appears to be more prone to acidosis in the first weeks of lactation. At that time intakes of forage are low, and high concentrate levels may result in a high concentrate : forage ratio. Also, the substitution rate of concentrates for silage is highest in early lactation (Table 1). The combination of low forage intake and high substitution rate particularly in the first two weeks post calving could therefore be an important trigger to acidosis.

Rumen acidosis has been implicated in laminitis and solar problems (Weaver 1979; Andersson and Bergman 1980). High starch concentrates which predispose to

acidosis also lead to lameness (Kelly and Leaver 1990). This may be due to the production of bacterial endotoxins (Dougherty et al 1975). These are released from rumen bacteria when exposed to a low rumen pH (Dawson and Allison 1988). It is believed that endotoxins cause an allergic reaction in the capillary cells of the corium, the result being laminitis and the production of structurally incompetent horn (Andersson and Bergman 1980). The latter effect on lameness may only become apparent some weeks or months later when the incompetent horn reaches the solar region and cows become lame because of solar ulcers. The horn grows at about 0.5 cm/month (Manson and Leaver 1989).

Feeding management after calving.

The susceptibility of cows to acidosis and laminitis post calving suggests that the maintenance of a modest concentrate : forage ratio, and the avoidance of large concentrate feeds with high starch levels, are necessary to avoid lameness problems. These could be caused by laminitis in early lactation and at a later stage by solar ulcers.

There is little experimental evidence however to support these recommendations at present. Peterse and Van Vuuren (1984) for example found no difference in lameness between two groups of cattle with a slow and a fast rate of concentrate increase after calving. Nevertheless, knowledge of dairy cow nutrition indicates that the dairy cow is more susceptible to rumen acidosis and laminitis after calving and this should be further investigated. Forage quality, forage intake, concentrate quality, concentrate intake and frequency of feeding are interacting variables which must be taken into account in the experimental design.

Experimental approaches

There is a need to examine in detail, variations in dietary inputs, in particular of acidosis inducing components in the first few weeks of lactation. This could involve a sequential period of experimental treatments e.g. feeding particular diets for 2, 4, 6 or 8 weeks post calving, in order to identify the most susceptible time. The diets could involve different concentrate forage ratios with different starch components in the concentrate or forage. Following the experimental treatment cows would be put on to a common diet. The influence of level and type of protein in the diet should also be studied.

The problem of finding statistically significant effects of treatments on lameness can be overcome by using the locomotion scoring technique (Manson and Leaver 1988a). This allows a regular (e.g. weekly) quantification of the lameness status of each cow. The data generated allows the prevalence, severity and duration of lameness to be analysed statistically. A consequence is that experiments can be carried out on relatively small groups of cows (e.g. 12 cows/group). In contrast most lameness studies have attempted to relate diet to recorded lameness incidents. A frequency analysis such as the X^2 test has to be used in such studies and very large groups of cattle are needed to obtain statistical significance.

To a large extent the significant relationships which have been established between diet and lameness are not well explained in terms of how or why they occur.

There is a need for research to establish the intermediary metabolism in these relationships. Also, effects of diet should not be studied in isolation, as interactions with other management variables can occur. For example, trimming hooves to Dutch Standards (in particular increasing the angle of the toe) can overcome many of the detrimental effects of diet on lameness (Manson and Leaver 1988b; 1989).

Conclusions

Nutrition has been clearly implicated as one of the predisposing causes of lameness in dairy cattle. Rumen acidosis appears to be linked with lameness and cows in early lactation appear to be more prone to this problem. Low forage intakes and high substitution rates of concentrates for forage at that time are likely reasons for the higher incidence.

Avoidance of high concentrate, and in particular high starch intakes in early lactation would appear to be important. So far however, there is little supportive experimental evidence, and there is a need to further examine the relationship between diet in early lactation and its influence on lameness. The locomotion scoring technique is likely to be helpful in this research.

Nutrition pre-calving does not appear to be an influence on lameness post-calving.

References

Andersson A. and Bergman A. (1980). Pathology of bovine laminitis especially as regards vascular lesions. **Acta Veterinaria Scandinavica 21**, 559-556.

Bauman D.E. and Elliot J.M. (1983). Control of nutrient partitioning in lactating ruminants. **In Biochemistry of Lactation** ed. T.B. Mepham, pp. 437-468. Elsevier, Amsterdam.

Dawson K.A. and Allison M.J. (1988). Digestive disorders and nutritional toxicity. In **The Rumen Microbial Ecosystem,** ed. P.N. Hobson, Elsevier, Amsterdam.

Dougherty R.W., Coburn K.S., Cook H.M. and Allisson M.J. (1975). Preliminary study of appearance of endotoxins in circulatory system of sheep and cattle after induced grain engorgement. **American Journal of Veterinary Research 36**, 831-832.

Kelly E.F. and Leaver J.D. (1990). Lameness in dairy cattle and the type of concentrate fed. **Animal Production** (in press).

Leaver J.D. and Webster D.M. (1983). Assessment of lameness in dairy cattle on different systems of concentrate feeding. **Crichton Royal Farm Report 1983**, pp 27-29, The West of Scotland Agricultural College.

Livesey C.T. and Fleming F.L. (1984). Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. **Veterinary Record 114**, 510-512.

Manson F.J. and Leaver J.D (1988a). The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. **Animal Production 47**, 185-190.

Manson F.J. and Leaver J.D. (1988b). The influence of dietary protein inta'e and of

hoof trimming on lameness in dairy cattle. **Animal Production 47,** 191-199.

Manson F.J. and Leaver J.D. (1989). The effect of concentrate : forage ratio and of hoof trimming on lameness in dairy cattle. **Animal Production 49,** 15-22.

Peterse D.J., Korver S., Oldenbrook J.K. and Talmon F.P. (1984). Relationship between levels of concentrate feeding and incidence of sole ulcers in dairy cattle. **Veterinary Record 115,** 629-630.

Peterse D.J. and Van Vuuren A.M. (1984). The influence of rate of concentrate increase on the incidence of foot lesions in freshly calved heifers. **Proc. 35th Annual Meeting of the EAAP,** p MC 5.5, The Hague.

Rowlands G.J., Russell A.M and Williams L.A. (1983). Effects of season, herd size, management system and veterinary practice on the lameness incidence in dairy cattle. **Veterinary Record 113,** 441-445.

Vernon R.G. and Flint D.J. (1984). Adipose tissue : metabolic adaptations during lactation. **Symposium of the Zoological Society of London 51,** 119-145.

Weaver A.D. (1979). The prevention of bovine laminitis. **Bovine Practitioner 15,** 70-72.

Table 1. <u>Effect of stage of lactation on substitution rate of concentrates for silage.</u>

Weeks of lactation	Substitution rate (kg silage DM/kg concentrate DM)	Milk response (kg milk/kg concentrate DM)
3 - 7	-0.66	+0.35
8 - 12	-0.41	+1.16
13 - 17	-0.39	+1.29
18 - 22	-0.29	+1.06
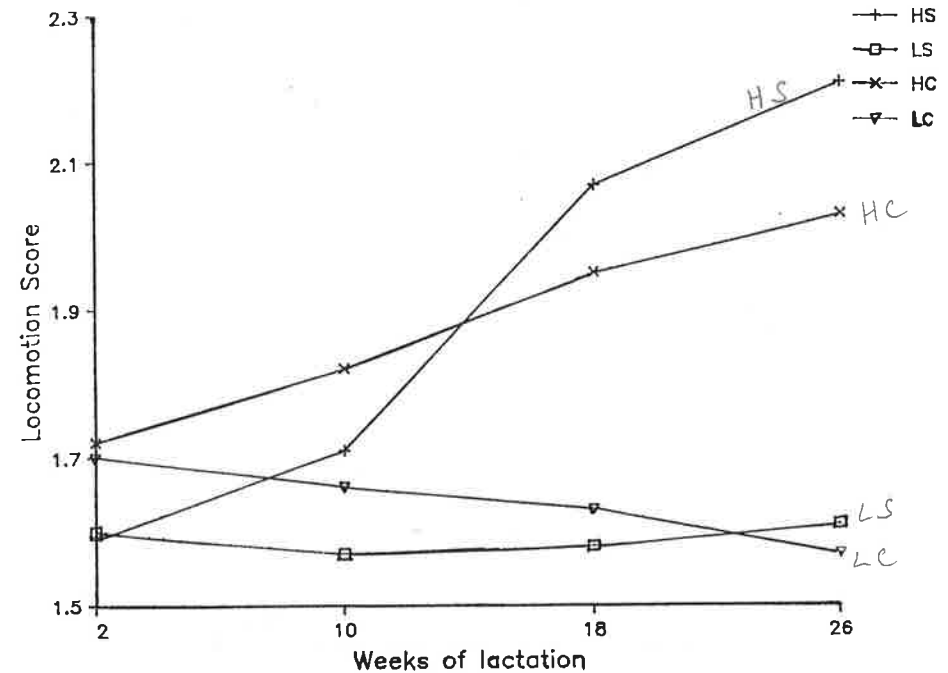
Figure 2. Effect of high starch (HS) vs low starch (LS) concentrate (Kelly and Leaver, 1990) and high concentrate (HC) vs low concentrate (LC) silage ratio (Manson and Leaver, 1989) on locomotion score.
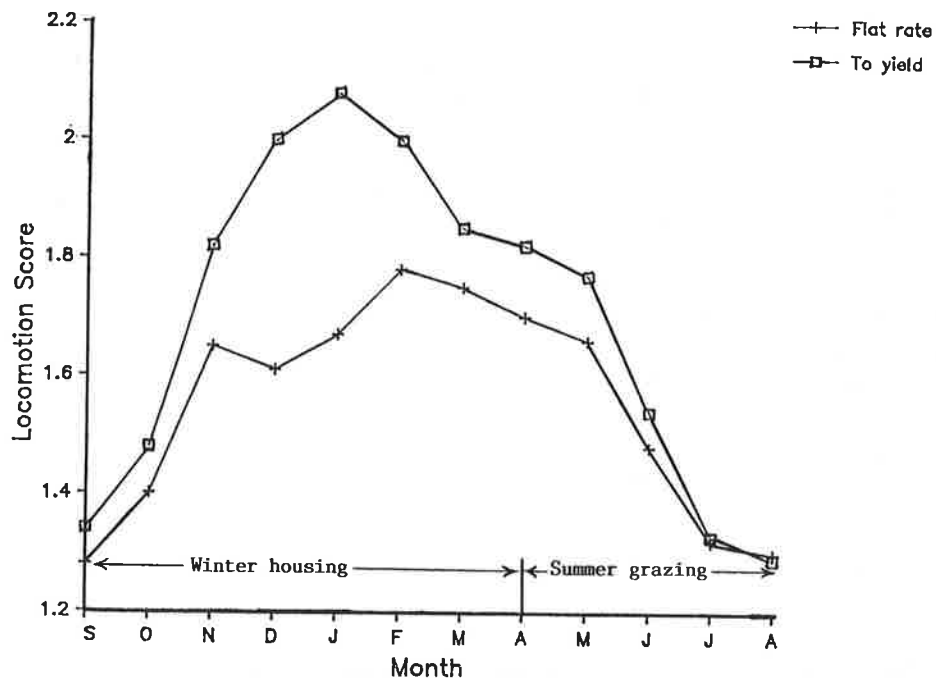
Figure 1. Effect of month and system of concentrate feeding in winter on locomotion score (Leaver and Webster, 1983)

Practical Footcare

D. W. Collick

Delaware House, Castle Cary, Somerset.

My approach to cattle feet has developed more by experience than formal education. Effort was directed at location, paring and treatment of claw lesions followed by the application of bandaging and systemic antibiotics. Little or no effort was given to claw conformation. Animals with gross claw abnormalities were pared by removing excess sole and 'tidying up' the side walls with hoof shears. Using this method the quick was often damaged.

'Glue on' blocks increased my armaments but the additional time factor (30 minutes) in practice meant that they were reserved for the more serious case. Adventuring into 'nail on' blocks proved a revelation, for with courage and practice this type of block can be applied in less than 5 minutes. For me, blocks replaced bandages allowing pain relief with rapid healing of the diseased claw.

Seven years ago I attended an Agricultural Training Board course (1 day) to learn the Dutch technique. I was impressed and subsequently adapted my technique. Further appreciation of the system was gained attending a week's course at the Practical Training Centre for Dairy Cattle and Grassland Management, Oenkerk, Holland in 1989.

Foot Trimming

The Dutch method is well documented (Toussaint Raven, 1985) and will not be described in detail here. In my opinion the method is a safe, step by step, teachable approach to restore good claw function. The following points need careful understanding.

1) Appreciation of claw balance with equal weight sharing.
2) Bearing surface of claw should be flat maximising ground contact area.
3) Measurement of claw length and matching length to second claw.
4) Medial claw is more 'normal' claw requiring preservation particularly in heel region.
5) Outer claw trimmed to match medial claw.

The key word is balance, the most common disparity observed being heel height of the lateral claw (figure 1).

Figure 1
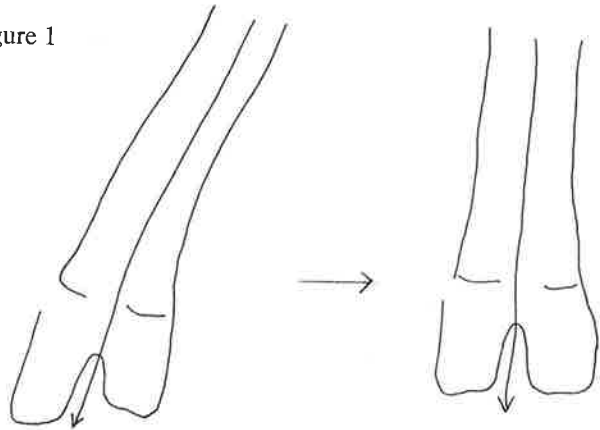
Balance restoration can be appreciated after trimming transforming a 'stickle' to a straight leg stance instantly.

The Lame Cow
I have developed a standard approach to the lame cow.
1) Pare claws to balance using the Dutch method.
2) Locate and pare lesion or lesions.
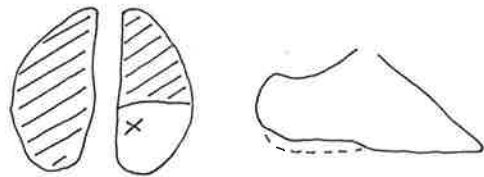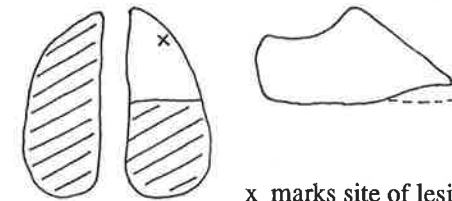3) Thin sole and side wall around the lesion (figure 2).

Figure 2

The majority of claw lesions occur in the posterior segment of the outer claw but for lesions nearer the toe, thin sole and reduce side wall towards the toe. (figure 3).

Figure 3

x marks site of lesion
shaded area represents weight-bearing area

4) Finally and importantly examine and pare the non-lame leg. Often imbalance and lesions are present here.

By this approach the inner claw is used as a functional block. Currently I use fewer blocks but find I always apply blocks where a white line abscess is located in the medial part of the claw, the as the resultant granuloma from claw instability is very painful. I rarely bandage disease claws, considering that drainage will be blocked and secondary anaerobic infections encouraged. I rarely use systemic antibiotic. For the more painful lesion I resort to local anaesthesia to allow better paring. Good anaesthesia can be achieved in about 5 minutes by nerve blocking sites (figure 4) distal to the fetlock joint with 5 to 7cc of 2% lignocaine.

Figure 4   x marks site of lesion.
            shaded area represents weight-bearing area
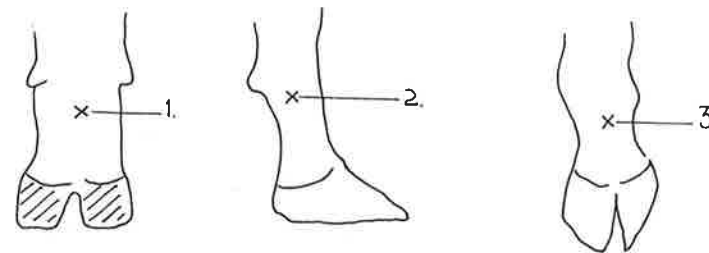
Restraint
I have experience of many different forms of restraint including specialist crushes, ordinary crushes, modified Artificial Insemination stalls and ropes over beams.

To achieve good quality foot work a good facility for handling the lame cow is essential.

Crushes can be divided into three categories.

1) The specialist or "Open ended" foot crush (WOPA, Willmott types).
2) The general purpose crush with facilities for "open end" arrangement (Morris and Mole Valley types).
3) The general purpose crush with "closed end". Many versions are available but all require the raised leg to be tied to rear upright.

In my opinion the 'open end' type crush is safe and comfortable for both cow and operator and importantly allows siting of the foot in the line of weight transfer (figure 5). This is essential to determine the correct claw balance.
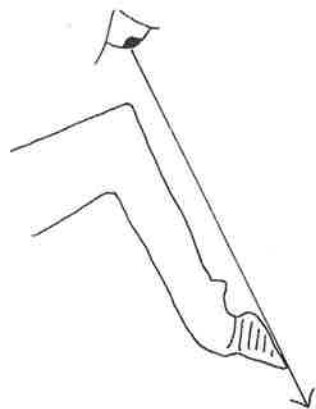
Figure 5.

Equipment

My foot paring equipment is of good quality and simple, consisting of paring knives and a single action hoof parer. The best knives available are hard stainless steel type (Inox, Aesculap or Buffalo) with relatively narrow blades for instep modelling. Sharpness is critical and this type of knife will retain a sharp working edge without rusting for long periods in continuous use. I use an adapted (reverse motor) bench grinder with a rubberised sanding wheel (120 grain) and a buffing cloth. The single action hoof parer (Diamond Farrier Shears) permit easy, safe and accurate cutting with the foot resting on the operator's knees.

Footbaths

These are often installed for the wrong reasons - the belief that it is the easiest answer to a lameness problem. These is no substitute for hoof paring, and foot-bathing

only makes the task more onerous. However where lesions of the skin around the claws is a problem, or after serious herd hoof trimming, footbathing will help maintain healthy claws. Digital dermatitis has recently been recognised in a few herds as a problem. I have achieved acceptable results with formalin where footbath medication was not previously practised. Where the condition has been recognised and increased bathing frequency with formalin has not reduced the condition I have used tetracycline medication. I consider the most effective treatment to be individual cleaning, drying of lesion followed by application of gentian violet/tetracycline spray.

Trimming Frequency

Once or twice a year or   At 'drying off'

This isa difficult question to answer. The safest time to pare is at drying off, the dry period giving time for resolution of any lesions found and recovery from over-enthusiastic paring. This I consider to be a herdman's task and if performed correctly restoration of claw balance should be sustained well into the next lactation.

Deleterious effects of claw overburdening are most manifest in early lactation especially if this coincides with winter confinement on unyielding surfaces. Therefore it is important that claw balance is assessed and rectified at this period of time. I do not recommend herd claw trimming at turnout in the spring.

A small study illustrate the effectiveness of this approach in treating and controlling lameness in dairy practice. The study was made on Somerset dairy farms which were visited regularly each week or fortnight as part of health and fertility control schemes. Foot lameness cases were recorded in 11 dairy herds from October 1988 forming part of a wider lameness project (An Epidemiological Study to Determine the Risk Factors of Lameness in Dairy Cows - Liverpool University). All animals were Friesian or Friesian/Holstein, housed in cubicle systems during the winter, fed silage.

All animals required lameness treatment or foot paring by either veterinarian or herdsman; records noting the leg, hoof shape, the lesion, extent of paring and treatment were made. At intervals throughout the winter all cows were gait scored using a locomotion scoring system (appendix 1, Manson and Leaver).

Results

Sole ulceration (table 1) accounted for a third of all lesions, and white line disease a quarter. Loor/Foul and digital dermatitis accounted for less than 5% of all lesions diagnosed. Estimations of foot conformation (table 2) shows that of 1,968 feet examined, only a quarter were considered normal, the rest were classified as abnormal. the majority of these became of unequal claw size.

Table 1. Major foot lesions observed on 10 Somerset farms Oct. 88 - Nov 89
Population approximately 1600 cows.

Case No./Cows	77
Sole ulcerations	34%
White line lesions	25%
Loor/Foul	3%
Digital dermatitis	1.5%
Miscellaneous lesions	36.5%

Table 2 . Hoof shape classification based on 1,968 observations

	Number of feet examined (%)
'Normal'	492 (25%)
Both claws overgrown	255(13%)
Unequal claw size	1023 (52%)
Corkscrew'	197 (10%)

Table 3   shows the classification of hoof shape relative to the two most important foot lesions diagnosed. Over 80% of feet with sole ulceration and over 50% of feet with white line lesions were classified as abnormal.

Table 3. Classification of hoof shape and observed lesion.

% of Hoof Shape Type

Lesion	Normal	Both overgrown	Unequal	Corkscrew
Sole ulcer	17	7	65	11
Solar bruising	27	7	60	6
Laminitis	14	23	53	10
White line lesion	41	11	41	7

Mean locomotion score for all cows was 2.22 (table 4) with an individual farm range of 1.95 to 2.72. After treatment, gait score had reduced to 1.87 (range 1.60 -

2.13). Those assessed initially as being lame (gait score 3 or more) were 24% of all cows (individual farm range 14% - 43%) reducing to 6% (individual farm range 1% - 17%) of all cows after treatment.

Locomotion scoring as an assessment of lameness has advantages similar to condition scoring. As a numerical assessment of lameness in a herd, one herd can be compared with another, creating a greater awareness of a lameness problem in both. The improvements noted in locomotion score following treatment in this study in part relate to an increased awareness and eagerness to improve the locomotion score, and in part to a long, dry, grazing season.

The evidence presented supports the Dutch ideal that overburdened claws are more prone to disease. Restoration of foot balance by regular trimming is likely to produce major benefits in the reduction of claw disease.

Conclusion

I consider there is immense potential for high quality foot trimming in dairy cows. My own experience strongly suggests it is a teachable art that requires much experience to appreciate the fullest implications. I consider there is an urgent need to develop a school of bovine chiropody on similar lines to the Dutch to fulfil this requirement. In addition dairy farmers should be urged to examine and improve management systems putting the cow's requirements first.

References
Baggott, DG, & Russell, AM (1981) British Veterinary Journal **135**, 113.
Cermack, J. (1987) Proceedings for 1986-87 British Cattle Veterinary Association p 173.
Colam-Aimsworth, P, Lunn GA, Thomas, RC & Eddy RG, (1989) Veterinary Record **125**, 573.
Collick DW, Ward WR, Dobson, H. (1989) Veterinary Record **125**, 103.
David GP,. (1986) 5th International Symposium on Disorders of Ruminant Digit. Dublin 1986 p68.
Eddy RG & Scott CO (1980). Veterinary Record **106**, 140.
Pinsent PJN, (1981) Bovine Practitioner **16**, 61.
Russell AM, Rowlands GP, Shaw SR, & Weaver AD (1982) Veterinary Record **111**, 155.
Toussaint Raven E. (1985) Cattle Footcare and Claw Trimming. Farming Press Ltd, Ipswich, Suffolk.
Whitaker DA, Kelly JM & Smith EJ (1983) Veterinary Record **113**, 60.

Functional Anatomy in Digital Joints of Cattle

H. Heyden, O. Dietz and L.F. Litzke

*University Clinic of Animal Surgery Berlin (Head: Prof. O. Dietz), Veterinary Practice
Barth (Head: Dr. H. Heyden)*

In recent times, loose housing has made new demands on veterinary medicine. Keeping in stalls has nearly always been a synonym for tied stalls. Burdening due to standing position combined with muddy conditions, accumulation of pathogens as well as neglected footcare therefore often results in lameness. In modern arrangements, however, cattle are forced to move on industrially produced floor, the hardness of which makes it comparable to high mountain biotopes, but the faecal layer and humidity produce a hardness of claw horn which is actually appropriate for marshy grounds. This expects too much of adaptation from cattle, and man is thus required to help.

The following experiments describe how the numerous recesses of the articular capsule of the digital joints behave with regard to synovia in the unburdened and the burdened state.

Materials and Methods

Twenty-seven distal limbs of cows (4-8 years old) were used for anatomical preparation. They had been separated at the tarsal or carpal joints. Another 21 digits (fresh from slaughter-house) of fully-grown cows were used for burdening experiments in the Moser lever (Moser 1908) modified by Knezevic (1962) (Fig. 1).

There was no discrimination between hind and front digits. After the fetlock joint had been clamped into the Moser lever, it had to be punctured. For this special purpose, a 2.5 mm needle was tied up airtight by a synthetic tube with scaled glass tube. The puncture was applied to the palmar/plantar recess of the fetlock joint capsule, which, according to Berg (1982), reaches some 7 cm proximally. All digit joints where the synovia did not go up in the glass tube in the first puncture could not be considered.

The immediate finding of the articular capsule at the burdened digits was easier because the recesses were sometimes bulging and well palpable. To get access to the site of the puncture, a 3-5 cm cut had to be made in the skin. The palmar/plantar articular capsule recess had to be prepared accordingly.

The glass tube, which was open on the top, could take up 1 ml of synovia, the respective fluid level being measurable at the scale of the tube. Six joints had so much synovia in the capsule that it overflowed out of the opening of the glass tube after the puncture, even before the claws could be deburdened.

In the cases where only little synovia or no synovia rose into the tube, the flat hand was laid on the burdened digit onto the dorsal recess of the articular capsule of the

fetlock joint to exert a moderate pressure. The synovia level generally went up in the tube. Sometimes the needle had to be turned a bit because in these cases it seemed that the tip of the needle was close to the articular capsule so that the sloping opening of the needle was blocked up.

After the digits had been burdened and deburdened four times, the respective synovia level was measured for each claw in both the burdened and deburdened states. The respective values could be recorded for 11 claws.
Fig. 2 shows a burdened digit where the palmar recesses of the joint had been punctured by a needle-tube combination. The ligament systems of the digit have been studied in the preparation for their biodynamic effect on the recesses of the articular capsule.

Results

Table 1 shows the increases in synovia levels recorded in the bovine digit under a burdening pressure of 230 pa.
Table 1 Increases in synovia levels in burdened digits.

Serial number	Synovia level (1/100 ml) deburdened	Synovia level (1/100 ml) burdened	Synovia increase ml
1	18	80	0.62
2	20	95	0.75
3	19	100	0.81
4	25	94	0.69
5	17	88	0.71
6	21	64	0.43
7	10	98	0.88
8	19	87	0.68
9	22	94	0.72
10	11	90	0.79
11	15	70	0.55
x̄	18	87.27	0.69

Unlike traditional opinion, the M. interosseus medius in cattle does not only pass through below the tendon of the M. extensor digitorum communis but does also largely join with its sheath, and thus becomes a backward fixing strap of the extensor tendon, the tension of which increases with dropping pastern (Fig. 3).

The lateral flat legs of the M. interosseus medius communicate, from the abaxial side, with the tendon sheaths and produce an effect which is quite similar to the backward fixation. If the interosseus muscle is prepared and if the digit is burdened in the lever, the crossed sesamoid bones pull themselves into the fetlock bone groove and straighten up from their sloping position. At the same time the large capsule of the fetlock joints emerge as a cherry-like bladder filled with synovia through a cleft between the crossed sesamoid bone ligaments.

The prepared palmar/plantar ligaments of the second digital joint clearly proved their self-carrying function in the burdened state in the Moser lever. A cleft between the second digital joint and the palmer/plantar ligaments enlarged, but did only exist in the axial region. In addition there was a connection between the interdigital ligaments and the outer chondroid part of the dorsal capsular recess of the fetlock joint (Fig. 4)

Discussion

The anatomic studies of burdened digits have shown that in the palmar/plantar region of the three digital joints a pressure is exerted, due to their self-carrying function, on the recesses of the articular capsule of these joints, i.e.
-through the interosseus muscle at the fetlock joints
-palmar/plantar ligaments at the second digital joints, and
-the ligament system of the distal sesamoid bones at the pedal joints.

The ligament systems are supported by the tension of the flexor tendons. In our experiments, however, this tension was lacking. We measured an average synovial fluctuation of 0.69 ml in the palmar/plantar recesses of the articular capsule of the fetlock joint. In Vitro these values are supposed to be higher. For the even pressing of the recesses of the capsular joints all the "cavities" between tendons, ligaments and bones have to be filled with fat. It has to be pointed out that this is not solid beef dripping but yellow and very soft fat which is infiltrated and coated with many elastic elements. In addition we also detected accumulations of fatty tissue in the recesses of the articular capsule of the digital joints as well. Sometimes they were linked with the capsule. It seems that these accumulations of fatty tissue reduce the necessary synovia to a minimum.

A similar interpretation should probably be given of the fatty tissue complexes described by Walla (1983), which develop with age at the bottom of the synovial groove. The synovia pressed out of the recesses of the articular capsule collects in the region of the force-weight plumbline of the corresponding digital joints.
These relations are shown in Fig. 4. The following conclusions can be drawn from Fig. 4 with regard to synovia distribution:
A Synovia collects at the fetlock joints:
1. as early as in the phase of flexion and forward movement of the leg in the region of the slightly widening articular cavity between the proximal sesamoid bones and the fetlock joint (1),
2. in the articular cavity, which is widening under burdening, between the proximal sesamoid bones and the pastern bones (2),
3. in the palmar/plantar recesses of the articular capsule which may pass as a cherry-like bladder through a cleft of the crossed sesamoid ligaments (3).

B Synovia collects at the second digital joints:
   In the region of the load plumbline where the large recess of the articular capsule passes through a cleft into the axial region of the palmar/plantar ligaments (4).
C Synovia collects at the pedal joints:
   1. During the phase of flexion and forward movement of the leg in the slightly widening articular cavity between the distal sesamoid bone and the second digital bone (5).
   2. in the small articular cavity between the distal sesamoid bone and the pedal bone (6), which is widening as soon as the digits are burdened and the ligaments of the sesamoid bones are stretched. The recesses of the articular capsule surrounding the distal sesamoid bones may thus increasingly absorb synovia.
   The collections of synovia in the region of the load plumbline of the digital joints generally takes place as early as in the phase of flexion and forward movement of the leg. Though after that, i.e. during burdening, the collecting recesses for synovia widen as a result of the described enlargement of the small articular cavities between the sesamoid bones and the pastern and the pedal bones or as recesses of the joint capsule filled with synovia, passing through the ligament clefts. But at the same time these accumulations of synovia are increasingly put under pressure
   -by pulling the sesamoid bones tight to the joints,
   -by pulling the crossed sesamoid bone ligaments of the sesamoid bones into the fetlock bone groove,
   -by pushing the synovia recess of the articular capsule at the second digital joint back into the initial position through the palmar/plantar ligament clefts as a result of the tension of the flexor tendons,
   -by pressing the distal sesamoid bones at the joints together with the surrounding recesses of the articular capsule through the tension of the deep flexor tendon.
   Altogether the sum of the pressures exerted must be higher than the spatial expansion, otherwise there would have been no increase in the synovial level during burdening of the digits in our experiments. Thus the lubrication under pressure of the digital joints is possible depending on the intensity of burdening. The dorsal recesses of the articular capsules have not been considered in our previous discussion. They do have nevertheless some biochemical peculiarities which have not yet been mentioned in literature. The sloping position of the digit and the effect of the extensor tendon produce a tension which make the tendon lift from the bone (Fig. 3). The synovia would be theoretically sucked into these recesses of the articular capsule. Such a behaviour would be biomechanically useless. Therefore the outer part of the dorsal recess of the articular capsule of the fetlock joint is purely chondroid (Fig. 5). Our anatomical preparations have shown that it is linked with parts of the interdigital ligaments in a way that is pulled tight to the joint if the digit is burdened (Fig. 5).
   From the abaxial side, this happening is supported by the lateral large legs of the interosseus muscle. In addition, the fibres of the interosseus muscle passing through the space between the articular areas produce an effect on the joint capsule of the second digital and the pedal joints (Fig. 3) The fibres connect with the dorsal recesses of the articular capsules in order to pull them tight to the respective joints under tension of the interosseus muscle. This is how, during burdening, synovia can be pressed out of the dorsal recesses of the digital joints.

The biomechanical significance of these processes cannot be deduced from statistics alone. Only dynamics, i.e. the whole course of the locomotion, permit to detect some correlations. Absorption of shock and lubrication under pressure depending on the burdening should be of prior interest here.

This principle of action may also be applied to the biomechanics of the synovial grooves. Walla (1983) found such formation in cattle digits only in the axial region of the pedal joints. After our investigations on the increase in synovial pressure in the palmar/plantar recess of the articular capsule of the fetlock joint during digital burdening we assume that the synovial grooves, which are developing with growing age and bodyweight of the animals, lead to an improved function and are necessary, in contrast to what has been supposed until now, for more than just lubrication of joints and nutrition of cartilage (Burki 1905; Palmgreen 1928; Bauer et al.,1930; Luy 1935; Loeffler 1975; Walla 1983).

Cohrs and Messow (1969) and also Walla (1983) consider the synovial grooves as physiological formations, whereas Schmidt and Andreae (1974) believe that their formation is caused by environmental factors.

A real improvement of the biomechanical function by synovial grooves may become necessary with increasing bodyweight. It can only be understood if the starting point is again an excessive filling during the forward movement of the unburdened leg. The influx of synovia originates from the pressurised recesses of the articular capsule. The unburdened leg produces a hydraulic effect in the excessively filled synovial groove. At the beginning of the burdening of the digit, a high synovial viscosity prevents a rapid reflux from the cleft between the two neighbouring bones of the articular cavity. Under increasing burdening the cartilaginous parts, however, touch each other and the synovia is locked up in the synovial grooves. Provided that the mutual elastic flattening of the articular cartilage layers is higher than the compressibility of the included synovia, there is a second hydraulic effect in which the osseous bottom of the synovial groove exerts pressure on the synovia. If the intrasynovial pressure exceeds the elasticity of the surrounding cartilaginous layers, synovia can also be pressed out of the synovial groove between the articular cartilage areas.

This biomechanical principle protects, on the one hand, the sensitive cartilage because the lower compressibility of the synovia prevents a stronger mutual flattening of the articular cartilage layers. It reduces, on the other hand, the friction coefficient in the region of the synovial groove since the articular regions no longer touch each other. This permits a lubrication of the joints in the cartilaginous areas surrounding the synovial groove also under extreme burdening. It is essential in this context that the efficiency of this principle increases with growing demands on performance.

Such an interpretation makes the localisation of the synovial grooves at the sites of extreme burdening (Burki 1905) appear to be logical. Loeffler's (1975) opinion has to be doubted when he said that these regions of articular cartilage where synovia develops are not needed for support and gliding. It has to be rather assumed that the joints with synovial grooves functionally meet the demands much better.

A sign of high biomechanical burdening can be that Walla (1983) detected synovial grooves in cattle covering some 40 to 50% of the total original area of the joint, which were 2 to 4cm deep. The pumping and distributing effect described provided also new functional and anatomical confirmation of the nutritional and greasing function of synovia (Burki 1905; Palmgreen 1928; Bauer et al., 1930; Luy 1935; Loeffler 1975; Walla 1983).

Summary

The experiments described in the present study investigated the distribution of synovia in the articular capsules in burdened distal parts of cattle. The morphology of the biomechanical devices needed has been described.

Finally there is an attempt to interpret the biomechanical function of the synovial groove anew.

Literature

The literature quoted can be provided by the authors.
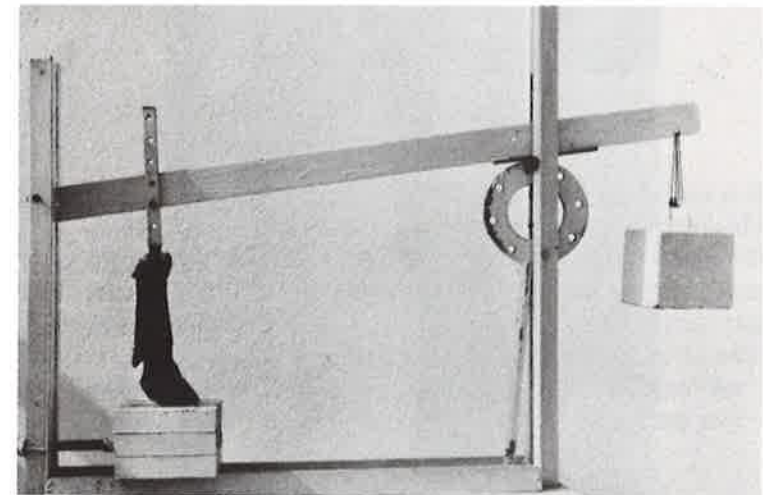
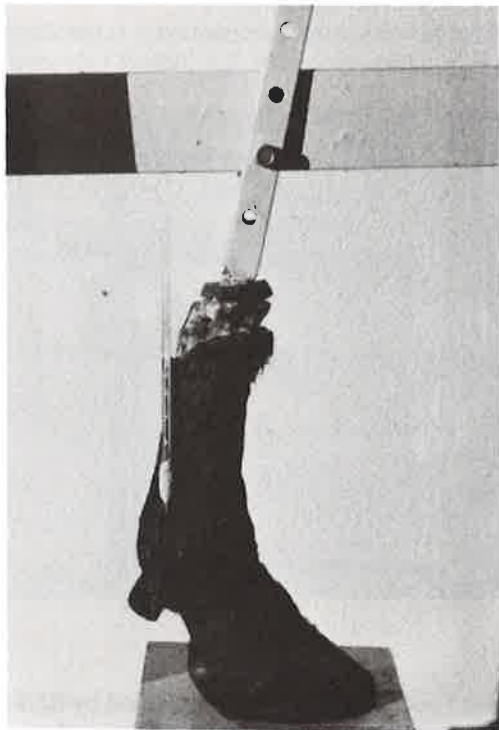Figure 1. The Moser Lever (MOSER, 1908) modified by KNEZEVIC (1962)

Figure 2. The scalded glass tube for synovia was punctured into the palmar recess of the pastern joint.

Figure 3. Scheme of insertion of the interosseus muscle at the tendon sheath of the *M. extensor digitorum communis*.
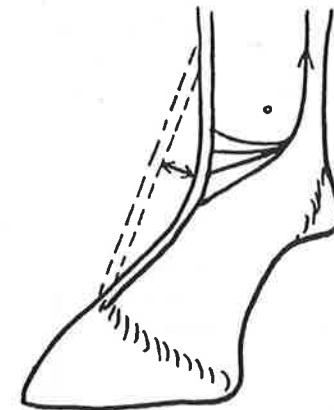

Figure 4   Scheme of the opening angle of the digital joints (broken line) in the case of the dropping fetlock, which leads to an enlargement of the small articular cavities between the pastern bone and the proximal sesamoid bone (2), the pedal bone and the distal sesamoid bone (6) as well as to an enlargement of the palmar/plantar ligament cleft (4). (1) and (5) are the articular cavities of the sesamoid bones which slightly widen in case of digital flexion. (3) is the cherry-like synovia-filled bladder of the articular joint at the pastern joint.
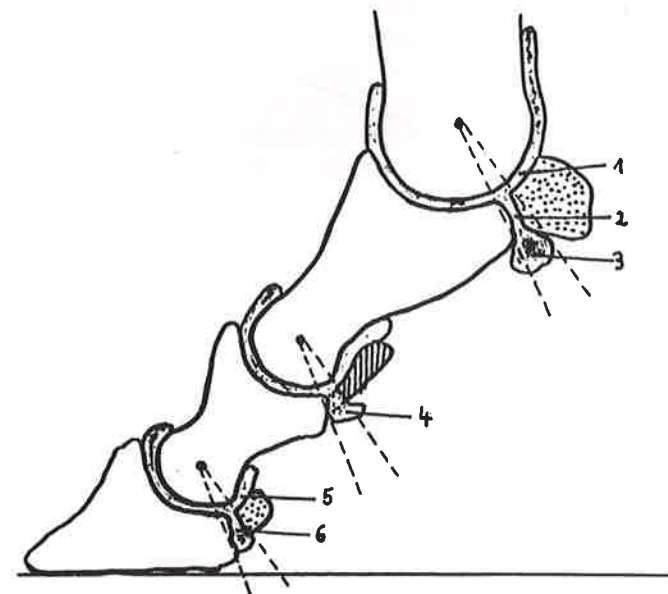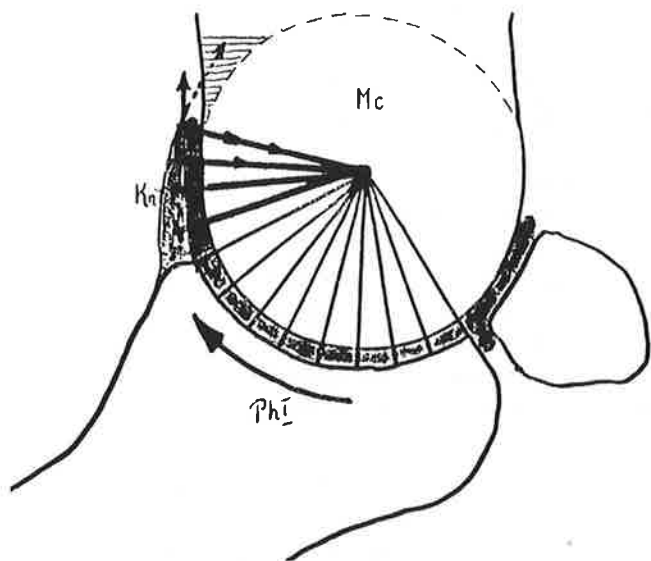
Figure 5. Scheme of effects of the dorsal parts of the interdigital ligaments. There is a tension arising from this ligament as soon as the first phalanx (Ph I) drops and the hatched part of the Mc pushes between the chondroid part (Kn) and the ligament links.

Biomechanics of Digital Joints in Cattle

Ch. Stanek and J. Kastner

Clinic of Orthopaedics in Ungulates, University of Veterinary Medicine, Vienna, Austria
(Head;Prof. Dr. Knezevic)

Summary

Biomechanics of the digital joints are of a quite complex nature, especially in ungulates. Forces acting from the body are transmitted to two digits, then forwarded to the ground by two hooves. Each digit is also subjected to forces deriving from the neighbouring digit, since the two digits are connected not only by tendinous structures, but also by the narrow tube of skin that surrounds them. The main direction of movement of all three digital joints is flexion and extension, but rotatory motions and combined motions are of significance as well.

Despite the various types of limb conformation found in sound cattle, such as base-wide position or cow-hocks, a vertical axis of the cannon bone should be accepted as a reference point for the following considerations:

Fetlock joint

The metacarpo- (metatarso-) phalangeal joint holds a special position, in that it is found in a state of dorsiflexion in the standing animal, achieving a state of flexion just prior to the moment when the hooves are lifted off the ground. Rotation and sliding in the joint are prevented by ligamentous structures and by the sagittal ridge at the distal end of the cannon bone which fits into the corresponding groove at PI. This locking mechanism is most effective while the joint is under load. In an extreme flexed position some rotation and abduction are possible. The fact that the axial part of the joint surface is located in a more distal position than the abaxial one also contributes to inhibiting rotatory and abductional movements.

Compared to the horse, the proximal sesamoid bones are in a more upright position. In conjunction with the collateral ligaments and the suspensory apparatus they prevent a dislocation of PI in the anterior direction.

The fetlock joint acts as a gynglimus, it's axis is transverse to the longitudinal axis of the cannon bone.

Proximal interphalangeal joint

The proximal interphalangeal joint (PIP) is described in literature as a saddle joint. This description applies to the form of distal articular surface of P1 only. The corresponding surface of P2 does not have the form of a saddle, but of an ellipsoid or condylar joint. The concavity of the distal joint surface corresponds to the convex proximal surface and a corresponding groove at P1 are evident, though smaller than in

the fetlock joint. This ridge is not exactly in a sagittal plane, but deviates in a dorsoaxial direction. Taking the axial contour of the claw as a reference line, an angle of about 8 degrees is formed.

Insertional points of collateral ligaments of this joint are situated palmar (plantar) to the pivot of the joint, acting as a brake when the joint is hyperextended. The dorsal ligament, acting as a long axial collateral ligament, in contrast inserts dorsal to the pivot, inhibiting the flexion of this joint.

As to joint geometry, the overall elliptical shape of the joint allows forces acting unilaterally, as in abduction or adduction, to be neutralised first in a gliding motion and then converted into a rotatory movement.

When the digit is unloaded, the PIP-joint forms an angle of about 170 degrees between the long axis of the fetlock bone and the long axis of P2. Loading of 2354 N induced a flexion (!) of the PIP joint to an extent of 8 to 15 degrees. This tendency of flexion under load is restricted by ligamentous structures, like the M. interosseus with it's branches and the superficial flexor tendon inserting at P2. The ligamentous connection > M.interosseus - accessory ligament deriving from the M.interosseus - superficial flexor tendon -(P1), P2 < plays an important role. In the landing phase of the stride, a vertically directed force acts on the cannon bone, inducing a hyperextension of the fetlock joint, but also a slight flexion of the PIP-joint and a DIP-joint. Flexor tendons and the branches of the M. interosseus are put under tension. Together with the anular ligaments they prevent a further flexion of the PIP-joint. They form one of the springlike constructional elements of the digit.

Distal interphalangeal joint

In contrast to the PIP-joint, the range of passive motion of the distal interphalangeal joint is relatively large. Flexion/extension are the main directions of motion. Beside the deep flexor tendon inserting at P3, there is another ligamentous structure which acts as a synchronising element: the elastic ligament connecting the distal end of P2 with the deep flexor tendon near the distal sesamoidal bone.

The axis of the DIP-joint is not exactly horizontal, but declining from abaxial-proximal. The distal articular surface of P2 has an axial and an abaxial portion. The diameter of the abaxial division of the joint surface is about two times the diameter of the axial portion. Overall the joint surface resembles a segment of a truncated cone, with the tip in the axial direction. The ridge on the articular surface of P3 and the corresponding groove at P2 are not parallel to the axial wall of the horny capsule. A direction of palmar-abaxial to dorso-axial is evident. In the plantar parts of this course an angle of 15 degrees to the axial wall exists; approaching the extensor process the angle increases to 30 degrees. This geometrical configuration causes a typical pattern of motion of the DIP joint: if we accept the middle phalanx as a resting system and the distal phalanx as the system in motion, the distal phalanx shows the motion of a nut turning on the thread of a bolt: flexion is combined with dislocation and (due to the increasing angle) also with spreading of the tip of the claws.

When the toe is passively moved through manipulation, flexion is distributed on all three joints, with the main flexion in the fetlock joint. A total flexion of the digit to an angle of 75 degrees is the result of flexion to an angle of 120 degrees in the fetlock joint, 150 degrees in the PIP-joint and 165 degrees in the DIP joint.

Address of the authors:
Doz. Dr. Ch. Stanek, Dr. J. Kastner, Linke Bahng. 11, A 1130 Wien, Austria.

The Effect of Floor Design on Skid Resistance in Dairy Cattle Buildings

John Dumelow and Richard Albutt

Farm Buildings Development Centre, Agricultural Development and Advisory Services, Reading, U. K.

Summary

The problem of determining the optimum slip-resistant floor surface for dairy cattle has been tackled using a combination of two methods. The horizontal displacement of a cow's foot while in contact with the floor has been used as an index of slip-resistance. The first method measured horizontal displacement of cows' feet on a walkway. However, the variability of the results required the development of a more controlled technique. Thus a mechanical simulator of cow movement was devised. For optimum slip-resistance, floors for dairy cattle should be finished so that they have parallel grooves 10 mm wide running perpendicular to the main walking direction of the cattle and spaced at 40 mm intervals. Alternatively, where the walking direction of the cattle is difficult to predict a pattern of hexagons with 46 mm sides formed by 10 mm grooves is preferable.

Introduction

This study is concerned with improving the slip-resistance of solid concrete floors for cattle. Slippery floors are the major cause of leg lameness in dairy cows. Leg joints, ligaments and tendons are the main tissues damaged when a cow slips and falls onto or against hard surfaces.

Concrete is the most commonly used floor material used in dairy cattle buildings and various methods for applying textures to this surface have been used. The currently recommended finish for new concrete in yards and passages is tamping. For existing worn concrete in the same area a grooved or scabbled finish is advocated. It is not known which of these surface textures has the greatest slip-resistance.

Walkway Test Rig

The background to this study and the original method used are described by Albutt and Cermak (1985) and Albutt, Dumelow, Cermak and Owen (1990). A floor testing facility was constructed in which the surfaces were tested. A walkway was made up of a number of removable panels which could be filled with a material having a specific surface texture. The cows were walked over the floor surface under test by driving them through a race.

Foot movement and forces were measured with a motion analysis system and force plate respectively, both of which were interfaced to a DEC Micro PDP-11 computer (see fig. 1). The motion analysis system measured the movement of the cows' feet while in contact with the floor, to detect the amount of slip occurring. The force plate measured the horizontal force magnitude and direction which are of use in determining the optimum orientation of ridges or grooves in a surface profile.

Slip was defined as the horizontal displacement of the foot which occurs whilst the foot is in contact with a floor surface. The direction of slip was measured relative to the direction in which the cow was walking; positive angles being anticlockwise.

Six surfaces have been tested on the walkway test rig; one soil surface and five different concrete surface finishes. Since cattle evolved to walk on resilient, yielding surfaces the soil surface was included to act as a reference against which foot movements on hard, unyielding concrete surfaces could be compared.

The concrete surface finishes tested were:-
(1) steel float
(2) laterally tamped
(3) longitudinally tamped
(4) laterally grooved
(5) longitudinally grooved

The lateral and longitudinal directions were taken to be normal to and parallel to the direction the cows were walking respectively. Each of the concrete surfaces was tested both dry and slurry covered. Russell et al. (1982) found that 76% of leg lameness occur in the hind limbs, so results are presented for the back feet only.

The mean slip magnitude for each treatment is shown in fig. 2 with the horizontal bars representing the 95% confidence intervals. An analysis of variance of the slip magnitudes showed that there were significant differences in these eleven treatments. However, the degree of variability in the data meant that it was not possible to establish significant differences between many of the surface treatments, although the results did show significantly less slip on soil than slurry-covered concrete surfaces. Each of the different concrete surface finishes appeared to be more slip-resistant when dry than when slurry-covered. Of the slurry-covered treatments, longitudinally tamped concrete appeared to be the most slip-resistant.

Although there were significant differences between treatments for slip direction, variation in the data again made interpretation of the results difficult. No effect due to texture direction was apparent. The overall mean slip direction for the back feet was +7° i.e. forwards and outwards.

From these results the following conclusions concerning progress towards the objective of designing slip-resistant floor surfaces can be drawn:

* Variability in the slip data meant that the standard error for each treatment was large and consequently it is difficult to investigate the effect of small changes in surface texture (e.g. the effect of changes in groove spacing).

* It is also difficult to investigate the effect of changes in texture because of the considerable time and effort involved in changing over the floor panels under

test. Changes to the orientation of the panels were also not feasible.

* Collecting data from the rig proved to be time consuming and slow which was compounded by frequent breakages.

Analysis of the data on horizontal displacement has indicated that most of the displacement takes place during the first and last 0.1 s of foot contact. Since displacement during the last 0.1 s is unlikely to result in a violent skid, as the cow is taking its weight off that foot at this stage, the first 0.1 s is considered as being of particular importance. Comparison of the results for the first 0.1 s and for the whole footstep has shown that the former can equally well be used as a slip resistance index. Both give similar relative resistance indices for the same floor surfaces (Dumelow and Albutt 1988)

Foot Simulator Rig

It became apparent that the approach described above had a number of weaknesses. In order to progress further in the development of improved floor surfaces it was necessary to have better control over variables such as cow behaviour, hoof geometry and hoof horn properties.

Accordingly, a machine was designed to simulate the behaviour of a real cow's foot in a more repetitive manner (fig. 3). Data for the design of the simulator was obtained from analysis of the force system generated by a cow's footstep, and measurement of the geometry and physical properties of hooves from slaughtered cows (Dumelow and Albutt 1988). As shown in figure 3 the foot simulator consists of a pendulum which, when released, swings down and then compresses a coil spring mounted on a telescopic shaft. A plastic foot, having similar physical and geometric properties to hoof horn, is attached to the lower end of the shaft. After descending, the foot comes into contact with the floor panel underneath and the spring is compressed to apply the force system. As the telescopic shaft is fitted with universal joints at each end, the foot is free to slide on the surface in any direction. Small springs control the movement of the lower universal joint to duplicate the muscles in the cow's foot as shown in fig. 4. The magnitude and direction of slip during the first 0.1 s is measured with two linear displacement transducers as also shown in this figure.

Tests were carried out with a slurry-covered concrete panel having a single groove across it in order to determine the effect of different positions of the groove relative to the foot. Fig. 5 shows the effect of the position on initial contact of a lateral groove on slip magnitude. The shaded areas in the plan represent the main contact areas on the foot. The two troughs in the centre indicate that the slip is minimised if some of these contact areas are over a groove. A similar situation is observed with a longitudinal groove as shown in fig. 6

It is interesting to note that in both fig. 5 and fig. 6 the troughs (and main contact areas) are approximately 60 mm apart. To achieve skid resistance in all situations at least one of the main contact areas should always be over a groove. For parallel

grooves this can be best achieved (without going to unrealistically small groove spacings) by using a groove spacing equal to two thirds of 60 mm i.e. 40 mm. This is illustrated graphically in fig.7. In all four foot positions illustrated there are at least two main contact areas over a groove. With a groove width of 10 mm this gives a void ratio (i.e. the proportion of the total area taken up by grooves) of 25% which is about the maximum acceptable without creating excessive pressure on the cow's feet.

Separate experiments have been conducted using the simulator to determine the effect of groove direction on slip magnitude. These have shown that while parallel grooves help prevent skids in all directions they are most effective when they run perpendicular to the direction of walking.

Clearly, therefore, in order to arrest skids in all directions, parallel grooves will help considerably, but a pattern of 40 mm squares would be better. However, with a groove width of 10 mm this gives a void ratio of 44% which is much too high. A number of alternatives were considered, and eventually a pattern of hexagons having sides equal to $\frac{4}{3\sqrt{3}} \times 60$ mm (i.e. approximately 46 mm) was chosen. fig. 8 shows how this floor virtually always ensures that at least one contact area is over a groove. For 10 mm grooves the void ratio is 23%. Grooves narrower than this are likely to get blocked and prove ineffective in the long run while a greater width will increase the void ratio unacceptably.

Thus the floor surface in dairy cattle buildings should either have parallel 10 mm grooves spaced at 40 mm intervals or, preferably, it should consist of a pattern of hexagons with 46 mm sides formed with 10 mm grooves.

Unfortunately, floors of this type are not easy to manufacture. If the intention is to renovate an existing floor, then the only realistic option is to cut parallel grooves using a flail or diamond grooving machine. It is not possible to produce hexagons. For new floors it is possible to produce either the parallel or hexagon type of grooving in fresh concrete. However, the techniques are not very straightforward and depend very much on the workability and mix design of the concrete. Alternatively, precast slabs or paving bricks could be used. ADAS is at present working on reliable techniques for producing grooves in new concrete.

Conclusions

A skid resistance index of a floor can be determined by measuring the horizontal displacement of a cow's foot (i.e. the slip) while in contact with the floor. Experiments conducted to measure this, both with live cows and a foot simulator, indicate that the ideal skid-resistant floor surface should consist either of parallel 10 mm grooves spaced at 40 mm intervals or, preferably, a pattern of hexagons with 46 mm sides formed by 10 mm grooves.

References

Albutt R.W. and Cermak J.P. (1985) An approach to the development of improved floor surfaces for dairy cows. Farm Buildings and Engineering **2(2)**, 21-23.

Albutt R.W., Dumelow J., Cermak J.P and Owen J.E. (1990). Slip-resistance of solid concrete floors in cattle buildings. Journal of Agricultural Engineering Research, **45(2)** , 137-147.

Dumelow J. and Albutt R.W. (1989). Developing improved designs of skid resistant floors for dairy cattle buildings. Proc. Third International Livestock Symposium, Toronto, Canada, ASAE Publn. **1-88**, 163-170.

Russell A.M., Rowlands G.J., Shaw S.R. and Weaver A.D. (1982). Survey of lameness in British dairy cattle. Vet. Record, **111**, 155-160.

Figure 1. Instumentation for walkway test rig showing force platform and motion analysis system interfaced to computer.
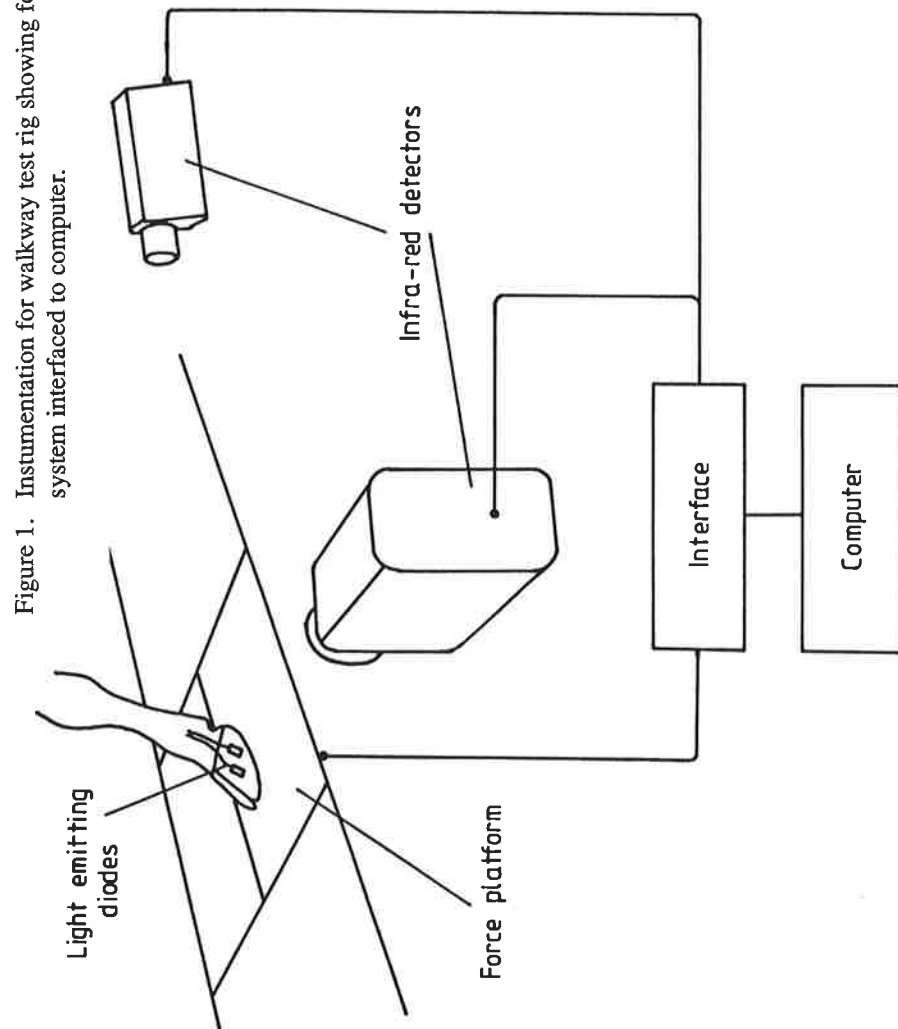
Figure 2.   Mean slip magnitudes and 95% confidence limits for cow's rear feet.
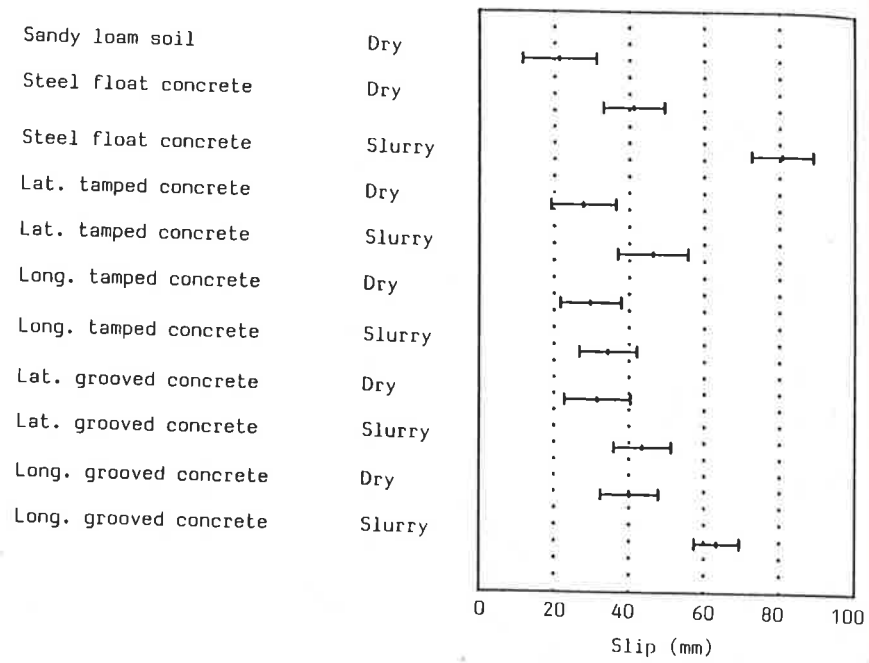
Figure 3.   Foot simulator rig.
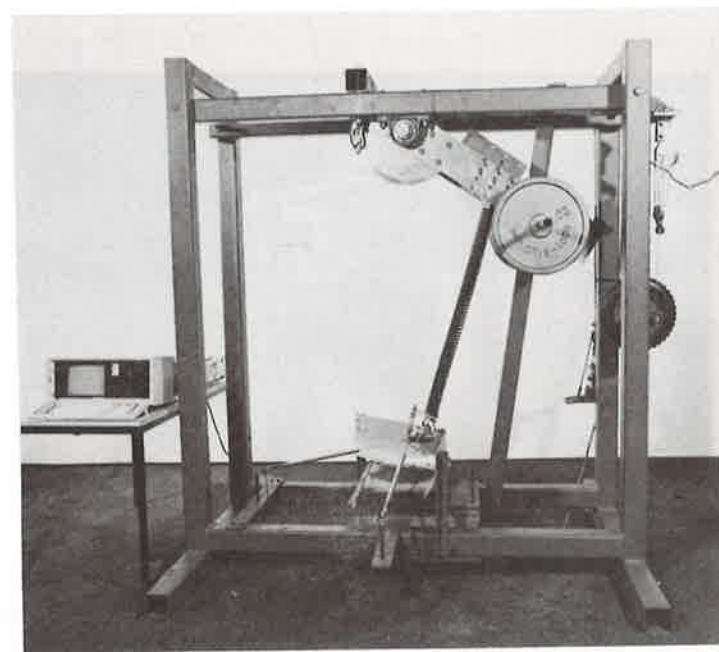
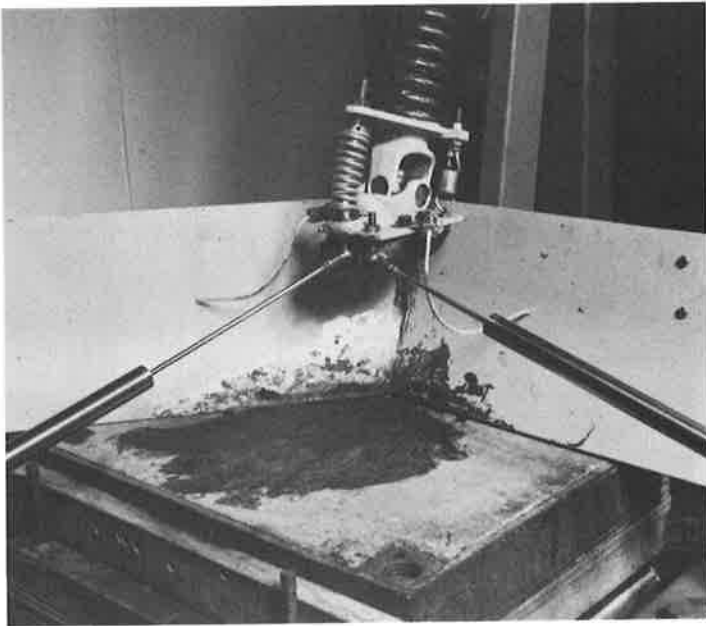Figure 4. Linear transducers attached to plastic foot for measuring horizontal movement.

Figure 5. Effect of position of lateral groove on slip magnitude.
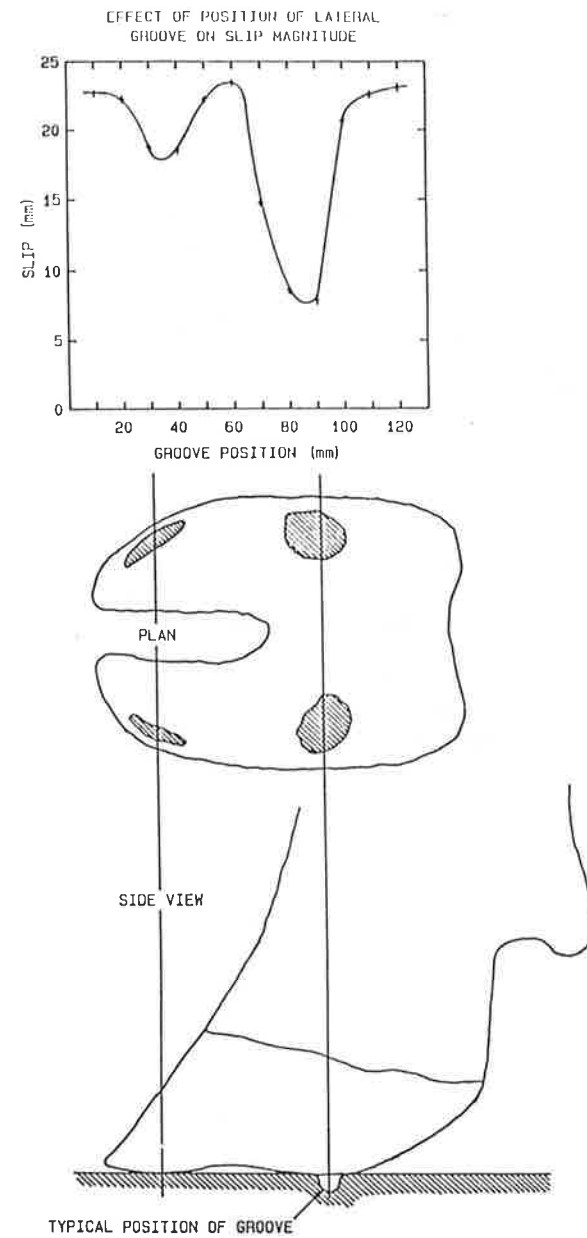

138

139

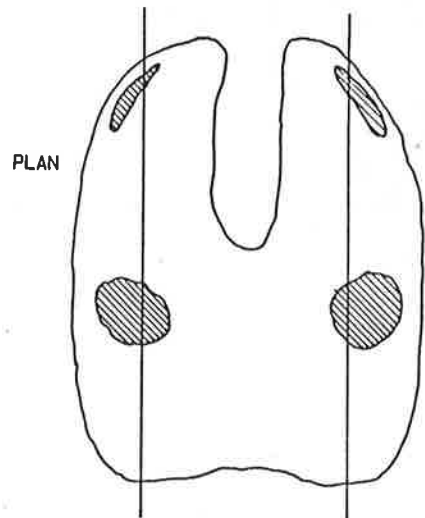Figure 6. Effect of position of longitudinal groove on slip magnitude.

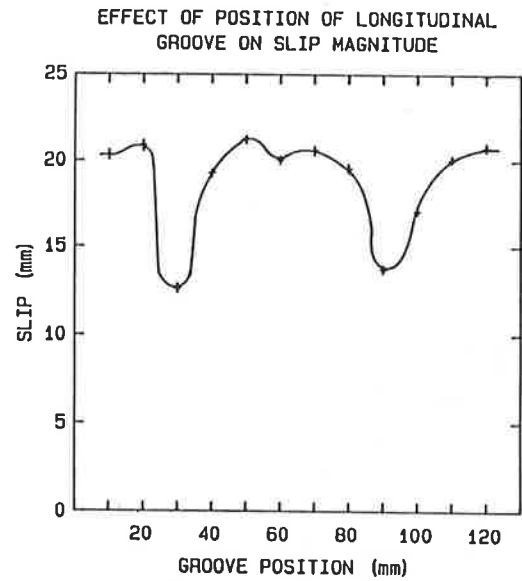EFFECT OF POSITION OF LONGITUDINAL
GROOVE ON SLIP MAGNITUDE

PLAN

Figure 7. Interaction between main foot contact areas and 40 mm groove spacing.
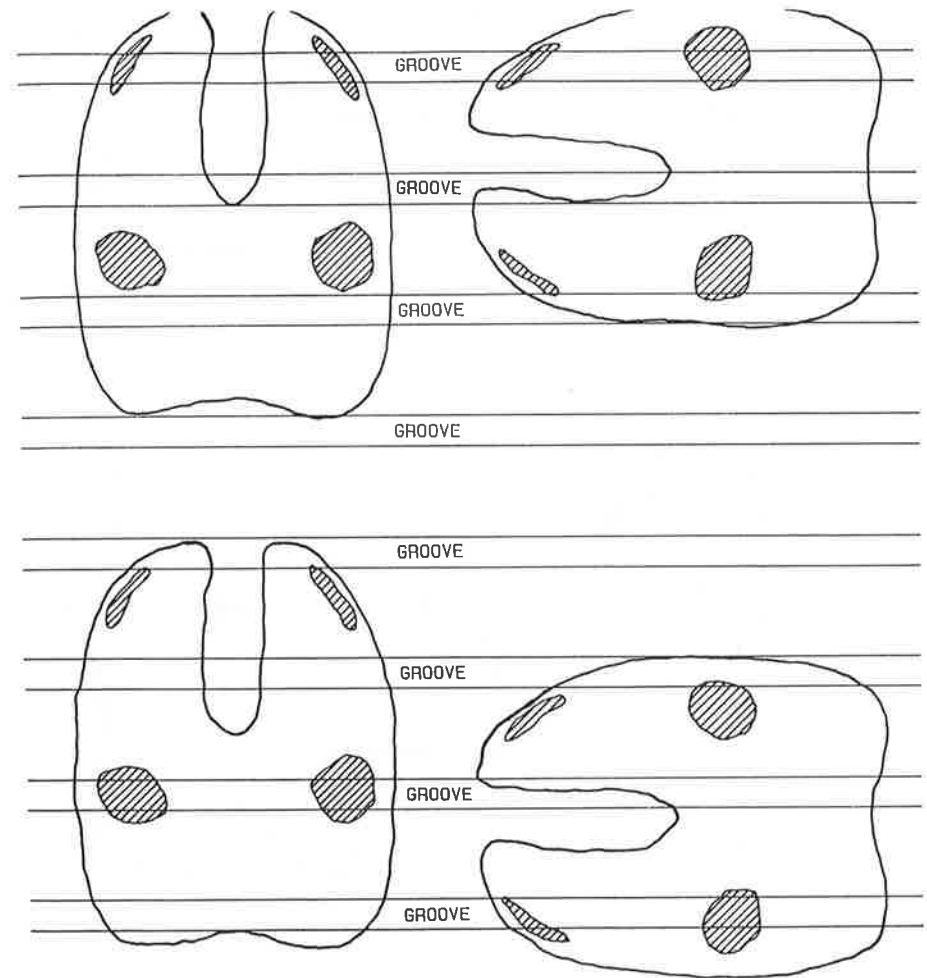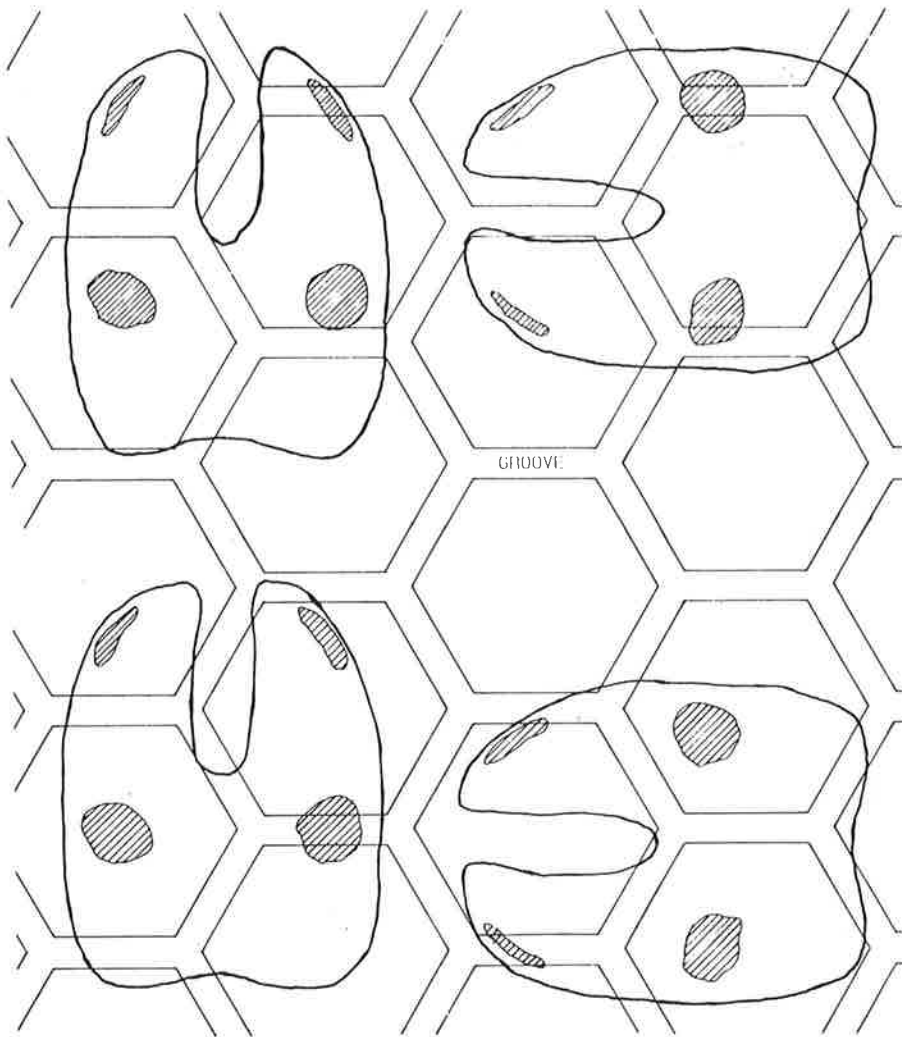
Figure 8.   Interaction between main foot contact areas and pattern

of hexagons with 46 mm sides.

Pedobarometric Forces At The Sole/floor Interface

O. Distl and A. Mair

Institut fur Tierzucht und Tierhygiene der Universitat Munchen Veterinarstrasse 13, 8000 Munchen 22 - FRG.

Introduction

Claw diseases are very frequent in cattle and represent after infertility and udder diseases one of the most important culling reasons. Claw diseases have relevant economic importance in cattle production. The causes of claw diseases are environmental factors and also a genetic predisposition by the morphology and physiology of the claws and limbs seems to exist. Claw characteristics, which measure the function of the supporting structures in the claw, should have a higher relationship to claw diseases in the claw than morphological claw parameters. Many claw diseases are caused by traumata of the dermis of the sole, which are sequels of an extreme local overload of the dermis of the sole (figure 1). In order to clarify the association between the local pressure on the sole, it is necessary to register the pressure distribution of the foot-to-ground contact area.

Measurement of the local pressure distribution in the ground surface of the claw can provide information on functional characteristics of all structures in claw for attenuation of pressure. Important structures for diminishing pressure are the horny capsule, digital cushion, ligaments and fibres. Also the position and surface of the distal phalanx and of the distal sesamoid bone can have considerable influence on the pressure distribution on the sole of claw. Also the influence on pressure distribution caused by different environmental conditions e.g. kind of floor and the position of limbs can be demonstrated.

The first measuring devices for the quantification of pressure distribution within the human-physiological area were presented by Morton in 1935 with his "Kinetograph". Wedge-shaped ribs on the bottom of a rubber mattress thereby produced on a suitable groundbase parallel lines of different widths, depending on the extent of pressure. Lord (1981) gave an overview on similar measuring processes.

Whilts Dhanendran et al. (1978) used fixed plates of a few centimetres thickness onto which strain gauges had been applied, Nicol and Henning (1978) developed flexible measuring mattresses according to the capacity measuring principle. Brunner et al. (1983) achieved a considerably higher area resolution in a further development of this process.

Other electronic measuring devices for pressure distribution are based on the Piezo effect (e.g. Cavanagh and Michiyoshi, 1980) or use inductive principles, as did Stumbaum and Diebschlag (1980) and Kurz et al. (1986).

In the case of animal-physiological pressure distribution measurements, Webb