participation of Papillomavirus in the pathogenesis of the disease.

PRELIMINARY REPORT ON THE ONSET AND EVOLUTION OF DIGITAL SKIN DISEASES IN A GROUP OF 32 HEIFERS

C.M. Mortellaro, S. Romussi, and G. Scavia

Università degli Studi di Milano
Clinica Chirurgica Veterinaria
Via Celoria, 10 20133
Milano, Italy

Introduction

Bovine Digital Dermatitis (D.D.) is characterized by erosive/ulcerative and proliferative changes of the skin bordering the plantar aspect of the interdigital space. However, interdigital skin itself may be sometimes involved and seldom represents the sole location of the disease. In this eventuality D.D. may frequently D.D. complicate a pre-existing Interdigital Hyperplasia. Clinical symptoms such as discomfort or lameness could accompany or not the disease. All these findings, commonly encountered, were observed in a herd that has experienced D.D. for many years. Young heifers were predominantly affected with an estimated prevalence rate of 21.7% (9 out of 42) in spring 1993.

The polymorphism of the disease was the main clinical feature in spite of the homogeneity of the housing and environmental conditions. Based on this observation we decided to carry out a survey on this herd in order to evaluate the possible onset of D.D., the evolution and finally the correlation between the antithetical (apparently) features of the disease.

Materials and Methods

The study was carried out between May 1993 and April 1994, in an intensive dairy farm on a group of 32 heifers. The criteria of inclusion were no previous contacts with animals affected by digital diseases, absence of clinical signs and symptoms related to digital diseases. During the study the group was kept under the normal farming conditions and neither preventive nor therapeutic measures were accomplished to control Digital Dermatitis. All the heifers underwent monthly a close inspection on the four feet. Pathological changes of digital skin, interdigital skin and horn were recorded. Heifers with serious lesions were re-examined two weeks later.

Data concerning variations of the diet and housing system were also collected.

Results

The overall number of clinical observations was 1540 carried out over 16 visits. Twenty-eight out of 32 heifers (58 out of 128 feet) were affected by some pathological changes. In 19 animals, lesions involved more than one limb. Several alterations were observed, including mild sclerosis of the transitional area between digital and interdigital skin, erosion/ulceration of digital-interdigital skin, finger-like keratinized projections. Erosions, fissures and underrunning were the main lesions of the heel horn. According with the current diagnostic criteria, D.D. was diagnosed in 19 feet. In 17 cases we observed the same evolution of the disease which can be summarized in the following steps.

1. Sclerosis/swelling of the plantar border of the interdigital cleft. Erosion and ulceration of the interdigital skin.
2. Erosion and ulceration of the sclerotic border.
3. Extension of the erosion/ulceration to the digital skin (erosive D.D.).

A granulating feature ("strawberry" D.D.) was subsequently observed in 13 cases among which two lesions progressed up to a "papilliform" aspect. Two cases out of 19 experienced a different evolution both for an atypical D.D. location and for a sudden onset of the ulceration.

Discussion and Conclusions

Since the results are still in progress no definitive conclusions can be drawn. However, owing to the great number of lesions showing the same progression (17 out of 19), the early stage of D.D. should be represented by mild sclerotic changes of the skin bordering the plantar aspect of the interdigital space accompanied or immediately followed by small erosions of the skin itself. The above-mentioned erosions are sometimes localized only in the interdigital space. Why these initial alterations may progress to a clear Digital Dermatitis or remain stable or finally may disappear is unexplained. Individual factors (e.g. abnormal skin defense mechanism) may play some role.

Concerning the correlation between the different morphological features of D.D., our results support

the hypothesis that they could be related to a different stage of evolution of the disease.

The low occurrence of lameness in the animals affected with the disease could explain a possible delay in the diagnosis of D.D. In such case, an early mild erosion lesion could progress up to proliferation and therefore the condition could be diagnosed, only at this time, as proliferative/papillomatous Digital Dermatitis.

EPIDEMIOLOGICAL INVESTIGATIONS OF DIGITAL DERMATITIS IN DUTCH DAIRY CATTLE

D. Döpfer[1,3], *A. Koopmans*[1], *F.A. Meijer*[1], *Y.H. Schukken*[1], *A. Brand*[1], *W. Klee*[3], *J.L. Cornelisse*[2]

[1] Dept. of Herd Health and Reproduction
Universiteit Utrecht
Netherlands

[2] Dept. of Bacteriology
Universiteit Utrecht
Netherlands

[3] Tierärztliche Hochschule Hannover
Fed. Rep. of Germany

The picture of digital dermatitis seems to have changed since 1974, moving towards milder, less painful, and dryer forms (MORTELLARO 1985). Increasingly hyperkeratotic processes can be observed, leading to chronical states of disease with recurrence (CHELI 1986, MERKENS et al. 1992).

In the present study a classification system for lesions caused by digital dermatitis has been developed. In order to study the dynamics of the course of disease the evolution of skin lesions from normal skin to the typical ulcerations and vice versa was studied. Early, typical, healing and suspect cases were differentiated.

A quantitative classification system was developed in order to compare the severeness of conditions in the bovine digit and to standarise the description of signs recorded in a longitudinal cross sectional study with multiple follow-up periods.

This scoring system allows a quantitative evaluation of the course of disease both on the individual and group level. Applying epidemiological methods, risk indicators for disease in the two study groups (2x45 Dutch dairy cows followed for 4 and 11 months respectively) have been described.

The gross clinical findings of this and other studies were compared to histological findings using silver stains for spirochaetes and an

immunofluorescence test for *Campylobacter faecalis* applied to smears of wound exudate.

The present classification and scoring systems promote a standardised discussion of the signs of disease and are a contribution to the disentanglement of this complex digital disease with unknown etiology and economic importance.

Literature:

CHELI, R., and C.M. MORTELLARO (1986):
Digital Dermatitis today and tomorrow.
in: 5th International Symposium on Disorders of the Ruminant Digit, Dublin, Ireland 1986. Proc., p. 8-13. Weaver, A.D. (editor), College of Veterinary Medicine, University of Missouri, Columbia/USA.

MERKENS, H.W., H.R. SUICHIES, E.N. NOORDHUIZEN-STASSEN, and T.G.J.M. LAM (1992):
Dermatitis digitalis? The effect of treatment in a chronically contaminated farm.
in: 7th International Symposium on Disorders of the Ruminant Digit, Rebild Denmark 1992, Proc., (in press)

MORTELLARO, C.M., R. CHELI, E. TOUSSAINT RAVEN, and J.L. CORNELISSE (1985):
Dermatitis digitalis bij het rund.
Pro Veterinario, 3, p. 11.

PRELIMINARY RESULTS FROM A SPRAY APPLICATION OF OXYTETRACYCLINE TO TREAT, CONTROL, AND PREVENT DIGITAL DERMATITIS IN DAIRY HERDS

J.K. Shearer and J.B. Elliott

College of Veterinary Medicine
University of Florida
Gainesville, FL, U.S.A.

Despite aggressive treatment of individual animals and the rigorous use of foot baths in herds, digital dermatitis persists as a cow and herd problem. We hypothesize that one reason for its persistence in herds may be that, in general, only animals with visible lameness or lesions undergo treatment. In reality, many more animals are affected but show no evidence of lameness or lesions. We proposed that periodic topical treatment of all feet of all exposed animals would have the effect of treatment, control, and prevention of the disease depending upon the animal's disease status. To test our hypothesis we formulated an antibiotic preparation for topic application containing 25 mg/ml of oxytetracycline in 20% glycerine and deionized water. This preparation was delivered as a spray onto the heels of all cows (N=350) in the herd once daily for 5 days using a garden-type spray applicator. Of 89 animals affected with visible lesions on the rear feet and 5 animals with lesions on the front feet, nearly all were visibly improved after a 5-day course of therapy. Samples of milk and urine were assayed from individual animals and herd bulk milk. All samples tested negative for antibiotic residue. Work is continuing in order to determine optimal drug concentration and treatment frequency.

EXCESSIVE DIETARY PROTEIN AS THE CAUSE OF HERD OUTBREAKS OF "MORTELLARO DISEASE"

Uri Bargai

The Hebrew University of Jerusalem
Koret School of Veterinary Medicine
P.O. Box 12
Rehovot, Israel, 76100

"Mortellaro disease" (digital dermatitis) is very rare in Israel. It was never recorded, and if recognized, it is only seen in an individual cow. During 1993, three outbreaks of the disease occurred in different dairy herds, which were investigated fully. All forms of the disease were manifested: inflammatory, ulcerative and proliferative. About 30% of the affected cows were studied by laboratory for hematology and clinical pathology findings.

Laboratory results revealed universal hyperproteinemia due to elevated albumin values and extremely high blood urea levels. Several elevated liver enzymes were found in some of the cows. Investigation into the management indicated a definite association with extremely high levels of dietary protein in all three outbreaks. In two of these, these levels were recommended by a feeding counselor. In the third outbreak those levels resulted from a feeding accident. It was concluded that excessive dietary protein may be the etiologic agent in "Mortellaro disease" in cattle.

THE MINIMAL SOLUTION FOOTBATH - AN AID TO TREATMENT OF DIGITAL DERMATITIS

W. Robert Ward

Liverpool University
Leahurst, Neston, S Wirral, L64 7TE, U.K.

Digital dermatitis (Mortellaro disease) has become a widespread nuisance in UK since it was first reported (Blowey & Sharp, 1988). Surveys show that sole ulcer and white line disease remain the two most common foot lesions, but at meetings farmers keep asking about digital dermatitis. A common treatment is the use of footbaths containing a tetracycline 2-4g/litre (Blowey, 1990) or 6 g/litre (Blowey, 1992), or other antibiotics, which are more costly than formalin or copper sulphate. Mike Stables, a farmer from North-West England, has designed a footbath that holds much less liquid, and therefore saves antibiotic. It is now manufactured by Paxtons of Nantwich, Cheshire.

The footbath's shell is the same as that of the traditional type, but a soft foam base lies below a waterproof membrane. When a cow steps in the bath, the liquid moves to bathe her feet. The bath needs only 10-15 litres of liquid, compared with 125-200 litres in a traditional footbath, and then about 4 litres for every 25 cows. Cows appear to walk willingly through the bath.

Treatment with oxytetracycline 8g/litre twice on day 1 was compared with weekly treatments using copper sulphate 5% and an untreated control group in an 11-week trial using 57 cows at Harper Adams College, Shropshire (Crighton, unpublished). The cows' locomotion score (Manson & Leaver, 1988) in both treated groups in weeks 2, 3 and 4 were significantly better than in the control group ($P<0.05$), and not significantly different from each other. In both treatment groups locomotion score deteriorated after week 5, but remained better than in the control group.

David (1993) reported severe cases of foul (interdigital phlegmon) in three herds, one of which was using the MS footbath. Contamination might occur more quickly in a small volume of antibiotic than a large volume of formalin.

In one herd, a knapsack sprayer was used to apply lincomycin with spectinomycin to the heels of cows in the parlour, and the prevalence of digital dermatitis reduced rapidly.

Further trials are needed to compare different antibacterial agents and treatment regimes.

References:

Blowey, RW (1990) Digital dermatitis control. Veterinary Record, **126**, 120 (letter)

Blowey, RW (1992) Digital dermatitis. Veterinary Record, **131**, 39 (letter)

Blowey, RW, Sharp, MW (1992) Digital dermatitis in dairy cattle. Veterinary Record, **122**, 505-508

David, GP (1993) Severe foul-in-the-foot in dairy cattle. Veterinary Record, **133**, 567-568

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

IN SEARCH OF AN EPIDEMIOLOGIC APPROACH TO INVESTIGATING BOVINE LAMENESS PROBLEMS

Paul R. Greenough Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Sask CANADA S7N 0W0
Jos Vermunt Faculty of Veterinary Sciences, Massey University, Palmerston North, New Zealand.

INTRODUCTION

The objective of this paper is to recommend a practical approach to managing herd lameness problems. The system involves a series of logical steps that a veterinary practitioner may take in order to gain insight into the predisposing causes of the lameness and be able to develop a rationale that will ameliorate the problem.

Maintaining health records has become an established requirement for herd health programs. The inclusion of special, appropriate or adequate lameness data is not necessarily part of routine record keeping. Many systems will yield a diagnosis of 'foot rot' or 'lame'. If no herd problem exists the owner/veterinarian is likely to be unwilling to record more information. Unfortunately, the transition from a situation where there are a few cases of individual cow lameness to one in which a herd problem exists can occur imperceptibly. Historical data tends to increase awareness and is invaluable in establishing the general characteristics of a problem. Elaborate record keeping is only desirable when lameness becomes a serious herd problem. There are several levels of lameness record keeping that are suggested in this paper.

LEVEL ONE - Routine Lameness Monitoring

A data capture sheet may be used that is similar to that designed for the University of Liverpool study. It is essential to quantitate the quality of the individual incidents of lameness. The following system is simple to use.

Locomotion scoring system
(after Manson & Leaver, 1988)

1.0 Minimal abduction/adduction, no unevenness of gait, no tenderness
1.5 Slight abduction/adduction, no unevenness of gait or tenderness
2.0 Abduction/adduction present, uneven gait, perhaps tender
2.5 Abduction/adduction present, uneven gait, tenderness of feet
3.0 Slight lameness, not affecting behaviour
3.5 Obvious lameness, some difficulty in turning, not affecting behaviour pattern
4.0 Obvious lameness, difficulty turning, behaviour pattern affected
4.5 Some difficulty in rising, difficulty in walking, behaviour pattern affected
5.0 Extreme difficulty in rising, difficulty in walking, adverse effects on behaviour

A score of 3 or above was used in the Liverpool study as an indication of lameness. However, it would appear preferable to use only the scores over 3.0 rather than struggle with 9 different scores which would be open to conflict between observers.

It is essential that an accurate diagnosis shall be recorded. The encoding system suggested in the following table would tend to induce discipline in the use of already accepted International Nomenclature and

thereby avoid confusion at a later date. The codes used would be invaluable if, at a later date, an epidemiologic study was required.

Proposed lesion coding system

01.	Hemorrhage of sole	12.	Interdigital fibroma
02.	Sole ulcer	13.	Digital dermatitis
03.	White line disease (region 2)	14.	Distal interphalangeal joint sepsis
04.	White line disease (region 1)	15.	Retroarticular abscess
05.	Heel erosion	20.	Vertical fissure
06.	Worn sole	21.	Horizontal fissure
07.	Double sole	22.	Normal overgrowth
08.	Sole trauma	23.	Slipper foot
09.	Sole abscess	24.	Corkscrew claw
10.	Foot rot (foul)	30.	Hardship groove
11.	Interdigital dermatitis	31.	Reaction ridge

Identifying a herd problem

We can attempt to measure the extent of a problem by expressing the number of animals lame in a given year as a percentage. There is no general agreement on the significance of the percentage. However, in this paper it is suggested that it may be useful to consider the following criteria. This concept is not to be interpreted as a 'epidemiology of lameness cookbook' but to be considered as an approach to identifying the point at which herd lameness can be considered to exist.

Lameness of mixed etiology (excluding Interdigital Phlegmon) up to 5%

This probably represents indiscriminate lameness incidents, including accidents, that do not warrant special preventive protocols.

Lameness resulting from one etiologic entity

- Interdigital Phlegmon frequently affects a large percentage of a herd and usually is a problem that is distinct from other forms of lameness.

- In recent years Digital Dermatitis has been recognized as a herd problem that is also distinct from other forms of lameness. However, it wise to commence instituting control measures whenever the incidence exceeds 10%.

- Interdigital Dermatitis may, in mid winter, affect 50% of the herd but only be clinically relevant in less than 5% of the herd. When combined with heel erosion, the resulting phenomenon will assume economic significance if lameness is observed in 2% of the animals in any given month.

Lameness resulting from lesion associated with laminitis.

The conditions listed 01 to 07 plus 23,30 and 31 in the proposed lesion coding system are all considered to be associated with an underlying incidence of sub-clinical laminitis. When the collective annual incidence reaches 10%, the herd should move to the level of problem surveillance.

The annual incidence of lameness will trigger an awareness that a problem exists. However, it should be born in mind that any high incidence in a measurable period of less than a year should trigger an emergency response.

At the monitoring level information regarding frequency and type of treatment as well as age

and other descriptive information would be available from general health records. The important issue is that the veterinarian should not remain ignorant of the magnitude and characteristics of a problem that exists.

LEVEL TWO - Problem Surveillance

Having identified that a problem exists, a veterinarian has two choices: a) to gather more detailed information at the level of individual animals or b) to proceed to the third level of problem investigation. The choice depends on a number of factors such as, the size of the herd, the wishes of the client and the potential economic impact of the problem condition.

The surveillance should establish which groups of animals are most severely or first affected, and in what geographic location, if any, were they affected. When were the animals affected in respect to age, to parturition and time of year? The data capture sheet designed for the Liverpool University Study is a particularly valuable instrument for this work.

The objective of the surveillance is to measure the severity of the individual lesions and where applicable describe them objectively.

Lesion severity grades

Grade 1. A lesion exists but is not significant at the stage at which it is observed. For example, an animal may have a lameness-producing sole ulcer on one claw. If the contralateral limb is examined, a similar lesion may be seen developing.

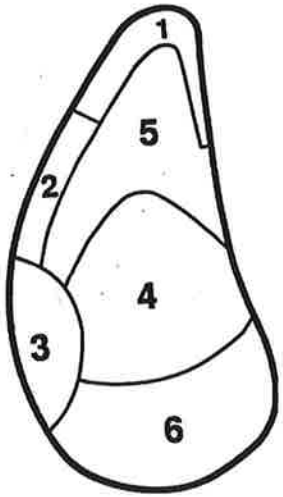
Grade 2. The lesion is obvious and causing grade 3 lameness. The prognosis is good.

Grade 3. The lesion is extensive, causes severe lameness, requires treatment by a veterinarian over a period of time or the prognosis may be uncertain.

Grade 4. This grade should be reserved for really serious cases that are usually complicated.

The regions of the sole

The following diagram of the sole which is a modification of several previous outlines was proposed as an International Standard at the 7th Symposium in Rebilt and has since been used by seceral authors. These regions are, of course, useful to research workers but they also can and are used by practitioners seeking advice from experts.



Zones of the sole amended to conform with recommendations established at the 6th Symposium on Diseases of the Ruminant Digit, Liverpool, 1990. Zone 1 White zone at the toe, Zone 2 Abaxial white zone, Zone 3 Abaxial wall-bulb junction, Zone 4 Sole-bulb junction, Zone 5 Apex of the sole, Zone 6 Bulb

LEVEL THREE - Problem investigation

At this stage the problem has been defined and it has been established that it is of very significant economic importance. Most, if not all, problem investigations will relate to sub-clinical laminitis in dairy cattle. The objective now would be to identify the combination of factors that are of most importance in herds under study. The following steps are suggested.

A: Establish when the insult occurred

For example, sub-clinical laminitis may be identified as occurring in young cattle. The causative insult may be associated with a managemental problem occurring between 8 and 20 months of age. The presence and location of hardship grooves or reaction ridges can be used to identify the time of the insult. The degree of severity of sole hemorrhages can be scored. These scores will assist the investigator in determining the severity of the insult or the period over which it was present. The following scale was used in an earlier study (Greenough and Vermunt 1991).

1. Slight discoloration
2. Moderate hemorrhage
3. Severe hemorrhage
4. Exposed corium

In mature animals the presence of hemorrhages can also be a useful indicator of the time at which the major insults occurred. A serious hemorrhage of the sole is observed more or less three months after the event that caused it.

B: Relate time frame to data collected

C: Evaluate potential stressors

Herd Lameness Check List

PREDISPOSING FACTOR AND DESCRIPTION	MILK COW	DRY COW	HEIFER
ENERGY TDN 70% = 0 points + 1 point for each 1% more			
PROTEIN 16% dry matter = 0 + 1 point for each 0.5% more			
FIBRE 35% dry matter = 3 points + 1 point for each 2% less			
WATER pH greater than 8.5 add 1 point per 0.1 more pH less than 4.0 add 1 point per 0.1 less Nitrate greater than 100mgs/litre add 1 point for each 50 mg greater. Sulphate greater than 800mgs/litre add 1 point for each 75 mg greater.			
RATION MANAGEMENT Total Mixed Ration deduct 4 points 5% change in any component of ration = 1 point Days change dry cow to production ration 0 days = 7 points - 1 point for each day of gradual change Increase in rate of concentrate feeding during first 16 days post partum 500 gms per day = 0 pts + 1 pt per 200 gms			
NITRATE USE (nitrogen or nitrate fertilizer) Use of nitrates or heavy manuring of forage crops 1 to 5 points. Presence of nitrates in forage 1 point per 0.05% Over 0.5% (as KNO3, dry weight)			
FOOT BATH Deduct 1 point for each use in 12 months no use + 5 points			
CLAW TRIMMING Twice each year deduct 5 points Once each year deduct 2 points No trimming add 5 points			

PREDISPOSING FACTOR AND DESCRIPTION	MILK COW	DRY COW	HEIFER
MANAGEMENT Straw yards, deduct 1 pt per month in yards Stanchion\Tie Stall system = 0 Loose stall\cubicle = 7 points PLUS Slurry management factor 1 - 6 points Loafing area 1 point for each .1 sqm less than 4 sq meters per cow Cubicle size\cow comfort factor 1 - 5			
REPLACEMENT REARING Heifers Average daily weight gain over 800 grms per day after six months of age to breeding at 15 months 1 point per 50 gms per day. Average first calving at 30 months = 0 + 1 point per month less than 30 months. Introducing heifers individually to the dry group = 5 points Observed confrontation/reduced movement of 1 in four heifers = 5 points			

The foregoing table is not intended to be comprehensive or for that matter accurate. What it does do is create an awareness of the multiplicity of factors that can be implicated in an incident of subclinical laminitis.

DISCUSSION

This paper has been somewhat anecdotal. We have used ideas that have arisen from discussions at previous symposia. Many of these ideas were never documented but neither were they forgotten. The fact is that lameness is becoming more, rather than less, of a serious problem. Over the next few years we will be obliged to develop a scientific approach to information gathering and data analysis.

Figure 1. **INDIVIDUAL COW FOOT RECORDING SHEET**

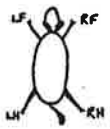
Please complete a separate sheet for each foot that you pick up

Farm: [redacted] Date: 2/2/90

Treated by: Stockman. Cow no: 101

Lameness	Trimmed but not lame	Slight	Moderate	Severe
			✓	

Foot/Leg

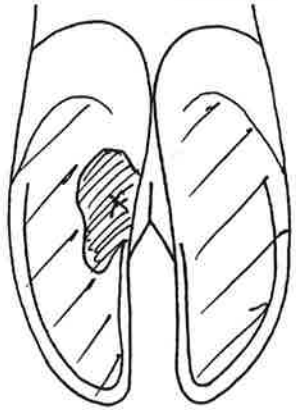


RF	
RH	
LF	
LH	✓

Condition of claws

Normal	
Both Overgrown	✓
Unequal Size	
Rotation and narrowing of toe	

Solar view (please distinguish inner from outer claw)



Comments

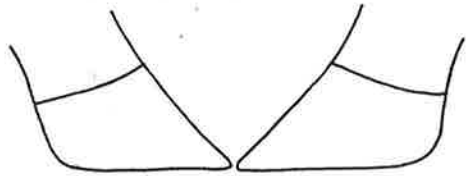
Ulcer

Treatment

Purple spray

Side view of affected claw

(please distinguish: inner or outer wall)



X Site of main lesion

Extent of horn/skin directly associated with any lesion, or which is bruised.

Other areas of the claw/foot from which horn has been removed in order to correct foot shape etc.

DATA CAPTURE SHEET - INDIVIDUAL COW FOOT RECORD

re 2.

Description	Code		Column
Record Type	0 2		1 - 2
Herd	[redacted]	See attached sheet for code	3 - 4
Cow	0 1 0 1	Freeze brand no. (9999 if unknown)	5 - 8
Date	0 2 0 2 9 0	Day, Month, Year. (99 if any unknown)	9 - 14
Treated by	2	1 = vet, 2 = stockman, 3-5 = contractors 6 = student, 9 = unknown	15
Degree of lameness	3	1 = trimmed but not lame, 2 = slight 3 = moderate, 4 = severe, 9 = unknown.	16
Leg lesion	0	1 = right fore, 2 = left fore, 3 = right hind 4 = left hind, 9 = unknown, 0 = none.	17
Foot lesion	4	1 = right fore, 2 = left fore, 3 = right hind 4 = left hind, 9 = unknown, 0 = none.	18
Condition of claws	2	1 = normal, 2 = both overgrown, 3 = unequal size 4 = corkscrew, 9 = unknown.	19

20 - 67

	I. C	Site	Lesion	Treatment
Claw				
	O. C	**Site**	**Lesion**	**Treatment**
	1 3		0 1	1 2 0 4
Skin		Site	Lesion	Treatment

RECENT STUDIES ON THE EPIDEMIOLOGY OF LAMENESS

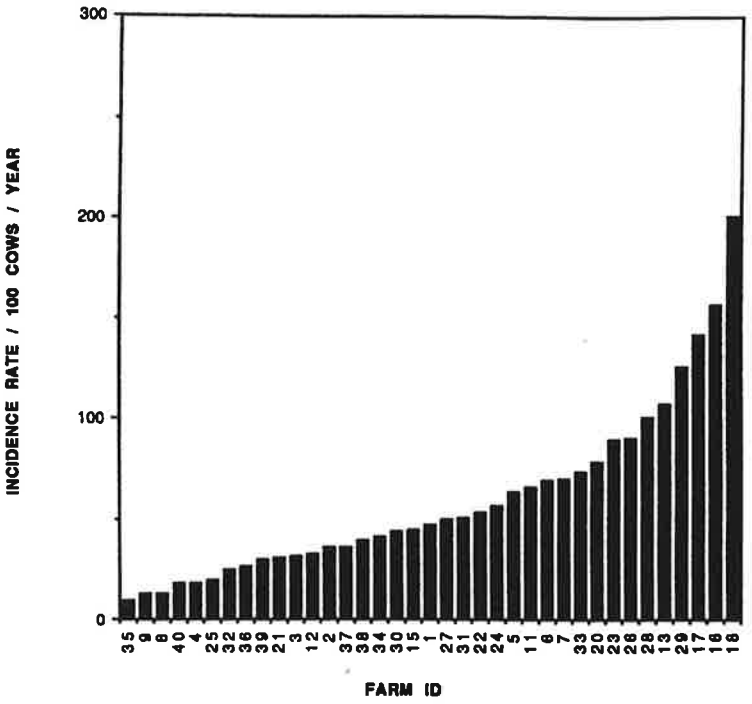
W. Robert Ward
Liverpool University, Leahurst, Neston, South Wirral, L64 7TE, UK

An epidemiological study on 37 farms in England and Wales over three years showed a mean incidence of almost 60 new cases of lameness per 100 cows per year, and a mean prevalence of over 20%. The most common lesions were sole ulcer and white line lesion. When the farms were ranked according to the incidence and prevalence of lameness, significant risk factors included long toes, high heels; grass silage with low dry matter and fibre, and high protein; cubicles with high kerbs, lack of bedding, lack of borrowing space, and no head-rail; and yards with smooth concrete. Separate studies showed that lameness in summer was more prevalent when farmers made cows rush along tracks, and when farmers were less knowledgeable, less well-trained, and less aware of lameness. We believe that the incidence and prevalence of foot lameness in UK dairy cows is a most important economic and welfare problem, but that we can now advise effectively on its reduction.

Incidence of foot lameness.

The number of cases of foot lameness was measured by asking each person who treated a lame cow to complete a form showing a diagram of the foot, and describing the lesion and treatment. (Clarkson and others 1993). Michael Clarkson analysed the data with David Downham and Wanda Russell who assisted with analysis of data from the whole study. The mean number of cows at risk over the three years was 4,230, and 6,521 cases of lameness occurred. The incidence rate averaged 59.5 new cases per 100 cows per year, and ranged from 9.3 to 200.7. (A new case was one that occurred more than 28 days after a previous case, or occurred in a different digit.) On average each lame cow suffered 1.5 episodes of lameness in a six-month period.
The mean incidence of lameness was higher in the winter (34.6 cases per 100 cows per winter) than in summer (24.9 cases per 100 cows per summer).Winter was defined as November to April, to reflect the period when UK cows are usually housed, and summer as May to October. In seven of the 37 farms, however, the incidence was higher in summer than in winter, while in three the incidence was the same in summer as in winter.
On farms where cows had many lesions in the claw, the cows also tended to have many lesions in the skin ($r^2 = 0.5$). This surprising finding suggests some common factors, which could include wet conditions, and poor stockmanship.
The incidence of lameness is higher than in previous reports, but it is noticeable that the most recent other report (Esslemont and Spincer, 1993) also showed a high incidence (36%) in UK dairy herds. It seems likely that lameness in UK dairy herds has indeed become more common in recent years, to a point where it is a major economic and welfare problem. The large range in incidence shows that some farms have 'got it right',

Fig 1. Ranked mean annual incidence rate



and if we can analyse what the 'good' farms are doing correctly, we should be able to tell the 'bad' farms what to do.

Prevalence of foot lameness.

The prevalence of lameness was assessed during visits to the farms by trained observers (Felicity Manson and Jane Merritt), using the method of locomotion scoring of Manson and Leaver (1988), in which a lame cow is scored 3 - 5, tender feet 2, and normal gait 1. The mean prevalence over the three years was 20.6%, ranging from 2.0 to 53.9%. A single locomotion scoring of a herd in the middle of summer or in the middle of winter was found to be closely correlated to the mean prevalence calculated from a series of observations during the summer or the winter (r^2 = 0.7 - 0.8).

Locomotion scoring gave an independent assessment of the amount of lameness in a herd. In most herds the prevalence of lameness was related to the incidence in the usual way, that is, by the mean duration of a case of lameness. In a few herds the incidence of lameness was much lower than would be expected from the prevalence measured by independent observers, and it is probable that in these herds some cases of lameness were not noticed or recorded by the farmer. In some herds the incidence was higher than would be expected from the prevalence, and here we believe that the farmers were particularly keen to observe lame cows early, and the veterinarian treated cases very efficiently, so that the mean duration of lameness was low.

A single locomotion scoring in the middle of each summer and each winter can clearly provide a valid estimate of the amount of lameness in a dairy herd.

Foot lesions.

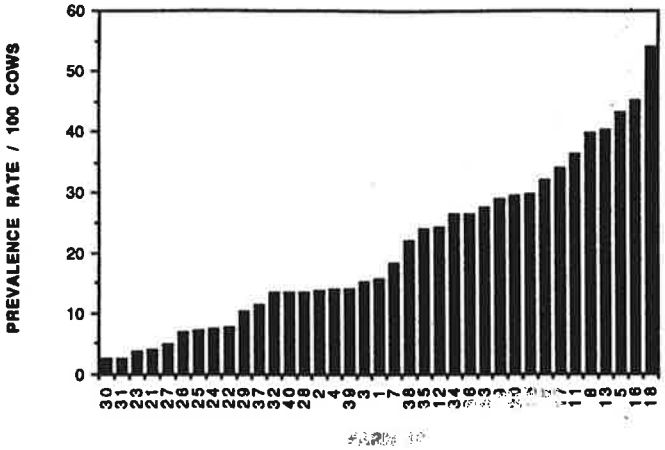
Nearly 9,000 lesions were diagnosed. Information from the forms filled in on the farms was transferred by Richard Murray, Felicity Manson and Bill Faull. On occasions, two significant lesions were found in the same foot. As in previous studies, a very high proportion of lameness occurred in feet (99%) and a high proportion of the foot lesions were in the hind feet (92%). In the hind feet, 68% of lesions occurred in the outer claw, 12% in the inner, and 20% in the skin. In the front feet, 46% occurred in the inner claw, 32% in the outer, and 22% in the skin.

Sole ulcer accounted for 28% of lesions, to which can be added local sole bruising in the ulcer area (8%) making a total of 36%; white line lesions came to 22%. These two conditions together therefore account for over half of all the foot lesions. The next most common problems were digital dermatitis, which made up 8%, and foul-in-foot, interdigital hyperplasia and foreign body, which each amounted to 5%. Heel horn erosion was described as the main lesion in 4% of cases, heel abscess in 3%, under-run sole, horn overgrowth and generalised laminitis each in 2% of cases. Interdigital dermatitis was found in 1%, sandcrack in 0.4%, and all other lesions together amounted to 4%.

Foot shape and claw trimming.

Trained observers (Felicity Manson and Jane Merritt) visited the 37 farms regularly, and assessed the shape of one hind foot

Fig 2 Ranked mean prevalence rate for whole period



while the cows were in the milking parlour. Pilot studies showed that this was a reliable method. Over 25,000 observations were made, and analysed by Richard Murray and David Downham. Toe length and heel height were significantly related to the prevalence of lameness. A cow with long toes would be likely to have a locomotion score 0.6 higher than one with short toes, while a cow with high heels was likely to have a locomotion score 0.2 higher. Deviation of the wall (corkscrew claw) was not significantly related to locomotion score.
Claw trimming was carried out as a preventive measure on almost 4,000 claws, mainly by farmers, with a small number by professional claw-trimmers. The shape of the feet improved after trimming, but the locomotion score did not always do so. There was some evidence that trimming feet just before the cows were turned out to pasture may have worsened locomotion score.
We conclude that foot shape is an important risk factor in lameness in UK dairy cattle. The shape of the foot is determined partly by genetics, and we are now analysing the genetic input. Based on the work of Russell(1987), it should be accepted that foot lameness has a genetic component. Foot shape is clearly affected by claw-trimming, and the disappointing locomotion scores in some herds after trimming suggests that people who trim cows' feet should be trained, and that trimming just before turn-out can be damaging.

Feeding.
The effects of feeding are discussed elsewhere in this Conference.

Environment.
In preliminary studies we attempted to find objective methods to measure walking and lying surfaces, but these proved unsatisfactory. Surfaces were therefore scored by Bill Faull and John Hughes, using eyes, hands and knees!
Indoor.
All the cows lived in cubicles during the winter, though on some farms some cows lived in straw yards for periods.
Observations of Holstein-Friesian cows on pasture showed that they need 2400 x 1200mm (8 x 4 feet) lying space. Most of the cubicles (87%) were too short(<2300mm), and 31% were too narrow (<1150mm); 19% were too wide(>1220mm), and 91% of top rails were too low (<1110mm), both of which tempt cows to turn round. The bottom rails were also unsatisfactory, which can lead to cows being trapped and injured: 22% were too low (<340mm) and 48% too high (>400mm). Although we believe that the cubicles are unsatisfactory in limiting the cows' lying space, we were unable to show significant relationships between the size of cubicle and the amount of lameness.
Kerb height was, however, related closely to lameness. The prevalence and incidence of lameness increased as the kerb height increased. Only 4% were satisfactory (<160mm, 6 inches), and 76% were classed as very high (>200mm, 8 inches).
The amount of bedding was classified as satisfactory in only 26% of the cubicles, and in 11% there was 'next to none'. Farms with more bedding on the cubicles had significantly less lameness.
Borrowing space was assessed as a measure of the ease with which a cow could use space in an adjoining cubicle for her head.

Cubicles with wide wooden divisions, for example, provided no borrowing space (6% of cubicles), 82% provided some, and 12% provided lots. There was significantly less lameness in one of the two winters in farms where the cubicles provided more borrowing space.
Farms with very smooth concrete surfaces (33% of observations) or smooth surfaces (55%) had a significantly higher incidence of lameness.

Outdoor.

At the beginning of the study, lameness was believed to be almost entirely a problem of housed cattle. When we measured the incidence and prevalence of lameness in the summer, however, we found that some farms had more lameness in the summer.
Walking surfaces were classified as rough or very rough in 70% of cases. There was no statistically significant relationship with lameness, but the mixture of mud, broken bricks, slates, and rubbish around some of the water troughs and in some gateways was clearly not a satisfactory environment for dairy cows.

Stockmanship.

A separate study at Liverpool (Clackson and Ward, 1991) showed that lameness in the summer was significantly higher when the farmer pushed cows along tracks, rather than allowing them to find their own way.
Another study showed that farmers' knowledge, training and awareness were each closely related to a reduction in the prevalence of lameness. (Mill and Ward, 1994)

Appraisal of farms.

The Liverpool studies, and previous work, suggest that we have developed methods of measuring what we now know to be the key risk factors in a dairy farm. In a recent study, we attempted to put this into practice on 14 dairy farms (Diamond, Murray and Ward, unpublished). The prevalence of lameness ranged from 3 - 29%, and the estimated incidence from 2 - 82%. The prevalence of lameness was significantly correlated with an overall score for stockmanship, based on Clackson and Ward (1991), and Mill and Ward (1994) ($P<0.01$),with an overall score for housing based on the Liverpool study ($P<0.02$), and with treatment of cases by farmers rather than be veterinarians ($P<)>01$). The lameness prevalence was not, however, found to be related significantly to claw trimming, use of footbaths, breeding policy, nor feeding. In general, larger herds had more lameness, but against this trend the biggest herd, which housed the cattle in a straw yard instead of cubicles, had the lowest prevalence of lameness.

Conclusions.

In UK, lameness in dairy cattle remains predominantly a problem of sole ulcer and white line lesion in the outer hind claw. The total amount of lameness is enormous, and represents a major economic and welfare problem. The prevalence and incidence varies hugely between farms, suggesting that it does not have to remain a problem. Housing, foot shape (which may well have a genetic component), and stockmanship are clearly strongly related to the prevalence of lameness.Outdoor environment and feeding are less clearly shown to be major risk factors. Claw

trimming appears to be a possible risk factor unless done expertly and at the correct time.

Recommendations.

1. Any dairy herd with a suspicion of a lameness problem should have the prevalence assessed by a single locomotion scoring in the middle of the summer, and the middle of the winter.
2. Herds where cows live in cubicles should take expert advice on improving the cubicles or replacing them, either with large well-designed cubicles or with well-designed straw yards.
3. Bulls should be selected partly on information about lameness in their daughters.
4. Feet should be trimmed annually, by an expert, at a time when the cows will be housed on soft bedding or on pasture, and not when they will be driven over stony roads.
5. Farmers should be encouraged to learn more about foot lameness, and to let cows walk along roads at their own speed.

Acknowledgments.

The Liverpool study was carried out by a large team of people, some mentioned in the text, and in addition, DW Collick, TJ Briggs, N.M. Howie, DE Lewis, PEJ Gwynn, R Wilkinson, and GP David, and was funded by Ministry of Agriculture Contract. The Wellcome Trust and Universities Federation for Animal Welfare funded vacation scholarships.

References.

Clackson, DA, Ward, WR (1991) Farm tracks, stockman's herding and lameness in dairy cattle. Veterinary Record, **129**, 511 - 512.
Clarkson, MJ, Downham, DY, Faull, WB, Hughes, JW, Manson, FJ, Merritt, JB, Murray, RD, Russell, WB, Sutherst, JE, Ward, WR (1993) An epidemiological study to determine the risk factors of lameness in dairy cows. (Ref; CSA 1379). Final Report.
Esslemont, RJ, Spincer, I (1993) The incidence and costs of diseases in dairy herds. Report No. 2, DAISY - The Dairy Information System, University of Reading.
Manson, FJ, Leaver, JD (1988) The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Animal Production, **47**, 185 - 190.
Mill, JM, Ward, WR (1994) Lameness in dairy cows and farmers' knowledge, training and awareness. Veterinary Record, **134**, 162 - 164.
Russell, AM (1987) The influence of sire on lameness in cows. Proceedings for 1986-87. British Cattle Veterinary Association, 213 - 219.

CORRELATIONS OF LOCOMOTION AND BODY AND CLAW TRAITS WITH REGARD TO PRODUCTION IN DAIRY CATTLE

Dorothee Boelling

Agriculture Section
Wye College
University of London
Wye, Ashford, Kent, TN25 5AH, U.K.

For this study, data were collected on 126 HF-cows (68 cows lactations 1. - 3., 58 cows lactations 4. - 11.) in the Wye College Research Unit. These included locomotion score, rear leg side view (RLSV), udder size, claw measurements, production data and lactation number. The younger cows had a significantly better locomotion score than the older cows. Their feet were also smaller, though the foot angle was the same in both groups. The claw trait diagonal showed the best correlation with locomotion, with a coefficient of 0.2. The relationship between the locomotion score with RLSV and udder was 0.34 and 0.41 respectively, showing that the older cows had more sickled legs and larger udders than the younger ones. No significant correlation between production data and locomotion could be found, as the average total milk, fat, and protein yields were the same in both groups. An LSQ-analysis revealed that number of lactation, RLSV and diagonal were the only significant factors influencing locomotion. It is concluded that the change in body conformation due to age has the most notable effect on locomotion.

A SURVEY OF DIGITAL DERMATITIS TREATMENT REGIMES USED BY VETERINARIANS IN ENGLAND AND WALES

Paul David Graham, BVSc, MRCVS

The Veterinary Investigation Centre
Kendal Road
Harlescott
Shrewsbury, Shropshire, SY1 4HD, U.K.

A large number of treatment regimes have been used by UK veterinarians to control digital dermatitis. In order to establish which of these treatments were the most useful, a postal survey was carried out. 118 questionnaire returns were received from 53 veterinary practices in South-West England and the Midlands. No returns were received from Welsh practices. The respondents were asked to give details of both individual treatment and whole herd treatment using footbaths. Of the footbath treatments, a combination of Lincomycin and Spectinomycin was the most effective treatment, with 89% of respondents considering there was a significant reduction in the incidence of disease. A wide range of concentrations were used, the majority being between 0.3 and 1 gram per litre. The efficacy of Lincomycin alone was slightly lower than that of the combined drug but showed similar results with 85% of respondents reporting a significant reduction of disease. Oxytetracycline used either alone or with citric acid was the third most effective treatment. This was commonly used in concentrations between 2 and 4 grams per litre. Marginally better results were achieved with concentrations in excess of 4 grams per litre. There was little correlation between the efficacy of any of the treatments and the frequency of application. The most commonly used being 2 or 3 consecutive treatments followed by fortnightly applications until the disease had resolved.

For individual treatment, response to systemic therapy with antibiotics was inconsistent. Tylosin (10mg/kg), Amoxycillin, Oxytetracycline (5mg/kg) and Sulphamethoxypyridazine were reported to be effective. Other antibiotics gave disappointing results.

Topical treatments were generally much more consistent. Administration of Oxytetracycline, Lincomycin, Gentian Violet and Gentian Violet with antibiotic having very similar success rates. Of 62

respondents, 90% of them considered it was essential to clean the lesion thoroughly before treatment.

THEORIES ON THE PATHOGENESIS OF BOVINE LAMINITIS

P.Ossent and C.J.Lischer*.
Departments of Veterinary Pathology and *Veterinary Surgery,
University of Zürich.Winterthurerstr 268,
CH-8057 Zürich, Switzerland.

Pododermatitis aseptica diffusa or laminitis, is an inflammation of the corium arising from a systemic disorder due to a wide spectrum of probably largely interdependent aetiological factors. These range from influences such as metabolic and digestive disorders, parturition or severe inflammatory processes (e.g. metritis or mastitis) to influences localized within the hoof such as trauma, claw or limb configuration (overload), lack or excess of movement, cold and "inherent" deficiencies in the keratinizing epidermis itself. Some factors have been known for thousands of years, others are new. However, all have something in common; in a first phase, predominantly vasoactive substances which have been released into the system, may trigger pathological mechanisms which ultimately cause degenerative change in the epidermal-dermal junction of the hoof.
As in every inflammatory process, the initial response centres around the blood vessels and is followed by exudation and swelling of the adjascent dermal tissues, further aggravating the situation. Vasodilation under the influence of histamine and the nervous system occurs at an early stage causing erythema which persists for the duration of the inflammatory process. Oedema in the corium develops after the vessel walls become permeable to fluid and the vascular pressure increases. Thrombi may supervene.
It is for anatomical reasons that the soft tissues in the hoof are in a unique predicament which renders them vulnerable to this cascade of adverse events. One special anatomical feature of the hoof is that the corium (dermis) is sand-

wiched within the inflexible narrow space between the pedal bone and the horn shoe. Any increase in the volume of the corium will aggravate the situation still further by increasing tissue pressure and inducing pain. A viscious circle analogous to the compartment syndrome in muscle develops.
By this stage all or some of the following have occurred; vessel wall damage, oedema, sludging, unphysiological arterio-venous shunting, hypoxaemia, tissue hypoxia, haemorrhage and thrombosis. In the bovine, it is not clear to what extent and at which stage inflammatory cell components contribute to these events or even whether their presence is obligatory.
Further special anatomical features of the hoof are the interdigitating dermal and epidermal laminae. A greater part of the body weight bearing on the limb is suspended from the dorsal and side walls by this structure whose integrity is imperative for the normal function of the foot. The multiplying basal epidermal cells, supplied from the underlying corium, require more nutrients and oxygen than any other tissues in the vicinity. Thus any deficiency in the supply of essential substances resulting from the changes in the corium mentioned above will affect the tissues of the laminar dermal-epidermal junction first, both for metabolic and then for mechanical reasons. The laminae will begin to separate by sliding past each other like fingers being drawn from a glove. The pedal bone, i.e. the digit, sinks within the horn shoe and compresses the thin layer of soft tissues of the sole and bulb. Another phase of pathological events is initiated.
Ensueing capillary damage and ischaemia lead to a second interreacting network of phenomena. Haemorrhage, necrosis, oedema, thrombosis and cellular inflammatory reaction develop under the pedal bone and present more reason for lameness and are a greater threat to the animal. Should the adverse influences continue they will become clinically apparent in the sole and bulb. Serous fluid, heamorrhages and necrotic debris either

hinder the production of horn altogether or they will be incorporated within the new horn layers being produced by the basal cells of the solar surface and emerge as double sole, red patches (hemorrhages) or cause perforations (ulcers). More diffuse lesions will lead to the production of inferior horn which appears some time later on the external solar surface as flakey masses or horn with a yellow soft cheese-like consistency. The site of necrosis is determined by the variable angles at which the pedal bone sinks and also depends on the bone's surface configuration. If the "keel" (i.e. the tuberositas flexoria), the most distal point of the sinking boat strikes the "sea bed" first, the compression and thus the lesion, be it heamorrage or necrosis, will be located adjascent to the "typical site". If it is sufficiently severe and persists long enough, it will eventually cause a sole ulcer. Correspondingly, toe lesions occur when the "bow" of the pedal bone strikes bottom. Necrosis in the solar corium is practically always focal and normally protuberances or the sharp abaxial edge of the pedal bone are palpable underneath.
Changes to the wall horn develop during the first phase of laminitic insult. The horizontal parallel corrugations seen in chronically laminitic animals are due to acute intermittent bouts of disease injuring the horn producing cells in the coronary region. Extensive exudation in the acute phase and, or reactive hyperplasia in the chronic stage of laminitis will lead to a widening of the laminar zone and eventually white line disease may develop.
The nature of the necropsy findings throw light on the pathogenesis of laminitis, give insight into the order of events and help to explain the nature of protracted sequels of laminitis which appear as lesions of the hoof's horn capsule.

BOVINE LAMINITIS (DIFFUSE ASEPTIC PODODERMATITIS) CLINICAL AND PATHOLOGICAL FINDINGS

Karen Mortensen
Troldnøddegården 21
2620 Albertslund, Denmark

INTRODUCTION

Laminitis in cattle is a diffuse, aesptic pododermatitis characterized by defective claw horn production and thrombosis and haemorrhages in the digital corium. The disease is painfull, especially bacause of the weight bearing and shock absorption of the digits. The pain induces a stress response (the sympathico-adrenal response), which results in peripheral vasoconstriction and further thromosis and haemorrhages in the corium. This viscious circle in the digital blood supply often leads to long-lasting cases of lameness, decreased fertility and production, and pre-mature culling. Thus, viewed from an ethical as well as an economic point, laminitis is an impotant digital disease in intensive production systems.

Avute laminitis may be caused by one single factor such as acute rumen acidosis, C. pyognes mastitis, or retained membranes. Following an acute attack, most cases become more or less chronic due to the viscious circle described above. On the other hand, chronis laminitis often occurs without any history of an acute attack ("subclinical laminitis"). The subclinical laminitis is characterized by a gradual change og the normal hoof to a hoof with concave profile, grooves and visible haemorrhages in the horn,

ulcerations, and increased white line.

Nilsson produced a monograph on laminitis in 1963 in which he described the clinical picture, the histopathology and discussed the pathogenesis and etiology of the condition. In the early 1980's Toussaint-Raven introduced the important hypothesis that many diseases, previously believed to be specific conditions that occurred randomly, were more frequently encountered in animals affected with laminitis. Peterse at the time working at Utrecht with Toussaint-Raven helped to substantiate the nutritional etiology of the disease which has become known as the 'sub-clinical laminitis syndrome'.

Once the concept of sub-clinical laminitis became accepted, not only was the earlier literature reviewed but new histopathologic studies were conducted. The advent of the sub-clinical laminitis hypothesis resulted in a significant number of publications dealing with the topic. Of particular note were those suggesting that a high incidence of lameness can be correlated with the incidence of production diseases or reproductive disorders. It is also suggested that the pathophysiologic conditions that cause laminitis affect other important body organs thereby causing heifers to fail to reach ther expected productivity. Perhaps the most valuable thought is that sub-clinical laminitis is a multifactoral disease involving managment and nutrition, thereby justfying an epidemiologic approach to the control of lameness.

Laminitis has been known to occur in horses for many years in which species the etiology has been extensively investigated (Obel 1948, Robinson et al 1975, Garner 1975, Garner et al 1975, Hood and Stephens 1981).

In the horse the disease varies in severity from a very painful life threatening condition (acute) to one that produces mild lameness (sub-acute). Chronic laminitis also occurs in the horse.

Current concepts suggest that a combination of factors influence the occurrence and severity of laminitis (Nilsson 1966; Weaver 1979; Peterse 1980,1982a, 1985,1986a; Andersson 1980; Mortensen and Hesselholt 1982,1986; Bazeley and Pinsent 1984; Livesey and Fleming 1984; Smart 1986; Brochart 1987; Bergsten 1988; Colam-Ainsworth *et al* 1989).

An inherited form of laminitis in Jersey cattle has been reported in South Africa (De Boom *et al* 1968), in the USA (Merrit and Riser 1968), and in the UK (Edwards 1972). A familial predisposition to laminitis has been observed by Nilsson (1963) and Maclean (1965). Bazeley and Pinsent (1984) mentioned a possible inherited predisposition to laminitis.

Friesian cattle are most susceptible (Nilsson 1963, 1966; Maclean 1966). Swedish Friesians are more often affected by sole hemorrhages and sole ulcer than Swedish Red and White cattle (Andersson and Lundström 1981). Brochart (1987) noticed a greater susceptibility to laminitis in Friesian cattle than in Holstein Friesian-cross cows. A tendency for a higher incidence of lameness in Dutch Friesians compared with Dutch Red and White cows has been detected (Grommers 1967).

There seems to be no doubt that the disease is related to a high energy intake, the frequency and quantity of consumption and the period of time over which the increase

intake occurs. The quantity and quality of fibre intake has also been suggested as major factor influencing the occurance of the disease.

The hypothesis of a multifactoral etiology extends beyond nutritional factors. Factors as genetic predisposition, claw size, body weight, architecture of limb angles, claw hardness, pigmentation of the claw and the quality of the surface over which the animal walks all influence the frequency of laminitis in a herd.

Exercise is essential for normal blood flow through the claw, therefore it has been suggested management practice that interferes with exercise may be a factor that influences the occurrence of lameness (Zeeb 1987).

Finally, in recent years behaviour has been suggested as a predisposing factor in lameness (Chesterton et al 1989, David 1986, Kempens et al 1987, Irps 1987). Furthermore, the effects of confrontation (between submissive heifers and dominant cows in the dry herd) could be considered to be a 'personality trait'that may be hereditable.

In recent years European veterinarians have come to realise that when lameness becomes a herd problem it is necessary to look further than at accidental causes for an explanation. More and more evidence is being accumulated that laminitis (founder) is the underlying cause of much of the lameness affecting dairy heifers and cows. Foot problems are being more frequently reported in first calving dairy heifers today than they were perhaps ten years ago.

Laminitis is not restricted to dairy cattle; accelerated weight gain in young beef bulls can bring about founder-like changes. It is highly probable that the feet of bulls can be ruined for life as a result of inappropriate management while they are still less than 14 months of age.

CLINICAL MANIFESTATIONS

Laminitis has been classified as being acute, sub-acute, sub-clinical and chronic. Such a classification may be misleading because there is no objective evidence to demonstrate that once incident of the disease can not manefest itself successively in more that one form of the condition. Histopathological evidence suggests that the disease is or, at least, can be, progressive. In other words it is conceivable that within one population of animals some animals could have sub-acute signs of the disease others could be classified as sub-clinical. If the disease had been present over a prolonged period it would be likely that chronic laminitis would be present. The distribution of the manefestations in a population would depend on:

a) The severity of the insults.
b) The frequency of the insults.
c) The individual (genetic) susceptibility of each animal in the population.
d) The length of time over which the population had been subjected to the insults.

Acute and sub-acute laminitis can be identified clinically as causing aberrations of gait that results from a disease process that is causing pain at the time that the observation is being made. Historical

evidence of the incident may or may not be observed at some time after the acute or sub-acute episode has resolved. The term 'sub-clinical' laminitis is usually applied to cases in which no aberration of gait is present but there is historical evidence in the hoof capsule that an insult has occured such as hemorrhages in the sole or grooves in the wall. There is also a concensus of opinion that certain hoof diseases are more frequently in herds in which sub-clinical laminitis is believed to occur than in herds in which it is believed to be absent. It is an unsubstantiated assumption that chronic laminitis results from one of the previously described forms of the disease. However, the incidence of chronic laminitis in cattle is very low (approximately 2%).

Claw lesions associated with laminitis

A. Primary Lesion

A primary Lesion is defined as one that occurs as a direct result of the histopathologic changes in the corium that have been brough about by the insult.

A1. ***Hemorrhages in the Sole***

Hemorrhages of the sole are the major and characteristic indication that laminitic insult has occurred at some time in the past (often months elapse before this phenomenon is observed). This sign is a historic record and not an indication of the status of the animal at the time of the examination. The rate of horn growth varies according to the season and to the rate of wear. As a rule of thumb, hemorrhages take from two to three months to appear at the surface of the sole.

The hemorrhages can take the form of a

slight pink tinge, a pronounced brush stroke of red coloration or a dark solid red stain. It is unlikely that thumb pressure will cause pain and usually it will be possible to pare away the affected layer of horn to reveal normal horn beneath. By comparison, bruising of the sole occurs when the sole has been worn very thin and trauma has caused the rupture of a blood vessel. In these cases the horn of the sole depresses on pressure and causes the animal pain. The color of the blood beneath the horn is usually dark blue or black.

Hemorrhages may be worse in heavy animals, animals with straight hind limbs, animals walking on concrete or in claws excessively softened by continual immersion in slurry.

A2. *Softening of the horn of the sole*
Although objective evidence is not available there is a consensus of opinion that sole horn produced after an episode of laminitis is softer than normal. This may or may not be associated with a yellow coloration of the horn.

It is believe that soft horn is more vulnerable to damage than normal.

B. Secondary Lesions

Secondary lesions are disease processes that, reportedly, occur more commonly in cattle that have been subjected to a lamanitic insult than those which have not.

B1. *Pododermatitis circumscripta* **(Sole Ulcers)**

The lesion most commonly associated with sub-clinical laminitis is Podermatitis circumscripta usually referred to as 'sole

ulcer'. Peterse and Antonisse (1981) and Peterse (1986b) estimated the heritability for sole ulcer in Friesian heifers at 0.14. Nielsen and Smedegaard (1984) gave a similar estimate. Petersen *et al* (1982) reported a much lower figure of 0.09. Heritability for sole lesions has been estimated at 0.1 and 0.2 by Petersen *et al* (1982) and Smit and Verbeek (1984), respectively.

Many workers believe that if this condition occurs in over 10% of the cows in a herd during any given year that the underlying cause is laminitis.

Although laminitis is considered to be the most important and common cause of sole ulcers, there are other causes. Excessive trimming of the sole or permitting animals to walk long distances over abrasive surfaces causes the sole to become flexible.

B3. *Interdigital Dermatitis* **(Stable Footrot)**

It is a strongly held belief in Holland that stable foot rot is more commonly encountered in herds with a high incidence of laminitis.

B4. ***White line (zone) disease***

This condition is also considered to be associated with laminitis. The time relationship to the laminitic insult is not known. The significance of hemorrhages in the white line of the toe is also unknown. In some cases the hemorrhage can be so severe in the toe region that the white line appears to disintegrate and a so called, 'toe ulcer' develops. This phenomenon may be associated with rotation of the distal phalanx. If these ulcers are neglected very severe infections

can result some of which require amputation of the digit.

B5. ***Heel Erosion*** As has been pointed out earlier in this paper, heel erosion can be an advanced complication of stable foot rot. It is now believed by some workers that laminitis can also be a predisposing cause of heel erosion. If this is the case the end result is exactly similar in appearance.

Laminitis causes layers of hemorrhages to develop in the horn of the sole. These layers end at the heel as tiny grooves. It is thought that bacteria, that are commonly found in manure, invade these grooves and flourish on the traces of blood that may be there. Although this is only a theory, researchers in the field believe that heel erosion occurs most frequently in animals affected with laminitis.

Whatever the cause, the erosions start as pits or craters and/or fine dark grooves in the heel horn. They deepen until most of the horn has been destroyed. Typically, the erosion may be seen as a deep black diagonal groove at the junction between the heel and the sole. The loss of heel horn that results from erosion severely hampers the shock absorption function of the heel and places strain on the remaining portions of the sole. The disease is common and difficult if not impossible to control. Intensive management systems, poor hygiene and heavy feeding are all likely to be implicated.

B6. ***Double Sole*** A double sole is produced after there has been a sudden and complete stop in the production of claw horn followed by a resumption of normal growth. The condition usually causes no alteration in gait and

is often only noticed during hoof trimming.

Histology of the corium of the Bovine Claw

The corium of the claw is a layer of fine blood vessels and elastic tissue sandwiched between the horny hoof and distal phalynx (toe bone). The blood supply to the corium feeds a network of very delicate vessels that nourish the stratum germinativum which is the layer of cells that produces horn. Every time the animal bears weight on its claws, these tiny blood vessels are compressed. Why then do they not burst under the pressure?

Three mechanisms to relieve pressure inside the claw.

1) Glomus bodies. Glomus bodies are expandable vessels forming bridges between arterioles and venules into which blood can be diverted when the pressure inside the claw increases. As the pressure from weight bearing is relieved, tiny muscles in the walls of the glomus bodies squeeze the blood back into the circulation.
The muscles in the glomus bodies can be paralysed by poisons in the blood.

2) Arterio-venous shunts are simple bridges between arteries and veins. Muscles are present in the walls of these shunts. When the animal bears weight on the claw, the muscles relax opening the shunt, allowing the blood to be diverted directly from larger arteries into veins. This mechanism prevents the build-up of pressure inside the claw. The muscles in the arterio-venous shunts can also be paralysed by poisons in the blood.

3) The coronary cushion lies directly

beneath the coronary band and is composed of a spongy network of veins. As the animal moves, the distal phalanx presses against the wall of the claw thereby squeezing blood back into the circulation. Exercise, therefore, is extremely important for keeping blood circulating through the vessels of the claw. These three mechanisms play an important role in laminitis.

Diagnosis

When a hoof wall is examined carefully, fine ridges (rugae) can be seen running around the claw more or less parallel to the coronary band. In fact the ridges diverge slightly from toe to heel. This indicates that the wall immediately in front of the heel grows and wears faster than it does at the toe. The high rate of horn wear at the heel (and compensating high growth rate) is due to friction. The heel region is the part of the claw that contacts the ground first during locomotion and tends to slide to a halt before the entire sole contacts the ground. From the perspective of disease, the heel is the most important region of the claw. Sole ulcers, white line disease and heel erosions are diseases that commonly affect this area and are found to occur most frequently in herds affected with laminitis.

The Hardship Groove The term 'hardship groove' is frequently used by cattlemen to describe a shallow groove that may be found running around the hoof wall parallel to the coronary band. This hardship groove, as the name implies, records an event that seriously stressed the animal. In adult cattle, acute

mastitis, a septic uterus or any serious disease can cause a temporary reduction in horn production and the tell tale hardship groove being formed. The groove is formed at the coronary band and does not become obvious until the claw has grown out a little. The hoof wall grows down from the coronary band at the rate of a approximately 2 1/2 inches each year (0.6 cm/month). By measuring the distance from the coronary band to the hardship groove in centimetres and dividing by 0.5 (0.5 in summer and 0.6 in winter) it is possible to calculate the approximate time at which an insult occurred.

A hardship groove is produced when beef calves are weaned. As the groove grows out it is usually noticeable that there is a difference in the appearance of the horn that was produced before weaning from that which was produced after weaning. That is to say the horn furthest away from the groove was produced prior to weaning and that closest to the coronet was produced most recently.

The quality and characteristics of the pre-weaning horn tells something about the management of the calf before it was weaned. If the horn in this area is rough or ridged it is often useful to enquire about the pre-weaning management. The mother may have been a poor milker for example, she might have had only three functional quarters or she could have been a heifer. If the horn between the groove and the coronary band is of poorer quality compared with the pre-weaning horn then this indicates that the animal did not adapt very well to the post weaning management or feeding program.

Congestion of the Blood Vessels of the Corium

Sometimes at livestock shows one may observe that the horn at the toe appears to have a pink tinge. This is only noticeable in animals with claw horn that is very light in colour. This pinkness or congestion can be seen in some animals that have been heavily fed for about two weeks. If the heavy feeding were to continue the animal will either develop laminitis or it may adjust to the ration.

Pinkness of the skin above the coronary band of the heel and of the dew claws can be an indication that a dairy cow is having its concentrate ration increased too rapidly. This reaction is extremely variable between individual animals. However, when this phenomenon is observed in several animals in the same herd it is useful to investigate the nutritional background of the group. The pinkness fades as the animal adjusts to the ration but if this is a constant feature in a herd the indicators are that the animals are being pushed too hard.

Rotation of the Distal Phalanx

During the routine examination of the claws of feedlot steers, it has been found that in some cases the tip of the distal phalanx (toe bone) is pressing down on the corium. A shallow depression is formed on the inside surface of the sole. The distal phalanx has rotated downwards in a similar manner to that observed in the horse. When this occurs in horses permanent irreversible damage has taken place. In all of the cases of rotation that we have observed, there has been a ridge on the outside of the claw wall running parallel to coronary band. Although the cause of rotation is not known, we have only observed it in animals that were

introduced suddenly to high energy feed after a prolonged period on a diet restricted mainly to forage. Any major change in the feeds going into the rumen will result in altered fermentation patterns and if the change is big enough, ie sudden addition of increased amounts of starch or protein, it takes up to 14 days for the rumen microflora to establish and equilibrium. Ridges running around the claws of young cattle may indicate that serious damage may have occurred inside of the claw.

PATHOGENESIS AND ETIOLOGY

Most research workers agree that nutritional factors are the most important cause of laminitis. This observation is by no means cut and dried. Animals fed exactly similar rations under apparently similar circumstances are not always affected in a similar manner. It has been suggested that a number of other factors are also involved in causing laminitis. It has a ***multifactorial etiology***.

It has been established that the consumption of high levels of readily digestible carbohydrates leads to the proliferation of Streptococcus bovis and Lactobaccillus species in the rumen. Rumenal acidosis occurs, with the result that gram negative organisms are reduced in number often resulting in the release of large amounts of endotoxin. Rumenal acidosis has been associated with both rumenitis and liver abscess.

The traditional explanation of the etiology of the disease involves an excess carbohydrate intake, followed by rumenal acidosis. As result of an environment that is

hostile to the normal rumen flora, toxic substances, that act on small blood vessels are produced. When absorbed into the blood stream these toxins have a devastating effect on the network of tiny vessels that nourish the horn producing tissues of the claw. At first blood stagnates causing increased pressure and pain in the claw. This process finally causes irreverible damage to some of the structures that produce horn.

Research has also shown that the consumption of high levels of carbohydrates is linked to the appearance of 'founder-like' changes in the claws. The release of toxins (poisons) that affect blood vessels (vasoactive) are believed to be involved in producing these changes. Correct ration management forms the basis for some of the measures for controlling laminitis and these will be discussed later in this paper.

The mechanisms by which vasoactive agents are produced is a topic about which there has been a great deal of speculation. Furthermore, because the true nature of these mechanisms has not been established there is no value in reviewing these theoretical mechanisms in this paper.

When vasoactive toxins reach the tiny vessels of the claw it is thought that the glomus bodies and the arterio-venous shunts are paralysed. The pressure inside the claw rises, the vessels are damaged and blood or the blood fluids soak into the horn claw staining it either pink or yellow. The flow of blood through the corium of the claw slows down, impeding the nourishment of the horn producing cells. The poor quality horn that results is softer and more easily damaged than is normal. Therefore, the tell tale signs of

laminitis are, blood staining and/or yellow discoloration of the sole and sometimes the white line. Usually the texture of the sole horn is much softer than normal and it wears more rapidly than is normal.

An increase in blood pressure inside the claw, and the associated reduced blood flow, frequently leads to blood clot formation which is another characteristic feature of laminitis. The clots form a fine layer on the inside of the walls of the tiniest vessels. Because the vessels are narrower than normal, the blood flow through the claw slows down and there are fewer nutrients reaching the horn producing tissues. The blood vessels can eventually become completely blocked and scar tissue starts to form in the claw.

Frequently young animals appear to recover from a laminitic incident. This may be because new blood vessels develop to form collateral circulation and take over the function of those that have been damaged. Nevertheless, each time an animal has a bout of laminitis, more scar tissue is formed and the animal be less able to recover from the next 'insult'.

Work by Danish researchers has demonstrated that the toxins which affect the claws also affects other important organs in the body such as the liver and kidneys. These workers have also shown that animals experiencing episodes of slight laminitis do not lactate as well as normal animals. This possibility should be born in mind if milk yields, particularly those of young animals, are 'disappointing'.

Earlier it was pointed out that the pumping action of the coronary cushion assists

the blood to flow freely through the claw. Lack of exercise will reduce the blood flow. Therefore, it can be argued that reduced exercise for animals that consume large quantities of carbohydrate may increase the risk of laminitis. Pregnant animals, particularly heifers that are allowed free range before suddenly being confined and introduced to a rich diet at calving may be particularly at risk.

CORRELATION OF LAMINITIS AND FERTILITY

D.W.Collick, BVSc,DBR,MRCVS.,
Delaware Veterinary Group,
Castle Cary,Somerset,England,BA7 7BN.

There are many reports in the literature of the harmful effect of peri- and post-partum disease on fertility. Milk fever and ketosis (3), retained placenta and metritis (27,18).Other workers have associated peri- and post-partum disease with delayed uterine involution and reduced fertility.

Dietary mismanagement has been linked to liver changes and associated with an increased incidence of peri-partum disease. Sommer(30) reviewing German work postulated that dietary errors (to little fibre, energy or protein excess) and excessive 'steaming up', produces liver damage which after 3 or 4 lactations causes the compensatory mechanisms to fail. Reid and Roberts (25) reported an increased fat content of the liver cells which they associated with pre-partum 'over conditioning' of cows. Cows with more liver fat had reduced appetites after calving, showed an increased incidence to reproductive disorders, infectious diseases (mastitis and metritis) and metabolic disorders (milk fever and ketosis).In this study, cows with more liver fat had on average a 33 day longer calving interval, associated with a delay in onset of ovarian activity and oestrus, and a reduced pregnancy rate.

Pattern and incidence of lameness in the UK has altered in the last decade. During this period both herd size and production per cow has increased (32). The predominant breed, the friesian, has reduced from 87 to 75% of the national herd and the Holstein or Holstein Friesian cross increased from 6 to 20%, whilst overall numbers have declined. Surveys in the late 70's and early 80's estimated annual lameness incidence to be about 5% (11,26), if practitioner based, to around 25% (33,24) if

farmer treatments were included. A more recent study of 37 farms in four geographical locations, covering a two and a half year period (16), reported an annual case incidence of 60% (range 9 - 200%) It appears that lameness is an increasingly more important condition in the dairy industry.

Incidence pattern of the various lesions recorded has also changed. In the original Compton survey (26), foul of the foot (interdigital phlegmon) was the most commonly identified lesion (15%). However white line disease (14%) and sole ulceration (pododermatitis circumscripta - 12%) were nearly equally as important. In contrast, in the Liverpool study (16), sole ulceration ranked the most common lesion and in combination with white line lesions accounted for 50% of all lesions recorded or 63% of claw lesions. Foul of the foot represented 5%, but a 'new' condition, Digital dermatitis was reported (8% incidence). Foul in the foot was likely to have been under recorded in the Compton survey since the condition is commonly recognized and treated by farmers. Additionally the Liverpool study comments that a diagnosis of laminitis was made in less than 1% of total recorded cases; generalised laminitis was diagnosed as severe in only 8 cattle, all of which exhibited great pain in all four feet and could hardly walk. It is the authors experience also that acute laminitis is rare.

Both sole ulceration and white line disease are associated with and considered to be a sequel of laminitis (20,17,22). Acute laminitis is recognized and understood by practitioners to be a manifestation of a nutritional or metabolic upset; histamine, lactic acid, toxin and endotoxin release having been implicated, leading to a reaction in the corium vasculature resulting in a disturbance of blood flow and permanent damage to the hoof. The clinician recognizes haemorrhages and yellow discolouration of sole horn and correlates this with sole growth to indicate 'insults' which

have occurred 6-8 weeks previously. These 'insults' have been termed subclinical laminitis and most likely represent a combination of management, housing and nutritional changes (12). Recent observations (6,29,23) indicate changes before or around calving and poor housing (cubicles) are important predisposing factors.

Lameness is recognized as a condition that disproportionately affects animals in early lactation (11,26,33,24). Dewes (9) studied two New Zealand herds at pasture and reported that lameness was followed by delayed oestrus, poor breeding performance, short lactation, low milk fat yield and sudden body weight drop. Cobro-Abreu and others (5) in a study of disease, production and culling in Canada were unable to demonstrate significant relationships between lameness and increased calving interval or culling. This study included only 21 cases of lameness. Hassall and others (13) observed cows at pasture. Lame cows lay down longer and grazed for shorter periods than the normal cows. In addition, lame cows had significantly lower bite rates than normal cows. They conclude that these behavioural differences between lame and normal cows indicated that lameness had serious effects on welfare and productivity.

Lucey and others (15) compared fertility in 770 cows. The median interval from calving to first service increased by 7 days and the median interval from calving to conception increased by 11 days for cows with sole or white line lesions. In particular if the lesions were observed in the period between 36 and 70 days after calving these intervals increased a significant 17 and 30 days respectively. Where lameness occurred within a period of 63 days after service, pregnancy was lower at 31% than at other times (40%).

Collick and others (7) compared 427 cows with foot lameness with the cow nearest in parity and calving in 17 dairy herds. Calving to first

service interval was 4 days and calving to conception interval was 14 days longer in the lame cows. More lame cows were culled. Categorising cases by type of lesion, cows with sole ulceration (pododermatitis circumscripta) had an interval from calving to conception 40 days longer than controls when the lesion was observed in the period to 71 to 120 days after calving. When divided according to severity and duration of lameness, cows with a high 'clinical effect score' had an interval from calving to conception 31 days longer than controls over the same period.

From these findings it is clear that lameness in dairy cows can have a marked effect on fertility. The magnitude of the effect depends on the type of lesion, severity and duration of the lameness and the time after calving when the cow is lame.

Stress

Pain caused by serious foot lameness is presumably stressful, although evidence of corticosteroid release in lame cows does not appear to have been published. Transport of cows in a lorry, which appears to be a mild stressor, blocks the release of luteinising hormone (19) and causes the release of corticosteroids. While the concentration of cortisol was elevated in the cows blood, LH was not released, during and after transport. Cortisol has been shown to interfere with release of LH in heifers (31).The observation of most deleterious effect on fertility with lesions identified in the period 71 to 120 days after calving is suggestive of an interference in LH release on both ovulation and luteal tissue support.

Housing/ Stress

Colam-Ainsworth and others (6) report the effect of poor cubicles and insufficient bedding, increasing incidence of lameness in heifers, particularly sole haemorrhage and ulceration. Eddy (10) reviewing management of 'repeat breeders' observes an effect of a change from grazing to permanent housing, in a reduction in

pregnancy rate, during the breeding season. This observation is recognized by the author. Eddy believes this to be stress related, pregnancy rates dropping from 46 to 29%. Treatment with GnRH (10mcg of Buserelin) on day 12 after service apparently restores pregnancy rates close to prehousing levels.

GnRH administered 11-13 days after service has been demonstrated to improve pregnancy rates(28). At this stage of the oestrous cycle it is thought to cause partial luteinisation or atresia of the developing follicular wave, probably by release of LH from the pituitary, resulting in a delay in luteolysis. It is thought that this short delay in return to oestrus gives the embryo more time to produce bTP-1 before prostaglandin is produced. In essence, the GnRH is buying time for the embryo to signal its presence to the dam (31).

Ova / Luteal Quality

Britt (4) reviewed body weight loss and reproductive function. Cows loosing 0.5 or more body condition during the first 5 weeks of lactation showed reduced fertility. Estimating that 60 days or more is required for the growth of an inactive primordial follicle to the ovulatory stage, the first or second ovulatory follicles after calving develop when energy balance is positive in the dry period. In contrast the third, fourth (and fifth) ovulatory follicles develop when energy balance is most negative after calving. He postulates that adverse metabolic conditions during its earliest development may somehow become imprinted on the developing primordial follicle so that it reflects that adversity in lower fertility. Using retrospective progesterone measurements, after first and second heats on animals with below and above average condition loss, levels were similar. However, progesterone levels after subsequent heats were greater for those animals with below average condition loss which were more fertile.

With the work of Collick and others (7), cows diagnosed with sole ulceration showed an average 22 days increased interval to conception. Since the lesion is of longer standing it suggests that the effect on fertility occurred over a longer period and probably related to the time of initial 'insult'.

The Clinician / Lameness / Fertility

When examining the lame cow, the clinician diagnoses disease which increasingly appear to be related to subclinical laminitis. It is the author's opinion that the major predisposing factors are changes in management and nutrition around calving, in particular housing and cow comfort. Poor cow comfort, increased standing time, leads to a localised coriitis. Nutrition problems may exacerbate this effect.

Many factors are involved in the management of herd fertility. The clinician can define action areas. Animals not served by a defined interval are treated and served. Until that animal is diagnosed as in calf the fate of that service is in the hands of the stockman's ability to observe heat returns. Milk progesterone can improve return detection rate and ultrasound scanning may now help fill the gap to manual pregnancy diagnosis in revealing empty animals. This will be of immense economic benefit to the farmer and should also aid the veterinarian's understanding as to where and how lameness is affecting fertility.

References

1. Arkins,S. (1981) Lameness in dairy cows. Irish Veterinary Journal; 35, 135-140.

2. Baggott,D.G., Russell,A.M. (1981) Lameness in cattle. British Veterinary Journal; 137, 113-132.

3. Bostedt,H., Huber,C. (1982) Fertility of cows after acute metabolic disturbances during the peripartal period.Proceedings of the 12th world on diseases of cattle,the Netherlands. Volume 1, pp 605-608.

4. Britt,J.H. (1992) Influence of nutrition and weight loss on reproduction and early embryonic death in cattle. Proceedings of 17th world buiatrics congress, St.Paul, Minnesota, USA. Volume 2, 143-151.

5. Cobo-Abreu,R., Martin,S.W., Willoughby, R.A., Stone,J.B.(1979) The association between disease, production and culling in a University dairy herd. Canadian Veterinary Journal; 20, 191-195.

6. Colam-Ainsworth,P. Lunn,G.A., Thomas,R.C., Eddy,R.G. (1989) Behaviour of cows in cubicles and its possible relationship with laminitis in replacement heifers. Vet. Record; 125, 573-575.

7. Collick,D.W., Ward,W.R., Dobson,H.(1989) Associations between types of lameness and fertility. Vet. Record; 125, 103-106.

8. De Bois,C.H.W. (1982) Some aspects of the therapy and prophylaxis of retained placenta and puerperal endometritis in the cow. Kerg and Schallenberger, M. Nijhoft. pp 479-509.

9. Dewes,H.F. (1978) Some aspects of lameness in dairy herds. New Zealand Vet. Journal; 26, 147-148 and 157-159.

10. Eddy,R.G. (1994) Presenting problem decision flow chart - the Repeat Breeder. Cattle Practice; Volume 2, 39-46.

11. Eddy,R.G., Scott,C.P. (1980) Some observations on the incidence of lameness in dairy cattle in Somerset. Vet. Record; 106, 140-144.

12. Greenough,P.R., Vermunt,J.J.(1991) Evaluation of subclinical laminitis in a dairy herd and

observations on ssociated nutritional and management factors. Vet. Record; 128, 11-17.

13.Hassall,S.A., Ward, W.R., Murray,R.D. (1993) Effects of lameness on the behaviour of cows during the summer. Vet. Record; 132, 578-580.

14.Liversey,C.T., Fleming,F.L. (1984) Nutritional influences on laminitis, sole ulcer and bruised sole in friesian cows. Vet. Record; 114, 510-512.

15.Lucey,S., Rowlands,G.J., Russell,A.M. (1986) The association between lameness and fertility in dairy cows. Vet. Record; 118, 628-631.

16.Liverpool University (1993) Report CSA 1379. An epidemiological study to determine the risk factors of lameness in dairy cows.

17.Maclean,C.W. (1971) The long term effects of laminitis in dairy cows. Vet. Record; 89, 34-37.

18.Morrow,D.A., Roberts,S.J., McEntee,K., Gray,H.G. (1966) Post-partum ovarian activity and uterine involution in dairy cattle. Journal of the American Veterinary Association; 149, 1596-1609.

19.Nanda,A.S., Ward,W.R., Dobson, H. (1989) Effects of naloxone on oestradiol induced LH surge in transported cows. Journal of Reproduction and Fertility; 87, 803-807.

20.Nilsson,S.A. (1963) Clinical, morphological and experimental studies of laminitis in cattle. Acta Veterinaria Scandinavica; 41, 9-304.

21.Oltenacu,P.A., Britt,J.H., Braun,R.K., Mellaberger,R.W. (1983) Relationship among parturition type, discharge type, cervix involution and reproductive performance -

Holsteins. Journal of Dairy Science; 66, 612-619.

22.Peterse,D.J. (1980) Assessment of the bovine hoof on the appearance of sole lesions. PhD Thesis.
Rijksuniversiteit, Utrecht, Netherlands.

23.Potter,M.J., Broom,D.M.(1990) The behaviour and welfare aspects of cattle lameness in relation to building design. Proceedings of 6th Symposium on diseases of the Ruminant digit, Liverpool. pp80-84.

24.Prentice,D.E., Neal,P.A. (1972) Some observations on the incidence of lameness in dairy cattle in West Cheshire. Vet. Record; 91, 1-7.

25.Reid,I.M., Roberts,C.J. (1982) Fatty liver in dairy cows. In Practice; 4, 164-169.

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

27.Sandals,W.C.D., Curtis,R.A., Cote,J.F., Martin,S.W. (1979) The effect of retained placenta and metritis.complex on reproductive performance in dairy cattle - a case control study. Canadian Vet. Journal; 20, 131-135.

28.Sheldon,I.M., Dobson,H.(1993) Effects of gonadotrophin releasing hormone administered 11 days after insemination on the pregnancy rates of cattle to first and later services. Vet. Record; 133, 160-163.

29.Singh,S.S., Ward,W.R., Lautenbach,K., Hughes,J.W., Murray,R.D. (1993) Behaviour of first lactation and adult dairy cows while housed and at grass and its relationship to sole lesions. Vet. Record; 133, 469-474.

30.Sommer,H. (1975) Preventive medicine in dairy cows. Veterinary Medical Review; 1/2, 42-63.

31.Stoebel,D.P. Moberg,G.P. (1982) Effect of adrenocorticotrophin and cortisol luteinising hormone surge and oestrus behaviour in cows. Journal of Dairy Science; 65, 1016-1024.

32.UK Dairy Facts and Figures (1992) Federation of Milk Marketing Boards, Thames Ditton, Surrey.

33.Whitaker,D.A.,Kelley,J.M., Smith,E.J. (1983) Incidence of lameness in dairy cows. Vet. Record; 113, 60-62.

PREDISPOSING CAUSES OF LAMINITIS

Jos J. Vermunt
Department of Veterinary Clinical Sciences, Massey University, Palmerston North, New Zealand.

Introduction

Lameness in cattle is of great importance to the dairy and beef industry because of economic and animal welfare considerations. Claw disorders account for 70-90% of diagnosed cases of lameness in cattle.[1][2][3] Laminitis is regarded by various authors from the northern hemisphere as an important predisposing factor in lameness caused by claw disorders.[4][5][6][7][8] It is many authors' opinion that the subclinical laminitis syndrome, including white zone (white line) lesions and sole ulcer is the most important condition affecting the claws of dairy cattle today.

By definition, laminitis or diffuse aseptic coriitis (pododermatitis) is an inflammation of the laminae within the claw. However, although the corium of the laminar region may be most susceptible to insult, other regions, particularly those related to the sole (papillar region), are frequently involved, especially in cattle.

Different forms of laminitis

Bovine laminitis is a debilitating disease that results in functional and/or morphological changes within the claw. It can be broadly classified into four forms depending on the severity and duration of the condition. The terms 'acute','subacute', 'chronic', and 'subclinical' laminitis are used.
Acute and chronic laminitis have been well described by several workers.[4][9][10][11][12][13]

In the acute and subacute stage of the disease, an aseptic inflammation of the corium coincides with a systemically sick animal. At this stage, the claw horn shows few, if any, visible changes. These forms of laminitis are

prone to recurrence at varying intervals and often progress to the chronic form.[4][13][14][15]

Chronic laminitis has no systemic symptoms; changes are localized to the claw. A disturbed horn growth pattern and an alteration in the shape of the claw, i.e. elongated with a flattened and broadened sole, are characteristic.[4][11][12] Grooves and ridges, caused by irregular episodes of horn growth, can be seen in the claw wall. This results in a distinct rippled appearance, the ripples being close at the toe and diverging at the heel.

The phenomenon of subclinical laminitis in dairy cattle was described for the first time in 1979.[16] Changes in posture or locomotion are usually not observed. However, in subclinical laminitis, also leading to chronic laminitis, significant changes in the claw horn are visible. The horn becomes physically softer and discoloured, rather waxy in appearance. It is often stained yellow and haemorrhages can be seen in the weight-bearing surface of the claw, in particular the white zone, apex of the sole and axial side of the sole-bulb junction.[6][8][16][17]

Aetiology of laminitis

Despite intensive study, both by experiment and by clinical observation, knowledge of the precise aetiology and pathogenesis of laminitis is still incomplete. Studies in sheep have contributed to an understanding of the disease. The equine model, based on starch overloading[18], has acted as a source of information for the study of bovine laminitis. Many of the findings in these species have been ascribed to cattle due to the lack of a reliable cow model.

The various theories on the aetiology of bovine laminitis have been reviewed recently.[19] In analogy to the horse, the most commonly accepted hypothesis is that of insults to the peripheral vascular system of the corium, resulting in a reduced digital capillary perfusion, particularly through the lamellae and papillae.

Vasoconstriction and ischaemia of the peripheral microcirculation in the corium of the bovine digit have been incriminated in the pathophysiology of laminitis.[19][20][21][22][23] In contrast, acute laminitis is recognized clinically by the presence of a bounding digital pulse and engorgement of the digital veins[9][12], manifestations of increased total blood flow to the digit. To rationalize these two apparently contradictory phenomena, i.e. the simultaneous presence in the digit of ischaemia despite increased blood flow, it has been proposed that the ischaemia results because blood is shunted away from the capillary circulation in the corium through dilated arteriovenous anastomoses (AVAs) under the influence of vaso-active substances.[24] Arteriovenous anastomoses dilate in response to, amongst others, histamine[25], lowered blood pH[26], and trauma[25], or compressive stress.[27]

Objective

The existence of AVAs within the hoof of large animals had been described only in the horse[28] until Vermunt and leach[23] demonstrated the presence of AVAs in the circulation of the corium of the bovine claw. The precise cause of laminitis is not known. Current concepts suggest that a combination of factors influence the occurrence and severity of laminitis; the aetiology is multifactorial. Many factors acting either directly or indirectly (through vaso-active substances) on AVAs, thereby resulting in the inappropriate activity of such AVAs, have been cited as predisposing causes of bovine laminitis.

The main objective of this paper is to review the major predisposing cause of laminitis. No attempt has been made to include every reported or suggested potential causative factor - 'the once in a lifetime type' -, since this would make the text simply confusing, and make the selection of probable causes difficult.

Predisposing causes of laminitis

Systemic diseases

A systemic disease process would initiate digital vascular changes which result in ischaemia of the laminae and papillae. The commonly accepted hypothesis is that the toxic substances histamine and/or endotoxin are produced during post-partum diseases such as mastitis, acetonaemia and (endo)metritis.[9][10][29] In this context, Toussaint Raven[29] also listed udder oedema and retained foetal membranes as possible causes of laminitis. High doses of systemic histamine on its own[9], or in combination with grain overfeeding[30], result in acute laminitis.

Endotoxin has received special attention because acute laminitis has been reported to occur in diseases in which endotoxaemia may be present. In contrast to recent studies[22], Mortensen *et al.*[21] were able to induce mild signs of laminitis in cattle by administering endotoxin. In their studies, however, they used extremely high doses of endotoxin which was injected directly into the digital arteries. Although endotoxin has been incriminated as a mediator or triggering mechanism in the onset of equine laminitis[31][32], the etiologic significance of endotoxaemia to the laminitis syndrome in cattle has not yet been fully established.

Nutrition

The increase in the occurrence of laminitis has primarily been ascribed to the change in feeding regimens. Nutrition, especially feeding rations high in carbohydrates, is the most commonly implicated factor in the initiation of the laminitis syndrome. A disturbed digestion in the rumen, or a toxic agent in the fodder are considered to be causative factors.

Barley grain -- Barley has been mentioned by several authors. Maclean[4] suggested that histidine in barley could be metabolized into histamine or histamine could be released

following an allergic reaction to barley after sensitization of the animal.[4][9] Weaver[33] reported a high incidence of laminitis after barley was incorporated into the ration of dairy cows. Little and Kay[34] found a high incidence of laminitis in heifers reared on a barley-beef diet.

Protein -- Other workers put more emphasis on a high level of protein in the diet. In Europe, lush growing grasses, particularly young rye grass pasture with its high protein and metabolizable energy content, have been considered in the pathogenesis of laminitis.[9][10] Manson and Leaver[35] found an increased prevalence of laminitis associated lesions in dairy cows that were fed high protein rations. Bargai *et al.*[36] reported outbreaks of laminitis in dairy calves fed rations containing 18 per cent digestible protein. In contrast, Greenough *et al.*[7] found that an increasing level of protein had no influence on the prevalence of sole haemorrhages associated with laminitis. Vermunt[19] suggested a possible link between cows grazing pastures high in protein (22% or more crude protein in the dry matter) and laminitis.

There is little research information available to indicate what levels of protein are high enough to cause laminitis and what mode of action protein (or ammonia) plays in the disease development process. Nilsson[9] suggested an allergic-histaminotic reaction to the protein. Urmas[37] and Chew[38] implicated toxins from protein origin in the aetiology of laminitis. Bazeley and Pinsent[39] suggested a link between nitrogenous breakdown products/heavy protein supplementation and laminitis.

Carbohydrate -- Most investigators agree that excessive carbohydrate intake is the principal, nutrition-related factor in the development of laminitis. Feeding rations high in carbohydrates, especially to unconditioned animals, can lead to a lactic acid accumulation in the rumen and a decrease of the rumen pH. The

lowered pH is accompanied by a change in the composition of the ruminal micro-flora from predominantly gram-negative organisms to predominantly gram-positive, lactic acid-producing bacteria.[40][41] Endotoxin is released on bacteriolysis from the outer cell wall of dying and disintegrating gram-negative microbes.[42][43]

Products of the deranged rumen fermentation, such as lactic acid, endotoxin and possible histamine, become toxic if produced and absorbed in sufficiently large amounts. Lactate and/or endotoxin may then enter the bloodstream and induce local disturbances or allergic reactions in the micro-circulation of the corium, either directly or through released vaso-active mediators.[20][21] The increase in intra-ruminal acidity induces a dysfunction of the ruminal mucosa, which further facilitates the absorption of these products. Histamine is very unstable within the rumen and also poorly absorbed through the rumen wall.[44]

Laminitis may occur when unadapted cows are placed in high production groups without precautions or when the feeding system allows the unrestricted consumption of concentrate. Many authors also implicate the practice of 'lead-feeding' ('steaming-up') and the rapid increase in the amount of concentrate fed in the peripartum period and the period up to peak milk yield.[16][17][45][46]

Substantial and sudden diet changes from a high-fibre diet (basal maintenance ration) during the dry period to a high-concentrate ration immediately after calving are believed to increase the risk of laminitis.[9][10][15][47] In a trial comparing a rapid and gradual daily increase of concentrate, no difference in the incidence of laminitis was observed between animals in the control and experimental groups.[48] Peterse and Van Vuuren[49] indicated that the critical factor is not the length of the period during which the cow is allowed to adapt to a new diet, but rather the high quantity of concentrate in the ration. Manson and

Leaver[50][51] came to a similar conclusion. In their studies, they found a higher incidence of laminitis in cows fed high levels of concentrate than in cows given low levels of concentrate. Other workers[52][53] also reported on an association between laminitis and 'bolus-feeding' of carbohydrates. Twice daily feeding of large amounts of concentrates in the milking parlour can lead to reduced forage intake with post-feeding episodes of ruminal acidosis and subsequent laminitis.[8][54] In contrast, Smit *et al.*[55] found no correlation between laminitis and feeding rations high in concentrate. Similarly, Frankena *et al.*[56] reported a negative association between the feeding of concentrates and the prevalence of sole haemorrhages in dairy calves. They concluded that nutrition may not be the most important factor, but that behaviourial, management and genetic factors are of considerable importance as well.

Intensively fed feedlot cattle receiving rations high in energy have a high prevalence of laminitis.[7][57] Similarly, the widespread practice of intensive feeding of young bulls at bull testing stations in order to achieve heavy weight gains by the time these animals enter their second year of life ('fitting' of young bulls for bull sales) has also been associated with laminitis.[58]

Roughage/fibre -- The quantity and quality of roughage (fibre) has been suggested as a major factor influencing the occurrence of laminitis.[59] A sudden increase in concentrates at the expense of a low quantity of roughage, which is often fed some time after the concentrates, is recognised as being very conducive to laminitis.[15][17] A high-fibre diet (19% crude fibre in the dry matter) led to less clinical laminitis than a lower crude fibre (16.5%) ration in a dairy herd in which crude protein and metabolisable energy concentrations were otherwise similar.[17]

At least one third of the total dry matter intake of a cow should consist of roughage with

the necessary qualities to guarantee good function of the rumen as well as structure in the rumen content.[24] An adequate particle length is required to ensure rumination and saliva flow; 2.5 cm or greater has been suggested.[60] Feeding of long-stem roughage is incompatible with the modern concept of a complete or total mixed diet, but not with a semi-total mixed ration. The provision of some long-stem hay may improve the animals' capacity to maintain rumen pH. From the study of Frankena *et al.*[56], it appeared that feeding of hay to dairy calves might be preventive for subclinical laminitis.

Other nutritional factors -- Vermunt[19] speculated that the high nitrate levels common to New Zealand's pastures may produce blood levels of nitrite capable of vasodilation and endothelial damage resulting in the leakage of blood components into the corium. Another dietary factor which may be of importance in the aetiology of laminitis is the presence of mycotoxin in fungi-damaged feedstuffs.[4][19] Mycotoxicosis can produce a haemorrhagic syndrome in cattle.[61]

Research has not fully established all the links between nutrition and subsequent related laminitis, but major changes of the diet for cows at calving, heavy feeding of concentrates and/or protein with little roughage after calving, and high proportions of cereals, e.g. barley, in the diet are all thought to be predisposing causes of laminitis.

The hypothesis concerning the nutritional factors in the aetiology and pathogenesis of laminitis is supported by successful preventive measures stipulated by Weaver[15] and Peterse.[62]

Management

Management factors considered important in the aetiology of laminitis are housing, in particular the quality of the floor surface, bedding and exercise.

Housing -- Numerous authors have stressed the importance of housing in the initiation of claw lesions. However, little experimental research has been conducted concerning the influence of housing on lameness, and more specifically laminitis, in dairy cattle. Nilsson[63] described a form of overloading laminitis in heifers recently introduced to concrete. Bazeley and Pinsent[39], and David[64] made similar observations. The incidence of laminitis was greatly reduced when heifers were gradually introduced to concrete several weeks before calving as recommended by Weaver.[15] Bergsten[53] found a positive correlation between laminitis-like lesions and concrete flooring.

Laminitis in heifers newly integrated into a herd housed on concrete has been observed by numerous workers, but few have tried to explain the possible aetiology of the condition in this type of animal.[8][19][39][60][65] Clinical studies of herds affected by laminitis have allowed a number of predisposing factors to be elucidated. These include sudden introduction to concrete surfaces and cubicles, lack of bedding and poor housing and cubicle design.

Shearing of horn tubules occurs when animals are constantly twisting and turning in narrow and poorly designed alleys[39], and by attempts to avoid confrontation.[65][66] Cattle may refuse to use cubicles which are uncomfortable or in which they have difficulties in rising.[67] Consequently, resting time will be adversely affected in situations where cubicle partitions are poorly designed and/or cubicle dimensions do not meet the space requirements of the animal.[60][67] The occupancy rate of the Newton Rigg cubicles by in-calf heifers was less than observed for the Dutch Comfort cubicles and, after calving, claw health deteriorated less rapid in the animals housed in the latter.[68]

In countries where year-round grazing is practised, housing is not a factor in the aetiology of laminitis. In these situations, laminitis in dairy cattle is associated with the

length, quality and design of the farm track, with the movement of animals on abrasive surfaces at each yarding, and with the length of time spent on concrete yards.[19][66][69][70][71]

Bedding -- The importance of a soft resting area in relation to claw lesions has been recognized.[39][56][64][65][72] Soft bedding results in longer resting times and less lameness associated with laminitis[65], supporting the importance of the burden factor (compressive stress or loading). Yielding surfaces will better distribute the load over the weight-bearing surface of the individual claw as well as between the lateral and medial claw of each limb.

Exercise -- Relative physical confinement leading to inadequate exercise has been suggested as a predisposing cause of laminitis.[15] The management of modern dairy cows requires that they be confined on hard surfaces for prolonged periods of time with limited opportunity for exercise. Also, the introduction of cattle to a feedlot and the restrictive penning of bulls for feeding trials causes abrupt changes in the amount of exercise. A significant reduction in the amount of exercise will decrease the blood flow through the corium of the claw making it more susceptible to insult.[59]

Similar to the horse[73], over-exercise, particularly on hard surfaces, has also been associated with the acute onset of laminitis in cattle due to compression and traumatic tearing of the laminae and papillae.[74]

Calving

A disproportionate number of cases of laminitis and its associated lesions occur in the first three months of lactation. Nilsson[9] used the term 'parturition laminitis' and suggested that histamine, which is produced in the uterus in cases of retained placenta and metritis, causes this type of laminitis. Laminitis, alleged to be associated with other common post-partum diseases, such as mastitis, ketosis and

particularly ruminal acidosis, occurs predominantly within a short period after calving.[13][15][17][52]

Sole lesions associated with laminitis are also most frequently observed in freshly-calved cows.[6][8][16][46][60][75][76] Often there is a history of a high-carbohydrate/low-roughage ration being fed.

Undoubtedly, calving and associated problems during the early post-partum period are important[76], but their exact role is not clear. It is difficult to separate the influences which the confounding factors of calving, stage of lactation and level of production have on the occurrence of laminitis. The highest level of production is invariably reached in early lactation, within three months of calving. This is also the period of management and rapid feed changes culminating in a maximum concentrate intake.

Season

Nilsson[9] observed the highest incidence of laminitis during the housing (winter) period, whereas Maclean[10] found that laminitis peaked in the spring. Peterse [77] concluded that the season of calving (housing period vs. grazing period) is a factor in the incidence of laminitis. It seems reasonable to suggest that, in dairy cattle, the increased seasonal incidence of laminitis is probably associated with housing and/or calving and the accompanying sudden change in the ration.

Bargai *et al.*[36] suggested that the seasonally repeated outbreaks of laminitis in young dairy calves were due to the calves' larger feed intake in the winter. Vermunt[60][78] used prolonged dilation of arteriovenous shunts at times of seasonal extreme cold as a possible explanation for the unexpected, high incidence of laminitis in dairy heifers housed outdoors.

In Australasia, a pronounced seasonal incidence of lameness and lesions associated with laminitis occurs, with most cases in late winter

and spring.[66][69][70][79] A distinct connection between calving pattern and lameness also exists in these year-round, pasture-fed herds. Calving and the period up to peak lactation coincide with maximum grass growth during spring. Vermunt[19] suggested that, amongst others, the high-protein/low-fibre content in the lush growing rye grass pasture may be a potential causative factor of subclinical laminitis.

Age

Laminitis in young dairy and beef calves has been described by several authors[4][6][7][36][60]. An age predisposition to laminitis has also been observed. Nilsson[9] found a high level of laminitis in first-calf heifers in high-producing herds. Moser and Divers[52] made a similar observation. Edwards[13] and Bradley *et al.*[6] stated that heifers and young cows are most prone to laminitis. Frankena *et al.*[56] found a positive relationship between age and the prevalence of sole haemrrhages in dairy calves.

As an acute disease, laminitis is seen most dramatically in heifers about the time of their first calving.[80] The common observation that laminitis is more often encountered in heifers than in cows, can partly be attributed to the fact that many heifers are fed little or no concentrate from an early age until they enter the milking herd. At that stage, the total intake of roughage is likely to be more restricted than the concentrate intake. Furthermore, heifers are also introduced to concrete surfaces as calving approaches.

Growth

Laminitis of varying degree is a common condition during the early growing period of dairy heifers.[6][60] However, evidence of a link between rapid growth during rearing and laminitis is scarce. Rapid rearing of dairy heifers, resulting in mean body weight gains exceeding 1 kg/day has been implicated in the occurrence of laminitis.[34] Greenough and Vermunt[8] suggested that growth rates greater than 0.8 kg/day may be

a factor in subclinical laminitis. High planes of nutrition giving these rapid growth rates must involve a low roughage intake, which has been associated with rumen acidosis and laminitis.[17] In his study with dairy heifers, Vermunt[60] found no evidence of a correlation between lesions associated with laminitis and average daily gain of 0.8 kg from birth to breeding.

Genetics

In this context, inherited factors implicated in the aetiology of laminitis include the breed and type of animal. A familial predisposition to laminitis has been observed by Nilsson[9] and Maclean.[10] Swedish Friesians are more often affected by lesions associated with laminitis than Swedish Red and White cattle.[9][81] Brochart[82] noticed a greater susceptibility to laminitis in Friesian cattle than in Holstein Friesian-cross cows. Peterse[75] reported a tendency for a higher incidence of laminitis in Dutch Friesians when compared with Holstein and Meusse-Rhein-Yssel cattle.

Although it is tempting to consider that particular strains of cattle are susceptible to laminitis[59][83], an inherited tendency towards laminitis has only been demonstrated in Jersey cattle in South Africa[84], in the USA[85] and in the UK.[86] Characteristics of claw and body conformation are known to be heritable and this could possibly explain the observed difference in susceptibility to laminitis between breeds of cattle.

Conformation

General conformation refers to body weight, shape of body and limbs (in particular hock angulation), and claw size and shape. There has been a universal move in the cattle industry to select animals on the basis of straight hocks.[59] Excessively straight legs ('post-legged') make the animal vulnerable to joint injury and claw problems.[60] Decreased angulation of the stifle and the hock joints reduces shock absorption by the muscles, the ligaments, and the tendons.

Greater force is thus received by these joints and ultimately the claws. Straight hocks, combined with excessive body weight and small claws, confers even more compressive stress on the terminal structures within the claw during weight bearing.

There are dangers in relating conformation traits to laminitis. It is necessary to be prudent when making these correlations in animals older than two years of age, because the combined effects of age and management can change hock angulation as well as claw shape and size.[60] Furthermore, care has to be taken to avoid confusion between cause and effect.

Behaviour

There is little documentation available on the behaviour of cattle and its relationship with laminitis.[64][65][87]

David[64] suggested that introduction to unfamiliar cubicle (free-stall) housing, which may not be well accepted in the first weeks, may lead to cows spending most of their time standing and walking on concrete. Colam-Ainsworth *et al.*[65] observed aberrant behaviour of replacement heifers in a dairy herd with a high incidence of laminitis. The animals were standing in the cubicles, were lying outside or half-in the cubicles and were being evicted from cubicles by dominant cows. In this particular case, the use of cubicles was related to their comfort (bedding). Singh *et al.*[87] suggested that lying behaviour was a significant factor in the development of lesions associated with subclinical laminitis. First-lactation cows showed more aberrant behaviour and had more severe lesions than adult cows.

Inadequate numbers of cubicles and too narrow passageways may create a problem for low-ranked heifers. In efforts to avoid aggressive confrontation with dominant cows, they may refuse using the cubicles and abruptly twist and turn away. These conditions could result in claws being subjected to increased abrasion and compressive stress as well as unnatural shearing

forces.[60] Also, heifers are often last to enter the milking parlour and bails and therefore spent long periods standing and scuffling on concrete yards.

Conclusions

To isolate one possible predisposing factor from another is difficult, if not impossible. However, it is the total effect of all factors combined on the occurrence of laminitis which is important.

Laminitis is said to be a stress-associated condition.[39] The many changes in feeding, management, environment and social grouping, to which heifers in particular are subjected around the calving period, likely cause considerable stress at a time when their body metabolism is changing rapidly.

References

(1) Russell AM, Rowlands GJ, Shaw SR, Weaver AD. Survey of lameness in British dairy cattle. Veterinary Record 111, 155-60, 1982.

(2) Whitaker DA, Kelly JM, Smith EJ. Incidence of lameness in dairy cows. Veterinary Record 113, 60-2, 1983.

(3) Choquette-Lévy L, Baril J, Lévy M, St-Pierre H. A study of foot disease of dairy cattle in Quebec. Canadian Veterinary Journal 26, 278-81, 1985.

(4) Maclean CW. Observations on laminitis in intensive beef units. Veterinary Record 78, 223-31, 1966.

(5) Peterse DJ. Lameness in cattle. Pp 1015-23. Proceedings of the XIVth World Congress on Diseases in Cattle, Dublin, 1986.

(6) Bradley HK, Shannon D, Neilson DR. Subclinical laminitis in dairy heifers. Veterinary Record 125, 177-9, 1989.

(7) Greenough PR, Vermunt JJ, McKinnon JJ, Fathy FA, Berg PA, Cohen RDH. Laminitis-like changes in the claws of feedlot cattle. Canadian Veterinary Journal 31, 202-8, 1990.

(8) Greenough PR, Vermunt JJ. Evaluation of subclinical laminitis in a dairy herd and

observations on associated nutritional and management factors. Veterinary Record 128, 11-7, 1991.
(9) Nilsson SA. Clinical, morphological and experimental studies of laminitis in cattle. Acta Veterinaria Scandinavica [Supplement] 4, 9-304, 1963.
(10) Maclean CW. Observations on acute laminitis of cattle in south Hampshire. Veterinary Record 77, 662-72, 1965.
(11) Maclean CW. The long-term effects of laminitis in dairy cows. Veterinary Record 89, 34-7, 1971.
(12) Greenough PR, MacCallum FJ, Weaver AD. Lameness in Cattle. J.B. Lippincott Company, Philadelphia, 1981.
(13) Edwards GB. Acute and subacute laminitis in cattle. Veterinary Annual 22, 99-106, 1982.
(14) Toussaint Raven E. Lameness in cattle and footcare. Netherlands Journal of Veterinary Science 5, 105-11, 1973.
(15) Weaver AD. The prevention of laminitis in dairy cattle. The Bovine Practitioner 14, 70-2, 1979.
(16) Peterse DJ. Nutrition as a possible factor in the pathogenesis of ulcers of the sole in cattle. Tijdschrift voor Diergeneeskunde 104, 966-70, 1979.
(17) Livesey CT, Fleming FL. Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. Veterinary Record 114, 510-12, 1984.
(18) Garner HE, Coffman JR, Hahn AW, Hutcheson DP, Tumbleson ME. Equine laminitis of alimentary origin: An experimental model. American Journal of Veterinary Research 36, 441-4, 1975.
(19) Vermunt JJ. 'Subclinical' laminitis in dairy cattle. New Zealand Veterinary Journal 40, 133-8, 1992.
(20) Maclean CW. The histopathology of laminitis in dairy cows. Journal of Comparative Pathology 81, 25-8, 1971.
(21) Mortensen K, Hesselholt M, Basse A. Pathogenesis of bovine laminitis (diffuse

aseptic pododermatitis). Experimental models. Pp 1025-30. Proceedings of the XIVth World Congress on diseases in cattle, Dublin, 1986.
(22) Boosman R. Bovine laminitis; Histopathologic and arteriographic aspects and its relation to endotoxaemia. Unpublished PhD Thesis, Rijksuniversiteit Utrecht, 1990.
(23) Vermunt JJ, Leach DH. A scanning electron microscopic study of the vascular system of the bovine hind claw. New Zealand Veterinary Journal 40, 146-54, 1992.
(24) Greenough PR. The subclinical laminitis syndrome. The Bovine Practitioner 20, 144-9, 1985.
(25) Grant RT. Observations on direct communications between arteries and veins in the rabbit ear. Heart 15, 281-303, 1930.
(26) Walder DN. Arteriovenous anastomoses of the human stomach. Clinical Science 11, 59-68, 1952.
(27) Clark ER. Arterio-venous anastomoses. Physiological Reviews 18, 229-40, 1938.
(28) Pollitt CC, Molyneux GS. A scanning electron microscopic study of the dermal circulation of the equine foot. Equine Veterinary Journal 22, 79-87, 1990.
(29) Toussaint Raven E. Cattle Footcare and Claw Trimming. P 59. Farming Press Ltd., Ipswich, 1985.
(30) Takahashi K, Young BA. Effects of grain over-feeding and histamine injection on physiological responses related to acute bovine laminitis. Japanese Journal of Veterinary Science 43, 375-85, 1981.
(31) Hood DM, Stephens KA. Physiopathology of equine laminitis. Compendium of Continuing Education for the Practising Veterinarian 3, S454-9, 1981.
(32) Hunt RJ. The pathophysiology of acute laminitis. Compendium of Continuing Education for the Practising Veterinarian 13, 1003-10, 1991.

(33) Weaver AD. Solar penetration in cattle: its complication and economic loss in one herd. Veterinary Record 89, 288-90, 1971.
(34) Little W, Kay RM. The effects of rapid rearing and early calving on the subsequent performance of dairy heifers. Animal Production 29, 131-42, 1979.
(35) Manson FJ, Leaver JD. The influence of dietary protein intake and of hoof trimming on lameness in dairy cattle. Animal Production 47, 191-9, 1988.
(36) Bargai U, Shamir I, Lublin A, Bogin E. Winter outbreaks of laminitis in dairy calves: aetiology and laboratory, radiological and pathological findings. Veterinary Record 131, 411-14, 1992.
(37) Urmas P. A new hypothesis about laminitis. Finsk Veterinaer-tidskrift 74, 11-20, 1968.
(38) Chew KH. Subacute/chronic laminitis and sole ulceration in a dairy herd. Canadian Veterinary Journal 13, 90-3, 1972.
(39) Bazeley K, Pinsent PJN. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Veterinary record 115, 619-22, 1984.
(40) Hungate RE, Dougherty RW, Bryant MP, Cello RM. Microbiological and physiological changes associated with acute indigestion in sheep. Cornell Vet 42, 432-49, 1952.
(41) Dunlop RH. Pathogenesis of ruminant lactic acidosis. Advanced Veterinary Science 16, 259-309, 1972.
(42) Dougherty RW, Coburn KS, Cook HM, Allison MJ. Preliminary study of appearance of endotoxin in circulatory system of sheep and cattle after grain engorgement. American Journal of Veterinary Research 36, 831-2, 1975.
(43) Nagaraja TG, Bartley EE, Fina LR, Anthony DH. Relationship of rumen gram-negative bacteria and free endotoxin to lactic acidosis in cattle. Journal of Animal Science 47, 1329-36,1978.
(44) Ahrens FA. Histamine, lactic acid and hypertonicity as factors in the development

of rumenitis in cattle. American Journal of Veterinary Research 28, 1335-8, 1967.
(45) Peterse DJ, Korver S, Oldenbroek JK, Talmon FP. Relationship between levels of concentrate feeding and the incidence of sole ulcers in dairy cattle. Veterinary Record 115, 629-30, 1984.
(46) Bergsten C, Andersson L, Wiktorsson L. Effect of feeding intensity at calving on the prevalence of subclinical laminitis. Pp 33-8. Proceedings of the Vth International Symposium on Disorders of the Ruminant Digit, Dublin, 1986.
(47) Morrow DA. Laminitis in cattle. Veterinary Medicine/ Small Animal Clinician 61, 138-40, 1966.
(48) Peterse DJ, Van Vuuren AM, Ossent P. The effects of daily concentrate increase on the incidence of sole lesions in cattle. Pp 39-46. Proceedings of the Vth International Symposium on Disorders of the Ruminant Digit, Dublin, 1986.
(49) Peterse DJ, Van Vuuren AM. The influence of a slow or rapid concentrate increase on the incidence of foot lesions in freshly calved heifers. Proceedings of the European Association of Animal Production (EAAP) Congress, The Hague, 1984.
(50) Manson FJ, Leaver JD. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Animal Production 47, 185-90, 1988.
(51) Manson FJ, Leaver JD. The effect of concentrate:silage ratio and hoof trimming on lameness in dairy cattle. Animal Production 49, 15-22, 1989.
(52) Moser EA, Divers TJ. Laminitis and decreased milk production in first-lactation cows improperly fed a dairy ration. Journal of the American Veterinary Medical Association 190, 1575-6, 1987.
(53) Bergsten C. Sole bruising as an indicator of laminitis in cattle. A field study. Pp 1072-6. Proceedings of the XVth World Buiatrics Congress, Palma de Mallorca, 1988.

(54) Blowey R. Diseases of the bovine digit. Part 2. Hoof care and factors influencing the incidence of lameness. In Practice 14, 118-24, 1992.
(55) Smit H, Verbeek B, Peterse DJ, Jansen J, McDaniel BT, Politiek RD. The effect of herd characteristics on claw disorders and claw measurements in Friesians. Livestock Production Science 15, 1-9, 1986.
(56) Frankena K, Van Keulen KAS, Noordhuizen JP, Noordhuizen-Stassen EN, Gundelach J, de Jong DJ, Saedt I. A cross-sectional study into prevalence and risk indicators of digital haemorrhages in female dairy calves. Preventive Veterinary Medicine 14, 1-12, 1992.
(57) Hale WH. Liver abscess and founder. Animal Health and Nutrition 40, 12-20, 1985.
(58) Greenough PR, Gacék Z. A preliminary report on a laminitis-like condition occurring in bulls under feeding trials. The Bovine Practitioner 22, 176-8, 1987.
(59) Greenough PR. Observations on bovine laminitis. In Practice 12, 169-73, 1990.
(60) Vermunt JJ. Lesions and structural characteristics of the claws of dairy heifers in two management systems. Unpublished MSc Thesis, University of Saskatchewan, 1990.
(61) Blood DC, Radostits OM. Veterinary Medicine. 7th Edtn. Bailliere Tindall, Toronto, 1989.
(62) Peterse DJ. Prevention of laminitis in Dutch dairy herds. Proceedings of the IVth International Symposium on Disorders of the Ruminant Digit, Paris, 1982.
(63) Nilsson SA. Recent opinions about cause of ulceration of the hoof of cattle. Nordisk Veterinaermedicin 18, 241-52, 1966.
(64) David GP. Cattle behaviour and lameness. Pp 79-86. Proceedings of the Vth International Symposium on Disorders of the Ruminant Digit, Dublin, 1986.
(65) Colam-Ainsworth P, Lunn GA, Thomas RC, Eddy RG. Behaviour of cows in cubicles and its possible relationship with laminitis in

replacement dairy heifers. Veterinary Record 125, 573-5, 1989.
(66) Dewes HF. Some aspects of lameness in dairy herds. New Zealand Veterinary Journal 26, 147-8 & 157-9, 1978.
(67) Cermák JP. The design of cubicles for British Friesian dairy cows with reference to body weight and dimensions, spatial behaviour and upper leg lameness. Pp 119-28. In: Wierenga HK, Peterse DJ (eds). Cattle Housing Systems, Lameness and behaviour. Martinus Nijhoff Publishers, Boston, 1987.
(68) Leonard N, O'Connell J, O'Farrell. The effect of cubicle design on behaviour and foot lesions in a group of in-calf heifers. Proceedings of the VIIth International Symposium on Disorders of the Ruminant Digit, Rebild, 1993, (in press).
(69) Harris DJ, Hibburt CD, Anderson GA, Younis PJ, Fitspatrick DH, Dunn AC, Parsons IW, McBeath NR. The incidence, cost and factors associated with foot lameness in dairy cattle in south-western Australia. Australian Veterinary Journal 65, 171-6, 1988.
(70) Chesterton RN, Pfeiffer DU, Morris RS, Tanner CM. Environmental and behaviourial factors affecting the prevalence of foot lameness. New Zealand Veterinary Journal 37, 135-42, 1989.
(71) Tranter WP, Morris RS, Williamson NB. A longitudinal study of the hooves of non-lame cows. New Zealand Veterinary Journal 39, 53-7, 1991.
(72) Cermák JP. Comfortable cubicles for profit. Proceedings of the RASE "Cow Cubicles" Conference, Stone Leigh, 1983.
(73) Baxter GM. Equine laminitis. Equine Practice 14, 13-22, 1992.
(74) Dewes HF. Transit-related lameness in a group of Jersey heifers. New Zealand Veterinary Journal 26, 45, 1979.
(75) Peterse DJ. De beoordeling van de runderklauw op basis van het optreden van

zoollaesies. Unpublished PhD Thesis, Rijksuniversiteit Utrecht, 1980.
(76) Kempson SA, Logue DN. Ultrastructural observations of the hoof horn from dairy cows: changes in the white line during the first lactation. Veterinary Record 132, 524-7, 1993.
(77) Peterse DJ. Het optreden van zoollaesies bij melkvee. Tijdschrift voor Diergeneeskunde 107, 132-7, 1982.
(78) Vermunt JJ. A possible role of the environment by means of inappropriate activity of arteriovenous anastomoses in the pathogenesis of bovine laminitis. Pp 121-4. Australian Association of Cattle Veterinarians Pan Pacific Proceedings, Sydney, 1991.
(79) Tranter WP, Morris RS. A case study of lameness in three dairy herds. New Zealand Veterinary Journal 39, 88-96, 1991.
(80) Weaver AD. Cattle foot problems part 2: diseases of the horn and corium. Agri-Practice 9, 35-40, 1988.
(81) Andersson L, Lundström K. The influence of breed, age, bodyweight and season on digital diseases and hoof size in dairy cows. Journal of Veterinary Medicine A, 28, 141-51, 1981.
(82) Brochart M. Foot lameness of the cow, a multifactorial disease. Pp 159-65. In: Wierenga HK, Peterse DJ (eds). Cattle Housing Systems, Lameness and behaviour. Martinus Nijhoff Publishers, Boston, 1987.
(83) Whitaker DA. Causes of laminitis in cattle. Veterinary Record 131, 471-2, 1992.
(84) De Boom HPA, Adelaar TF, Terblanche M. Hereditary laminitis of Jersey cattle, a preliminary report. Proceedings of the 3rd Congress of the South African Genetic Society, 1968.
(85) Merrit AM, Riser WH. Laminitis of possible hereditary origin in Jersey cattle. Journal of the American Veterinary and Medical Association 153, 1074-5, 1968.

(86) Edwards GN. Hereditary laminitis in Jersey cattle. Pp 663-8. Proceedings of the VIIth World Buiatrics Congress, London, 1972.
(87) Singh SS, Ward WR, Lautenbach L, Murray RD. Behaviour of lame and normal dairy cows in cubicles and in a straw yard. Veterinary Record 133, 204-8, 1993.

PREVALENCE OF LESIONS OF SUBCLINICAL LAMINITIS IN FIRST LACTATION COWS FROM HIGH PRODUCTION OHIO HOLSTEIN HERDS

R.H. Smilie[2], *K.H. Hoblet*[1], *W.P. Weiss*[3], *M.L. Eastridge*[3], *D.M. Rings*[2], *G.L. Schnitkey*[4]

[1]Veterinary Preventive Medicine
[2]Veterinary Clinical Sciences
[3]Dairy Science
[4]Agricultural Economics

A cross-sectional observational study was conducted to evaluate lesions associated with subclinical laminitis (pododermatitis aseptica diffusa) in 203 first lactation cows and bred heifers in 13 high producing Ohio Holstein herds. Each herd selected for study consisted of more than 100 lactating cows and all herds were producing over 8500 kg of milk per cow on a rolling herd average basis. Lactating cows in all herds were housed in freestalls and maintained in confinement on concrete. Animals examined in each herd included primarily first lactation cows in their first 100 days of lactation as well as several heifers 30-60 days prior to calving. All animals were placed in lateral recumbency on a foot-trimming table for visual examination and photography of their feet. A front and a rear foot were examined and photographed on each animal. For purposes of scoring, the medial and lateral claws of each foot were divided into 6 zones according to the Liverpool system, 1990. For purposes of this study observable lesions considered associated with subclinical laminitis included yellow waxy discoloration of the sole, hemorrhage of the sole, separation of the white line and erosion of the heel (erosio ungulae). The prevalence of sole and heel lesions by anatomic location were determined within, and across herds, cows, feet, claws, and zones. Lesions associated with subclinical laminitis were found in all 13 herds. When days in milk for each animal was treated as a covariate there were differences in the prevalence of lesions between herds by pairwise comparison. The difference in the occurrence of lesions among herds appeared to facilitate classification of certain herds as being either better or worse than others according to lesion and anatomic location.

A SCORING SYSTEM TO EVALUATE LESIONS ASSOCIATED WITH SUBCLINICAL LAMINITIS IN HIGH PRODUCTION OHIO HOLSTEIN HERDS

R.H. Smilie[2], *K.H. Hoblet*[1], *W.P. Weiss*[3], *M.L. Eastridge*[3], *D.M. Rings*[2], *G.L. Schnitkey*[4]

[1] Veterinary Preventive Medicine
[2] Veterinary Clinical Sciences
[3] Dairy Science
[4] Agricultural Economics
The Ohio State University
Columbus, OH 43210, U.S.A.

A cross-sectional observational study was conducted to develop and validate a scoring system for evaluation of subclinical laminitis (pododermatitis aseptica diffusa) in 203 first lactation cows in 13 high producing Ohio Holstein herds. Each herd selected for study consisted of more than 100 lactating cows and all herds were producing over 8500 kg of milk per cow on a rolling herd average basis. Lactating cows in all herds were housed in freestalls and maintained in confinement on concrete. Animals examined in each herd included first lactation cows primarily in their first 100 days of lactation and several heifers 30-60 days prior to calving. All animals were placed in lateral recumbency on a foot-trimmer's table for visual examination and photography of their feet. A front and a rear foot were examined and photographed on each animal. For purposes of scoring, the medial and lateral claws of each foot were divided into 6 zones according to the Liverpool system standard, 1990. Observable lesions considered associated with subclinical laminitis included hemorrhage of the sole, yellow waxy discoloration of the sole, separation of the white line, and erosion of the heel (erosio ungulae). The objective of this study was to validate a scoring system which can be used to evaluate and rank herds with regard to lesions associated with subclinical laminitis. Discoloration and hemorrhage of the sole were scored for severity according to the method described by Greenough in 1991. Lesions were also evaluated by a proposed alternative scoring system (Ohio method). In addition to scoring hemorrhage and discoloration of the sole, the Ohio method evaluated separation of the white line and erosion of the heel, which have also been considered associated with subclinical laminitis. The

Ohio method enabled formulation of a ranking system among herds. When days in milk for each animal were treated as a covariate there were significant differences in the occurrence of lesions between herds.

PREVALENCE AND SEVERITY OF LESIONS ASSOCIATED WITH SUBCLINICAL LAMINITIS IN HEIFERS AND COWS IN CONFINEMENT AND PASTURED OHIO HOLSTEIN HERDS

R.H. Smilie[2], *K.H. Hoblet*[1], *W.P. Weiss*[3], *M.L. Eastridge*[3], *D.M. Rings*[2], *G.L. Schnitkey*[4]

[1] Veterinary Preventive Medicine

[2] Veterinary Clinical Sciences

[3] Dairy Science

[4] Agricultural Economics
The Ohio State University
Columbus, OH 43210, U.S.A.

A cross-sectional observational study was conducted to evaluate and score lesions associated with subclinical laminitis (pododermatitis aseptica diffusa) in 140 growing and bred heifers, and first lactation cows in 5 confinement and 2 pastured Ohio Holstein herds in 1993. The prevalence and severity of lesions associated with subclinical laminitis had been evaluated in first lactation cows and bred heifers in 4 of the confinement herds in 1992. Lactating cows in 5 herds were housed in freestalls and maintained in confinement on concrete. For purposes of comparison, 2 additional herds which were maintained primarily on pasture were examined. All animals were placed in lateral recumbency on a foot-trimming table for visual examination and photography of their feet. A front and rear foot were examined and photographed on each animal. Observable lesions considered associated with subclinical laminitis included hemorrhage of the sole, yellow waxy discoloration of the sole, separation of the white line, and erosion of the heel (erosio ungulae). For purposes of scoring, the medial and lateral claws of each foot were divided into 6 zones according to the Liverpool system standard, 1990. The prevalence and severity of scored sole and heel lesions by anatomic location were determined within, and across herds, cows, feet, claws, and zones. Discoloration and hemorrhage of the sole were scored for severity according to a method described in 1991. Lesions were also evaluated by a proposed alternative scoring system (Ohio method). In addition to scoring hemorrhage and discoloration of the sole, the Ohio method evaluated separation of the white line and erosion of the heel. Apparent differences were observed in the occurrence of lesions among similar

animals in the 4 confinement herds which were examined in both years. The differences were not found consistent with respect to type of lesion between herds. It appears that the occurrence of lesions varied among similar animals within the same herd examined in different years. Differences in the occurrence of erosion of the heel were found when herds confined primarily on concrete were compared to herds maintained primarily on pasture.

Pathophysiological studies in dairy cattle affected with subclinical laminitis

Ch. STANEK, F. BRANDEJSKY, M: SCHUH
Clinic of Orthopedics in Ungulates, University of Veterinary Medicine, Vienna, Austria

Summary

The claw score was evaluated in a herd of 50 dairy cattle (36 heifers, 14 cows) over a time period of 2 months before parturition and 6 months after parturition. The ruminal function was evaluated on day 1 after parturition, the blood coagulation factors calcium-thromboplastin, partial thromboplastin, partial thromboplastin time and antithrombin III were evaluated on day 1 and day 8 after parturition. A significant increase of the claw score was observed in the prepartal phase, indicating a deterioration of the claw status. The score remained on the same level thereafter. Claw score, occurrence of ruminal acidosis and blood coagulation factors were found uncorrelated.

Introduction and literature

In high yielding dairy cattle, laminitis and resulting complications, like *Pododermatitis circumscripta* (Sole ulcer), infections in the white line area or solear abscessations, have an increasing importance causing economic losses and early culling of cows. In the last decade significant changes in the clinical picture of laminitis have been observed. The acute and subacute forms with a clinical picture similiar to the acute laminitis in

the horse are not as predominant. In addition to these classical forms (acute, subacute, and chronic) the subclinical form is becoming more and more important. The subclinical form is impressive in that typical claw lesions like circumscribed sangineous imbibitions and hemorrhages in the sole area, defects on the Zona alba and the formation of grooves on the wall are observed, but without any distinct lameness or changes in the general condition of the animal. Although the condition is seen in beef cattle, the primary affected group is dairy cattle.

The facts which can be taken out of literature regarding the pathogenesis of this subclinical form of laminitis in cattle are limited. An important proven fact is the influence of feeding high carbohydrate, high protein, low fiber diets especially around the time of calving. This diet induces ruminal acidosis with a shift in ruminal flora and damage to the ruminal mucosal wall. The direct correlation between the feeding level, the frequency, and severity of claw diseases has been proven (LIVESEY and FLEMING; 1984; MANSON and LEAVER, 1988a, 1988b, 1989; MORTENSEN et al., 1986; GREENOUGH, 1990). To what extent that ruminal disturbances influence claw disease, however, has not yet been thoroughly studied in a larger group of animals under field conditions.

Other factors, which must be taken into consideration regarding the pathogenesis of subclinical laminitis in the bovine species, are, for example, hormonal, social and housing factors correlating with the birth of a calf. The large number of important contributing factors explains the variable

epidemiological data: in intensive fed animals the majority of complications is seen in first calving cows, whilst these types of complications were observed at the Clinic of Orthopedics, Vienna, in second and third calving cows.

The patho-physiological correlation between the mentioned causal factors and the occurence of lesions in the claw region leaves many unanswered questions. The occurence of a disseminated intravascular coagulation, like in the horse, can be assumed, although no literature was found dealing with the possible correlation to coagulation disturbances.

In this underlying study a herd of dairy cattle was examined in the parameters "claw status", "ruminal status", and "coagulation factors" in a time period between 2 months prior to parturition and 6 months after parturition (BRANDEJSKY et al., 1994)..

Animals, material and methods

50 Braunvieh - dairy cows of the experimental herd of the University of Veterinary medicine Vienna (36 heifers, 14 cows between 18 months and 4.5 yrs.) were evaluated. The average milk yield in the year of the investigation was 5077 kg. Reference values for blood coagulation factors were determined from 12 non pregnant cows of the herd. 11 cows were kept in a loose housing system prior to parturition, thereafter they were kept in tied stalls. The remaining 39 animals were kept in a the tied stall system with straw bedding throughout the year.

The status of the claws was evaluated on the following days:

---- in the 7. or 8. month of pregnancy
within the routinely performed claw trimming
---- 1 day after parturition
---- 2 months after parturition
---- 4 months after parturition
---- 6 months after parturition

By recording a detailed claw score, the status of the claws was judged numerically. The occurrence of the following pathological changes was evaluated: hemorrhages on the sole, grooves on the dorsal wall, the distance between these grooves and the coronary border, the profile of the sole, defects on the white line, double sole formation an sole ulcers. The individual measures evaluated in one claw were added to a total claw score. At the same time a general evaluation of the gait of the animal and of possible lameness was carried out.

In addition, on the first postpartal day the ruminal status was tested for ruminal acidosis. Using a stomach tube, ruminal content was taken and smell, colour, consistency was judged, pH measured and the number of protozoa estimated.One day and 8 days post parturition the following blood coagulation factors were determined: calcium-thromboplastin, partial thromboplastin time, thrombin time and antithrombin III.

Results

The individual values of the claw score showed a wide variation at all examined periods. In the interv al between the first and the second scoring, within the last 8 to 4 weeks prior to parturition, a significant deterioration of the claw condition could be observed (Claw score: mean .8 weeks a.p. x = 41,5, peripartal x = 83,2; p 1 %). In the

first 6 months the claw score remained app. at the same level, with an decreasing width of variation (Claw score mean: 2 mths. p.p. 74,8; 4 mths. p.p. 81,9; 6 mths. p.p. 74,0). Only in 4 of the 50 investigated cows clinical symptoms of acute laminitis were observable. In 23 cows a ruminal acidosis was diagnosed postpartal (15 animals low degree acidosis, 8 animals medium degree acidosis). There was no statistical correlation to the height of the pre- or postpartal claw score. In comparison to control animals, the experimental cows exhibited a higher value of calcium-thromboplastin and partial thromboplastin time. In heifers a higher throbin time was found compared to cows. No significant correlations between blood coagulation factors and the claw scores were evaluated at any time.

Discussion

In this study various aspects are discussed. The first point of interest was the recording of the claw status in a larger group of animals over a longer period of time. This was done by recording the claw status in the time between 2 months prior to parturition and 6 months after parturition.The claw status worsened significantly in the pre-parturition phase and then remained at approximately the same level. Serious claw disorders like sole ulcers or solear abscessations were rarely seen. The claw status became more uniform in the evaluated group with an increasing interval from parturition. Significant is, that although the claw status worsened, no symptoms of acute or subacute laminitis were present. PETERSE et al. (1984) found, in comparison, serious worsening of the claw status in the

post-parturition phase. They also recorded a higher frequency of ulcers.

According to the above results, significant worsening of claw status is to be expected even at low feeding and performance levels. GREENOUGH (1990) and MANSON and LEAVER 1988a, 1988b, 1989) found a proven significant correlation between energy and protein content in feed and the occurence of hemorrhages in the sole horn. Changes are to be expected
even at a low level. The fact remains that a correlation between increased feeding level and and an increasing rate of sole ulcers etc. exists (PETERSE et al., 1984).

The second point of interest was the influence of ruminal acidosis on the claw status. Even though the importance of ruminal acidosis to the pathogenesis of laminitis has been pointed out by many authors, studies under field conditions are lacking. Experimental studies performed under extreme feeding regimes cannot necessarily be applied (MORTENSEN et al., 1986). BAZELEY and PINSET (1984) emphasize the possibility of laminitis occurrence in herds with cases of acidosis, that are not always recognized by the farmer and therefore not presented to the veterinarian. In our study all animals were examined regardless of clinical symptoms for ruminal acidosis even at a very low degree. Ruminal acidosis was not correlated with changes in the claw.

The third point of interest was the possibility of disturbed blood coagulation in the period immediatly after parturition. No corresponding literature regarding this parameter could be found. In our study no correlation could be found between our blood

coagulation parameters, occurence of ruminal acidosis, and pathological changes of the claws. The question regarding blood coagulation disturbances in the period before parturition remains.

As a whole the use of the term "subclinical laminitis", which means the occurence of typical pathological changes to the claw without any general symptoms, is justified. It has to be noted, that the increase of the claw score was determined in the praepartal era and not in the months after parturition. The importance of the individual factors remains controversial.

Literature

BAZELEY, K., PINSET, P.J.N. (1984): Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Vet. Rec. **115,** 619 - 622.
BRANDEJSKY, F., STANEK, Ch., SCHUH, M. (1994): Zur Pathogenese der subklinischen Klauenrehe beim Milchrind: Untersuchungen von Klauenstatus, Pansenstatus und Blut-gerinnungsfaktoren. Dtsch. tierärztl. Wschr. **101,** 68 - 71.

GREENOUGH, P. R. (1990): Observations on bovine laminitis. In Practice **12,** 169 - 173.
LIVESEY, C.T., FLEMING, E. (1984): Nutritional influences on laminitis, sole ulcer, and bruised sole in Frisian cows. Vet. Rec. **114,** 510 - 512.
MANSON, F.J., LEAVER, J.D. (1988a): The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Anim. Prod. **47,** 185 - 190.
MANSON, F.J., LEAVER, J.D. (1988b): The influence of dietary protein intake and hoof trimming on lameness in dairy cattle. Anim. Prod. **47,** 191 - 195.
MANSON, F.J., LEAVER, J.D. (1989): The effect of concentrate : silage ratio and hoof trimming on lameness in dairy cattle. Anim. Prod. **49,** 15 - 22..
MORTENSEN, K., HESSELHOLT, M., BASSE, A. (1986): Pathogenesis of bovine laminitis (diffuse aseptic pododermatitis), Experimental models. Proc. 14th World Congr. Dis. Cattle, Dublin, 1025 - 1030.
PETERSE, D. J., KORVER, S., OLDENBROEK, J.K., TALMON; F.B. (1984): Relationship between levels of concentrate feeding and incidence of sole ulcers in dairy cattle. Vet. Rec. **115,** 629 - 630.

Anschrift der Verfasser: Univ. Doz. Dr. Christian Stanek, Linke Bahng. 11, A 1030 Wien , Austria. Tel. Vienna 71155/372; FAX Vienna 714 91 06.

LESIONS OF THE HOOF IN FIRST-CALVING DAIRY HEIFERS

D.N. Logue[1], J.E. Offer[1], K.A. Leach[2], S.A. Kempson[2], and J.M. Randall[3]

[1] The Scottish Agricultural College
Auchincruive, Ayr KA6 5HW, Scotland

[2] Royal (Dick) School of Vet Studies
Summerhall, Edinburgh EH9 1QH, Scotland

[3] Silsoe Research Institute, Wrest Park
Silsoe, Beds., U.K.

We have recently compared the appearance of the weight-bearing surface of the hooves of several groups of first-calving heifers from a series of different experiments including five summer-calving heifers which were unique in that they were housed over winter in a straw yard. The other groups were autumn-calving and had only been housed in cubicles from approximately 9 months of age. All groups showed a substantial increase in mean total severity score from 0 to 3 months post-calving. The apparent similarity of the development of lesions of the weight-bearing areas of the hoof in all groups could be due to an interaction of a variety of different factors rather than a failure of the inferred increased lying time in the summer-calving group to give a reduction in such lesions. Further studies examining the connection between lying time and the interaction of the hoof with its environment need to be undertaken preferably in first-calving cows.

RELATIONSHIP OF DIET, HOOF TYPE AND LOCOMOTION SCORE WITH LESIONS OF THE SOLE AND WHITE LINE IN DAIRY CATTLE

D.N. Logue[1], J.E. Offer[1], and J. Hyslop[2]

[1] The Scottish Agricultural College
Auchincruive, Ayr KA6 5HW, Scotland

[2] SAC, Crichton Royal Farm, Bankend Road
Dumfries DG1 4SZ, Scotland

Three groups of 16 cattle each comprising 3 multiparous Jersey cross Holstein-Friesian cows (JC "type") and 7 primiparous (HFH "type") and 6 multiparous Holstein-Friesian cattle (HFC "type") were offered, ad libitum, three silage-based complete diets with different ingredients following an initial three-week covariate period. The relationship between these three diets and other parameters with lesions of the weight-bearing surface of the hoof was studied by scoring locomotion weekly and examining all the feet of all cows. There was no significant difference between the three dietary groups for a simple overall lesion score, however, there were fewer ulcers of the sole in one diet ($p<0.001$). Using this overall score there were no significant differences between the three dietary treatments but there were significant differences in the incidence of lameness between "types" HFH, HFC and JC (0.35, 0.21 and 0.11 respectively), prevalence of lameness (0.12, 0.12 and 0.01) and the presence of a sole ulcer (0.22, 0.10 and 0.11). It was concluded firstly that the simple lesion score required some adjustment or correction factor(s) to ensure that more severe lesions were given a greater weight than a simple unitary increase and secondly that previous insults were of considerable importance in the development of lesions of hoof horn and in understanding the interaction of diet and lameness.

LESIONS OF THE FOOT AND ULTRASTRUCTURE OF THE WHITE LINE IN RELATION TO LAMENESS IN DAIRY CATTLE

D.N. Logue[1], J.E. Offer[1], and S.E. Kempson[2]

[1] The Scottish Agricultural College
Auchincruive, Ayr KA6 5HW, Scotland

[2] Royal (Dick) School of Vet Studies
Summerhall, Edinburgh EH9 1QH, Scotland

A group of 16 dairy heifers was regularly monitored for locomotion, hoof lesions, hoof growth and wear and hardness (shore A meter) just prior to and during their first lactation. There was a significant covariate effect of mean locomotion score on daily milk yield ($p<0.0001$) and significant positive correlations ($p<0.05$) were found between locomotion score, the appearance of certain hoof lesions, in particular solear haemorrhage and ulceration and a simple ultrastructural score. However, the correlation of lesions and ultrastructural score although significant ($p<0.001$) was not high ($r^2=0.522$) and thus any relationship is not simple. There was also a similar significant ($p<0.05$) but relatively low negative correlation between hoof hardness and hoof lesions. These findings suggest that the hypothesis that the ultrastructural appearance of the white line was related to visual lesions of the hoof and could therefore be used as an objective measurement of insult to the sensitive horn-producing epidermis is worth investigating further. They also showed that care of the young first-calving heifer before and after calving is essential to limiting lameness.

THE IMPACT OF A LAMENESS MANAGEMENT PROGRAMME

Jos J. Vermunt*† and Meg E. Smart
Department of Veterinary Internal Medicine, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Sask., S7N 0W0, Canada.
*Present address: Department of Veterinary Clinical Sciences, Massey University, Palmerston North, New Zealand.
†Author for correspondence.

Introduction

Lameness in dairy cattle has considerable economic and animal welfare implications. Studies in Europe have identified lameness as the third most costly health problem in dairy cows, after mastitis and reproduction.[1][2] Several estimations of the total annual cost of lameness have been made[1][3][4][5], varying from Can $10[4] to as much as Aus $43 per cow.[5] 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, there are intangible losses such as decreased milk production, reproductive efficiency and bodyweight, premature culling and replacements costs. The latter ones are the major costs, but are often ignored.

The negative impact on herd production due to lameness, even at the most conservative estimates, could be substantial. A retrospective survey study found that on average, 20% of the total lactation was lost from each affected cow.[1] Another herd-based case study reported a marked decrease in milk production progressing to agalactia within about 30 days after the onset of lameness due to laminitis in improperly fed cows.[6] One report estimated the average annual overall loss per lame cow to be approximately US $ 200.[7]

Lameness is not a disease but a clinical sign of a disorder that causes a disturbance in

locomotion. A shift in the causes of lameness in cattle 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.[1][8] However, progress in understanding the causes of lameness has been slow and is still very incomplete. Claw disorders account for 75-90% of diagnosed lameness in cattle.[4][8][9][10][11] There appears to be agreement, at least amongst authors from the northern hemisphere, that (subclinical) laminitis is the most important predisposing factor in lameness caused by claw disorders.[12][13][14][15]

Most laminitis problems in cattle are thought to be associated with type of feed. However, the hypothesis of a multifactorial aetiology extends beyond nutritional factors. Management and behaviourial aspects may be just as important and need further investigation.[16] Vermunt and Greenough[17] reviewed the numerous factors that are implicated as predisposing causes of laminitis. They include individual cow factors (systemic disease, age or parity, stage of lactation, conformation and genetics)[8][10][11][18][19] and herd-level factors (nutrition, housing and flooring, level of exercise, behaviour and environment).[5][11][14][20][21][22][23]

There is a growing awareness of the importance of investigation, diagnosis and prevention of lame cattle. Recognizing which factor(s) may be causing problems in an individual herd is still an art and requires a methodical on-farm investigation. At the herd level, feeding strategies, nutritional imbalances and housing are often found as common sources of laminitis and several authors have put forward suggestions on management and nutrition to assist in the prevention of this form of claw disorder.[6][7][13][24][25][26][27][28][29][30][31]

This paper describes the investigation of protracted lameness due to laminitis in a dairy

herd, the identification of the potential causative factors and the recommendations and rationale given to avoid this type of problem.

History

In October 1989, a lameness problem in a dairy herd located on a farm near Swift Current, Saskatchewan was referred to the Department of Veterinary Internal Medicine of the Western College of Veterinary Medicine, University of Saskatchewan. For several years, this high-yielding herd suffered from a high prevalence of lameness. The problem was described by the referring veterinarian as follows:

"Stable foot rot occurs in 80% of the cows within one month of calving. Erosion of the interdigital skin, but no swelling of the coronary band. Responds to copper sulphate topically, but a copper sulphate footbath appears ineffective. Common to most high-producing herds, signs of founder (laminitis) are observed in most of the cows. A large number of cows, mainly heifers and first-calf cows, develop severe lameness after calving and may remain lame for their entire lactation period. Also, some of the lame cows have deep cuts between the toes of their rear legs. Prolonged treatment with high doses of penicillin are necessary and often corns develop in the chronic cases, requiring surgical removal. Cows will milk well initially, but 50% drop off production at about 40 days after calving. They may go down in production as far as 10 lbs (4.5 kg) per day; some have an acute drop in production overnight to virtually no milk at all. Feed intake remains satisfactory and, generally, cows recover approximately 3 weeks later".

The average annual milk production for the last three years had been 29 kg per cow (DHI records).

There is always a real temptation to take short cuts either based on received information

together with first impressions, or based on previous experience of what may well mistakenly be assumed to be an identical problem. Identifying what is wrong in the total management of the herd is often extremely difficult. The cause of the problem is likely to be multifactorial and difficult to identify with total conviction. Therefore, when investigating a lameness problem, a detailed history taking and on-farm investigation must be carried out to establish which factor or combination of factors is the cause of the lameness. It is beyond the scope of this paper to include every detail of the investigation and only findings relevant to the problem (in the authors' opinion) are listed.

Farm investigation

To make a thorough investigation of any herd problem inevitably takes much longer than inspection of an individual cow. However, it is likely that a single herd examination will produce leads which may then need to be followed by further investigations involving a multidisciplinary approach such as involvement of a nutritionist or farm building engineer.

The farm was visited in early November 1989. The dairy herd comprised of 125 commercial Holsteins (no more than 60 in milk at one time). All animals were raised on the farm. Calvings were more or less evenly distributed throughout the year.

Lactating cows

The average milk yield at that time was 25 kg per animal. The lower actual production, when compared with previous years, was ascribed to the feeding of less grain and protein supplement as a measure to reduce 'nutritionally induced laminitis'.

Management -- The milking herd was divided into a high and regular group according to level of production and stage of lactation. Cows were housed in a cubicle (free-stall) building with grooved, non-slippery, concrete passageways.

They had no access to an exercise area out of doors and, therefore, were confined totally on concrete during the entire length of their lactation. The earth-based, strawed-in cubicles had wooden partitions and were arranged in two parallel lines along the long sides of the barn. The dimensions of the cubicle beds measured: effective length 2.35 m, clear width 1.20 m and kerb height 20 cm. There were always more cubicles than cows. Plans had been drawn up to replace the cubicles for concrete-based, lipless cubicles with tubular steel divisions. A limited amount of cut straw would be used as bedding.

The feed bunk was situated centrally along the long axis of the barn and was sufficiently long for all cows to feed together. The passageways, separating the cubicles from the feed bunk, were 2.50 m wide and cleaned regularly with automatic scrapers. At a few places, some strands in the steel cable pulling the scraper had split and sharp ends were protruding from the cable surface.

Cows were milked twice each day in a 4-aside, herringbone parlour at about 4.30 am and 16.00 pm.

Cows had their claws trimmed by the owners (self taught) only in case of claw lameness or horn overgrowth. Regular use of a footbath was not practised and only the dry cows were given a single treatment for 2 hours during which period they stood in the bath containing a very strong copper sulphate solution (calculated to be 20-25%).

Generally, the cows were thin, the majority being in light to moderate condition. Many cows were lying in the cubicles and appeared to be contented, but only a few cows were ruminating (cudding) when the herd was inspected between 9.00-10.00 am and again just before afternoon milking. Some cows in the milking herd were clearly lame and the majority were tender when forcibly moved along the concrete alleys.

Nutrition -- The feeding programme was based on very finely chopped alfalfa haylage (50% dry

matter (DM), 18% crude protein (CP) in the DM), which was fed in the feed bunk. The remainder of the forage intake was in the form of long-stemmed alfalfa hay (85% DM, 24% CP in the DM). A grain mix (58% barley, 35% oats and 7% beet pulp, with 84% DM, 17% CP and 77% total digestible nutrients in the DM) was fed on the haylage. Additional grain was fed in the milking parlour and through computerised feeding stalls situated at the end of the feed bunk near the exit of the parlour. A 38% protein supplement (43% CP in the DM) was also fed through these computer stalls at a daily rate of 1.5 kg per cow.

Additionally, cows received on the haylage (half in the morning and half in the afternoon):

a) a 1:1 (14% Ca:14% P) cattle mineral mix; 100 g/cow/day,
b) sodium bicarbonate; 115 g/cow/day,
c) magnesium oxide; 60 g/cow/day,
d) niacin; 6 g/cow/day, and
e) yea-sacc®; 20 g/cow/day.

Cows had free access to cobalt-iodised salt blocks. The drinking water was sourced from a well and was high in sulphates, i.e 1,750 ppm (recommended level is <1,000 ppm).

The daily feeding regime was as follows:

- 1.5 kg of grain in the parlour, twice daily at 4.30 am and 4.00 pm.
- *ad lib.* alfalfa haylage after morning milking and before afternoon milking (total estimated DM intake of 6 kg).
- 1.5 kg of grain on the haylage (twice daily).
- 2.5 kg of hay, twice daily at 10.00 am and 8.00 pm (total DM intake of 4 kg).
- 1.5 kg protein supplement and up to 6.5 kg of grain through the computer feeder.

Therefore, high-producing cows might have had a total grain and supplement intake of 14 kg per day, bringing the total daily DM intake, including forage, to approximately 22 kg (close to the maximum of 3-4% of the body weight).

During the previous 12 months, frequent changes had been made to the ration composition for various reasons such as slight reductions in production, shortage of components and laminitis. It was planned to replace the 1.5 kg of protein supplement with 2 kg of alfalfa pellets (20-22% CP) and to also add zinc methionine (Zinc-Pro®) to the ration.

Calves

Calves were born in the calving barn and put in individual floor pens (indoors) the same day. Weaned heifers were maintained out of doors in groups of three to six; the outside pens had south-facing shelters.

Young stock

Heifers were transferred to a breeding group at 12-13 months of age. This group was confined to an earth-surfaced corral with a well-bedded (straw), open-fronted shelter. The animals were bred at 15 months of age. They were fed 1.5 kg of grain per head per day and *ad lib.* alfalfa haylage, plus a 2:1 (16% Ca:8% P) mineral supplement. Additional grain was provided when the outside temperature fell below 20°C.

In-calf heifers

When the heifers were determined to be pregnant, they were maintained in a corral similar to that of the young stock. The ration consisted of alfalfa and rye grass hay *ad lib.* and some alfalfa haylage. A 1:1 mineral supplement was mixed in with the haylage. The animals were in good condition, appeared to be content and no lameness was observed. Three months prior to calving, the heifers were mixed with the dry cows.

Dry group

Dry cows and pregnant heifers were kept out of doors on a dirt lot. They had access to a well-bedded (straw), open-fronted shelter. The dry cow ration comprised of alfalfa and rye grass hay *ad lib.* and some alfalfa haylage, which acted

as a vehicle for the 1:1 mineral supplement. All cattle were in good condition (heifers slightly better than cows) and none appeared to be lame.

Two weeks prior to the expected calving date, dry stock were moved to a calving barn (loose-housing system) where a thick layer of straw was used as bedding. Grain was added to the ration; the rate increased by 0.5 kg per day.

Immediately after calving, cows were moved to the cubicle building to join the milking herd. They were offered the same basal daily ration (3 kg of grain mix, 5 kg of hay and alfalfa haylage *ad lib.*). Parlour feeding of grain also started at calving, but computer feeding of grain and supplement started 3 days after calving. The amount of grain fed through the computer feeder was gradually increased with daily increments of 0.5 kg.

Other findings

As part of the investigation, the claws of six first-calf cows, which had calved 1-4 months previously, were examined. All 8 claws of each cow were cleaned and pared. Varying degrees of interdigital dermatitis and heel horn erosion was invariably present in all animals, mainly in the hind limbs. Signs of (subclinical) laminitis were also observed and occurred predominantly in the claw horn of the weight-bearing surface of the lateral hind and medial front claws. The horn was physically soft (very easy to cut), stained yellow and rather waxy in appearance. Haemorrhages of varying severity were present in the white zone (white line), apex of the sole (toe) and axial side of the sole-bulb junction (specific site of sole ulcers). Two cows had actually developed early sole ulcers at the latter site. Double soles were found in two cows that had calved longest. On the basis of these clinical findings together with the high prevalence of lameness in the herd (10% or more of the herd affected annually[29] [32]), the presumptive diagnosis of (subclinical) laminitis, complicated by interdigital dermatitis and heel horn erosion, was made.

Samples of the various components of the ration, i.e. alfalfa hay and haylage, dairy grain mix and protein supplement were taken for further testing, i.e. moisture, protein, estimated energy (TDN), acid detergent fibre (ADF), calcium, phosphorus, nitrate and pH. The feed analysis results revealed no major abnormalities or deficiencies. However, the alfalfa haylage had a low ADF test (39% in the DM), a low acidity (pH of 5), a strong, tobacco-like smell and a small particle size (less than 1 cm).

A comparison was made of the ration fed to the lactating cows with computed requirements (Greenbrae Consultants, University of Saskatchewan, Saskatoon, Sask., S7N 0W0, Canada). The ration was well balanced when using the following criteria:

a) an average daily milk production of 27 kg with 3.6% fat,
b) on average, 150 days in milk, and
c) an average bodyweight of 650 kg.

Recommendations and rationale

As in other cattle problems of diverse and complicated aetiology, there is no guarantee that normal preventive advice will be successful. There are still many aetiological facets not yet properly elucidated or understood. Nevertheless, of what is known from the literature, numerous recommendations have been forwarded to reduce the incidence of lameness due to laminitis. They can be summarized as follows:

> Take special care with feeding in the pre and post-partum periods. Avoid sudden ration changes and feed in such a manner that peak yield is reached at about 6 weeks rather than 3-4 weeks. Provide immediate access to forage after concentrate is fed. Aim to keep the roughage to concentrate ratio in the 40:60 to 50:50 range. Be cautious when using fermented silage or haylage as the only roughage source.

> Rationally, the system of complete diet feeding reduces the risk of nutritionally induced laminitis to an absolute minimum.
> Make sure that the particle length of the roughage in the ration is such that it still provides the necessary quality for fibre without the risk of separation.
> The use of a buffering agent such as sodium bicarbonate (up to 1% of the grain ration) is recommended if long-stemmed roughage does not form a substantial part of the diet.
> Introduce down-calving heifers to concrete surfaces several months before calving.
> Provide comfortable resting areas; cubicles must have plenty of bedding and the correct dimensions. Train newly introduced cattle to use the facilities.
> Handle first-calving cows carefully during the first 60 days of lactation.
> Allow lactating cows as much exercise as practicable, preferably outdoors on pasture or dirt lots.
> Separate dry cows from the milking herd and keep them on dirt or grassed areas.
> Keep concrete surfaces clean and in a good state of repair; make sure they are non-abrasive and not slippery.
> Provide regular claw care, i.e. inspection, trimming and the use of a footbath.

The recommendations listed below apply to the herd and farm described in the present paper.

Change the feeding method to a total mixed ration (complete diet).

Feeding high-energy rations to dairy cows is essential to maintain high milk yield. The practise of feeding large amounts of concentrate in the absence of roughage, for example in the milking parlour and through computer feeders, could lead to an increased acidity in the rumen which is believed to predispose the animal to laminitis.[6][16][31][33][34] Total mixed rations are fed to dairy cows with the intention to stabilize the rumen environment over the feeding period,

thereby minimising the risk of rumen acidosis.[34] The adoption of complete-diet feeding after calving would assure a more or less continuous consumption of a ration with a constant concentrate/roughage ratio. However, the complete diet concept is based on group rather than individual feeding and usually involves few, if any, changes in diet formulation according to milk yield.[34] Therefore, accurate estimates of the concentrate intakes of individual cows can no longer be made.

The change to a total-mixed ration implied the discontinuation of feeding concentrate in the parlour and the computer stalls. For practical reasons, cows could be fed a limited amount (0.5 kg) of grain as a topdressing in the parlour.

Two alternative total mixed rations were formulated using the same criteria as described above:

Ration A	alfalfa haylage	13.0 kg
	38% protein supplement	0.5 kg
	dairy grain mix	11.0 kg
	alfalfa hay	5.5 kg
Ration B	alfalfa haylage	13.0 kg
	dairy grain mix	12.0 kg
	alfalfa hay	5.5 kg

Both rations would provide adequate dry matter intake, net energy and by-pass protein, but were slightly in excess for crude protein and fibre. The excess of protein came from the alfalfa hay and haylage and could not be decreased further without influencing the composition of the ration. Both rations were short in phosphorus and zinc and possibly also copper when considering the high level of sulphates in the drinking water. It was recommended to feed a mineral supplement high in phosphorus, zinc and trace minerals, at the rate of 60 g per cow per day.

These feeding regimes did not provide an excessive amount of concentrates and the forage

to concentrate DM ratio was higher than the recommended, minimum 40:60 ratio.[28] [31] [33] [34] [35] [36]

Feed some long-stem roughage ('effective fibre') to the lactating cows immediately after morning milking.

Salivation plays a major role in keeping rumen contents buffered within the optimal pH range of 6.0 to 6.8.[37] Perhaps the most important factor determining saliva production is the time spent by the animal chewing (cudding). The physical qualities of a feed will determine the ease of its ingestion and also any subsequent need to regurgitate and remasticate. The most important factors in this regard are particle size and moisture content.[37] More saliva, and more buffering capacity, will be produced by a cow eating long-stem hay than by a cow eating finely chopped silage, haylage or ground or pelleted grain. Palatability of the forage in the ration is another factor to consider.

A diet without added long fibre will cause unstable rumen function, increased production of lactate and a fall in rumen pH.[16] Feeding of long-stem roughage is incompatible with the concept of a complete diet, but not with a semi-total mixed ration. The provision of some long-stem hay, immediately after morning milking when appetite is greatest, may greatly improve the animal's capacity to maintain rumen pH.[30]

Although the chemically determined amount of crude fibre in the total diet of the lactating cows was adequate, the average particle length of 1 cm for the alfalfa haylage might have been too short to ensure sufficient rumination and saliva flow, resulting in a lowered pH-buffering capacity of the rumen. Furthermore, the high pH and tobacco-like smell made the haylage less palatable.

Leave the magnesium oxide, niacin and yea-sacc® out of the ration.

The mineral compound magnesium oxide is commonly classified as a buffer although its ability to stabilise rumen pH is limited and

dependent on particle size.[38] Magnesium also plays a major role in the synthesis of milk fat in the udder and supplemental magnesium is commonly fed to increase milk fat percentage of cows consuming fat-depressing rations.[39] Magnesium sources tend to be unpalatable to cattle and therefore may reduce dry matter intake.

Considering the questionable palatability of the haylage, it was more practical to buffer the diet with other compounds such as sodium bicarbonate which had already been incorporated in the ration anyway. Furthermore, there was no evidence of a fat test depression to support the addition of magnesium oxide to the diet.

Research has indicated benefit from niacin supplementation for alleviating and suppressing ketosis in dairy cattle[40], the greatest response occurring in cows with a condition score of 3 or higher.[41] However, it has also been reported that response to niacin may be negative in thin cows.[43] The results of trials determining the enhancing effects of niacin on milk production in early lactation are inconclusive.[42]

Ketosis was not considered to be a problem in the study herd and most of the early lactation cows had a condition score of less than 3.

Feeding of a dried culture of live yeast cells (Yea-sacc®) is believed to have beneficial effects on, amongst others, rumen development, feed intake, digestion and production. However, the results of trials are variable with many studies reporting little or no increase in production.

Do not include zinc methionine in the ration and do not replace the protein supplement for alfalfa pellets.

It is known that sulphur-containing amino acids contribute the sulphur bonds that give horn tissue the strength and resilience needed to minimize lameness. However, studies in which methionine hydroxy analogues were fed to improve

horn flexibility lack conclusive research proof.[43][44] Zinc deficiency has been implicated in lameness in dairy cattle.[45] However, studies on the effect of oral zinc treatment on the incidence of the interdigital lesions have been mixed.[46][47]

The total amount of zinc in the ration fed (forage, grain mix and mineral mix) was adequate.

The inclusion of lucerne (alfalfa) nuts in rations fed before and after calving to increase rumen buffering capacity has been recommended in the prevention of laminitis.[24][26] All the forage components in the ration fed were already of alfalfa origin (haylage and hay) and the addition of more alfalfa in the form of pellets would not have provided the desired effect.

Once a decision on the type of ration and its composition has been made, adhere to it (do not change it).

In cows fully adapted to a high-grain ration, the numbers of *Streptococcus bovis* in the rumen are about the same as in the hay-fed animal and the rumen ecosystem is well balanced.[48] Frequent and sudden changes in the feeding management (regime and ration composition) can cause extensive changes in the ruminal microbial culture and its products.[37] These changes can greatly influence the physiology, production and health of the rumen and the animal as a whole.

Adapt the heifers to concrete and using cubicles before they calve, preferably at breeding age and again in late pregnancy.

Lameness due to laminitis in heifers newly introduced to concrete is common[16][49][50], and has been attributed to traumatic damage caused by increased activity on concrete surfaces to which the heifers are unaccustomed.[18][49][51] The incidence of laminitis is greatly reduced when heifers are gradually introduced to concrete several weeks before calving.[13][16][49] Without previous experience of concrete, heifers at point

of calving will not adapt well to concrete floors.[24]

If not previously trained, heifers will be unfamiliar with cubicles, resulting in decreased resting time and hence increased risk of developing laminitis and lameness.[31]

Introduce pregnant heifers to the milking herd several weeks before calving.

Behaviourial interactions occur after introduction of new animals into an established dominance hierarchy.[52] Early introduction will give the heifers time and opportunity to establish their rank in the herd hierarchy. In this context, the effects of confrontation between submissive heifers and dominant cows need to be considered. Sudden turning and twisting movements, seen where heifers take evasive action to avoid tussling and bullying, may predispose to claw damage. Also, for low-ranking, cubicle trained cows, the cubicles act as both a resting area and a 'safety zone', where effective personal distance is increased by the partitions.[53]

Allow the lactating cows as much daily exercise as possible, preferably on a dirt lot or loafing area.

There is no objective evidence to support the proposal that a low level of exercise is an important factor in the aetiology of laminitis. However, it has been suggested that lack of exercise decreases the blood flow through the corium of the claw and makes the claw more susceptible to insult.[31][32]

Do not change to concrete-based cubicles, but make sure the earthen bases of the cubicles are not hollowed or lumpy.

It has been established that cows spend less time lying down on concrete cubicles without significant bedding.[16][54] Resting time in cubicles is markedly improved by the provision of a deep straw bed.[54][55] Furthermore, the dimensions of the present cubicles satisfy the

space requirements of the animals; current proposals for adequate cubicle dimensions for Holstein cows are 2.4 m long and 1.2 m wide.[56][57]

Replace the steel cable of the automatic manure scraper.

The width of the passageway between cubicles and feed bunk was only 2.5 m (the current recommendation being 3-3.5 m.)[57][58] Therefore, the cable was situated at the level of the hind limbs when cows were standing at the feed bunk. The damaged cable was most probably the cause of the cuts in the interdigital skin of the hind limbs in some of the cows. The injuries could have happened when the scraper was operated at the same time as cows were feeding.

Install a footbath at the exit of the parlour, so cows walk through it after each milking.

Footbaths are used to bring claws in contact with a disinfecting, astringent chemical.[11] Formalin and copper sulphate have been advocated to aid in the control of interdigital dermatitis and heel horn erosion.[59][60][61][62] Formalin (5%) looses its disinfection activity when the ambient (barn) temperature drops below 15°C.[63] However, formalin will adhere to the (clean) interdigital skin for about 30 min and rapidly acquire the temperature of the skin (20°-30°C), resulting in adequate disinfection.[62] Therefore, contact between the interdigital skin and manure or mud must be prevented for some time after bathing. Copper sulphate (10%) can be used the whole year around, but becomes rapidly ineffective when grossly contaminated with organic material.[30] Dry chemical footbaths, being a mixture of copper sulphate and lime, are useful in sub-zero temperatures.[11]

Horn quality becomes more important in animal production as confinement increases. The astringent action of the chemicals in footbaths will result in hardening of the claw horn, thereby improving quality and resistance to damage and disease.[64][65]

Let the cows walk through the medicated footbath 2 to 3 days each week. Use only water in the bath for the remaining days. After cows have walked through a medicated footbath, they should be kept in a holding area for 15 to 20 min, during which time the medication can have its desired effects. The footbath should measure 3 m long, 1 to 1.5 m wide and 20 cm deep, and be close-fenced to prevent cows attempting to jump over. Fill the bath to such a level that the coronary band and interdigital skin are well submerged, i.e approximately 10 cm.

Cows should have their claws trimmed twice a year.

The most important trimming is the one just after calving in order to correct any overgrowth which may have occurred during the dry period when cows were housed outdoors. Furthermore, corrective claw trimming to stimulate the growth of healthy horn has been advocated in the treatment of subclinical laminitis.[64]
The objectives for preventive claw trimming are:

a) correction of the relative overgrowth that leads to overburdening of the hind lateral claw,
b) restoration of a normal bearing surface within each claw, and
c) correction of developing claw lesions and defects at an early stage.[59][66]

It has been postulated that the lower incidence of lesions in claws with smaller measured traits (length of the dorsal border and heel height) indicates a potential benefit of regular trimming in the prevention of lameness.[67] Manson and Leaver[22][35] reported that trimming claws, to maintain a short dorsal border and steep angle, reduced the prevalence of lameness as well as duration of clinical cases.
Note. Another visit was made to the farm in the middle of December, 1989 to trim the claws of all in-calf heifers, dry cows and lactating cows and to teach the herdsmen the basics of preventive claw trimming.

Postscript

It was emphasised that solving or just reducing this lameness problem was going to take a long time. Improvements or changes would not be noticeable overnight. An improved nutrition and management programme had to be in place for quite some time (at least 6 months was suggested) before any conclusions of its success could be made. It was suggested that production as such was not always a good indicator and that things were improving when the newly introduced heifers were encountering less lameness problems. Also, a slight drop in production was likely to occur while cows were adapting to the changed diet.

All recommendations, except the change to a complete-diet system, were adopted or put in place. However, the timing of the hay feeding had been changed to immediately after milking. The manager of the dairy herd, who was contacted on a regular, monthly basis, reported dramatic improvements in the overall claw health of the milking cows while maintaining satisfactory production. The farm was visited again in August 1990 and by then the lameness incidence was reduced to less than 5%.

References

(1) Whitaker DA, Kelly JM, Smith EJ. Incidence of lameness in dairy cows. Veterinary Record 113, 60-2, 1983.
(2) Collick DW, Ward WR, Dobson H. Association between types of lameness and fertility. Veterinary Record 125, 103-6, 1989.
(3) Dijkhuizen AA. Economic aspects of diseases and disease control among dairy cattle. Unpublished PhD Thesis, Rijksuniversiteit Utrecht, 1983.
(4) Choquette-Lévy L, Baril J, Lévy M, St-Pierre H. A study of foot disease of dairy cattle in Quebec. Canadian Veterinary Journal 26, 278-81, 1985.
(5) Harris DJ, Hibburt CD, Anderson GA, Younis PJ, Fitspatrick DH, Dunn AC, Parsons IW, McBeath NR. The incidence, cost and factors associated with foot lameness in dairy

cattle in south-western Australia. Australian Veterinary Journal 65, 171-6, 1988.
(6) Moser EA, Divers TJ. Laminitis and decreased milk production in first-lactation cows improperly fed a dairy ration. Journal of the American Veterinary and Medical Association 190, 1575-6, 1987.
(7) Amstutz HE. Prevention and control of lameness in dairy cattle. In: Ferguson JC (ed). Veterinary Clinics of North America: Food Animal Practice 1, 25-37, 1985.
(8) Russell AM, Rowlands GJ, Shaw SR, Weaver AD. Survey of lameness in dairy cows. Veterinary Record 111, 155-60, 1982.
(9) Eddy RG, Scott CP. Some observations on the incidence of lameness in dairy cattle in Somerset. Veterinary Record 113, 140-4, 1980.
(10) Arkins S. Lameness in dairy cows. Irish Veterinary Journal 35, 135-40 & 163-70, 1981.
(11) Baggott DG, Russell AM. Lameness in cattle. British Veterinary Journal 137, 113-32, 1981.
(12) Greenough PR, Vermunt J, McKinnon JJ, Fathy FA, Berg PA, Cohen RDH. Laminitis-like changes in the claws of feedlot cattle. Canadian Veterinary Journal 31, 202-8, 1990.
(13) Greenough PR, Vermunt JJ. Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. Veterinary Record 128, 11-7, 1991.
(14) Peterse DJ. Lameness in cattle. Pp 1015-1023. Proceedings of the XIVth World Congress on Diseases in Cattle, Dublin, 1986.
(15) Bradley HK, Shannon D, Neilson DR. Subclinical laminitis in dairy heifers. Veterinary Record 125, 177-9, 1989.
(16) Bazeley K, Pinsent PJN. Preliminary observations on a series of outbreaks of acute laminitis in dairy cattle. Veterinary Record 115, 619-22, 1984.

(17) Vermunt JJ, Greenough PR. Predisposing factors of laminitis in cattle. Britsh Veterinary Journal (in press).
(18) Dewes HF. Some aspects of lameness in dairy herds. New Zealand Veterinary Journal 26, 147-8 & 157-8, 1978.
(19) Rowlands GJ, Russell AM, Williams LA. Effects of stage of lactation, month, age, origin and heart girth on lameness in dairy cattle. Veterinary Record 117, 576-80, 1983.
(20) McDaniel BT. Management and housing factors affecting feet and leg soundness in dairy cattle. Pp 41-49. Proceedings of the 16th Annual Convention of the American Association of Bovine Practitioners, Oklahoma City, 1983.
(21) Manson FJ, Leaver JD. The influence of concentrate amount on locomotion and clinical lameness in dairy cattle. Animal Production 47, 185-90, 1988.
(22) Manson FJ, Leaver JD. The influence of dietary protein intake and of hoof trimming on lameness in dairy cattle. Animal Production 47, 191-9, 1988.
(23) Chesterton RN, Pfeiffer DU, Morris RS, Tanner CM. Environmental and behaviourial factors affecting the prevalence of foot lameness in New Zealand dairy herds - a case-control study. New Zealand Veterinary Journal 37, 135-42, 1989.
(24) Weaver AD. The prevention of laminitis in dairy cattle. The Bovine Practitioner, 14, 70-2, 1979.
(25) Allenstein LC. Lameness in cattle. Canadian Veterinary Journal 22, 65-7, 1981.
(26) Pinsent PJN. The management and husbandry aspects of foot lameness in dairy cattle. The Bovine Practitioner 16, 61-4, 1981.
(27) Peterse DJ. Prevention of laminitis in Dutch dairy herds. Proceedings of the IVth International Symposium on Disorders of the Ruminant Digit, Paris, 1982.
(28) Greenough PR. The sub-clinical laminitis syndrome. The Bovine Practitioner, 20, 144-9, 1985.

(29) Weaver AD. Lameness in cattle - investigation and diagnostic check lists. British Veterinary Journal, 141, 27-33, 1985.
(30) Vermunt JJ. Lesions and structural characteristics of the claws of dairy heifers in two management systems. Unpublished MSc Thesis, University of Saskatchewan, Saskatoon, 1990.
(31) Blowey R. Diseases of the bovine digit. Part 2. Hoof care and factors influencing the incidence of lameness. In Practice 14, 113-24, 1992.
(32) Greenough PR. Observations on bovine laminitis. In Practice 12, 169-73, 1990.
(33) Livesey CT, Fleming FL. Nutritional influences on laminitis, sole ulcer and bruised sole in Friesian cows. Veterinary Record 114, 510-2, 1984.
(34) Owen JB. Complete-diet feeding of dairy cows. In: Haresign W, Cole DJA (eds). Recent developments in Ruminant Nutrition. Pp 312-24. Butterworths, Toronto, 1981.
(35) Manson FJ, Leaver JD. The effect of concentrate:silage ratio and hoof trimming on lameness in dairy cattle. Animal Production 49, 15-22, 1989.
(36) Anon. Lameness in dairy cattle. MAFF Publication PB 1150, London, 1992.
(37) Garry F, Kallfelz FA. Clinical aspects of dietary buffers for dairy cattle. Compendium on Continuing Education for the Practicing Veterinarian 5, S159-66, 1983.
(38) Schaefer DM, Wheeler LJ, Noller CH, Keyser RB, White JL. Neutralization of acid in the rumen by magnesium oxide and magnesium carbonate. Journal of Dairy Science 65, 732-9, 1982.
(39) Kincaid R. Macro elements for ruminants. In: Church DC (ed). The Ruminant Animal. Digestive Physiology and Nutrition. Pp 326-41. Prentice Hall, Englewood Cliffs, New Jersey, 1988.

(40) Fronk TJ, Schultz LH. Oral nicotinic acid as a treatment for ketosis. Journal of Dairy Science 62, 1804-7, 1979.
(41) Jaster EH, Hartnell GF, Hutjens MF. Feeding supplemental niacin for milk productions in six dairy herds. Journal of Dairy Science 66, 1046-51, 1983.
(42) Huber JT. Vitamins in ruminant nutrition. In: Church DC (ed). The Ruminant Animal. Digestive Physiology and Nutrition. Pp 313-25. Prentice Hall, Englewood Cliffs, New Jersey, 1988.
(43) Clark BL, Rakes AH. Effect of methionine hydroxy analog supplementation on dairy cattle hoof growth and composition. Journal of Dairy Science 65, 1493-1502, 1983.
(44) Randy HA, Sniffen CJ, Nocek JE, Wildman EE, Braund MW, William H. Effect of zinc methionine supplementation on milk yield, lameness and hoof growth in lactating dairy cows. Journal of Dairy Science 68, [Suppl 1], 277 (Abstr), 1985.
(45) Lamand EA. Zinc deficiency in ruminants. Irish Veterinary Journal 38, 40-7, 1984.
(46) Demertzis PN, Mills CF. Oral zinc therapy in the control of infectious pododermatitis in young bulls. Veterinary Record 93, 219-22, 1973.
(47) Egerton JR, Laing EA, Mulley RC. Failure of oral zinc therapy to alleviate *Bacteroides nodosus* infections in cattle. Australian veterinary Journal 62, 85-9, 1985.
(48) Hungate RE. The rumen microbial ecosystem. Annual Review of Ecological Systems 6, 39-66, 1975.
(49) David GP. Cattle behaviour and lameness. Pp 79-86. Proceedings of the Vth International Symposium on Disorders of the Ruminant Digit, Dublin, 1986.
(50) Weaver AD. Cattle foot problems. Part 2: Diseases of the horn and corium. Agri-Practice 9, 35-40, 1988.
(51) Vermunt JJ. 'Subclinical' laminitis in dairy cattle. New Zealand Veterinary Journal 40, 133-8, 1992.

(52) Fraser AF. Farm Animal Behaviour. Baillière Tindall, London, 1974.
(53) Potter MJ, Broom DM. Behaviour and welfare aspects of cattle lameness in relation to building design. Pp 80-4. Proceedings of the VIth International Symposium on Disorders of the Ruminant Digit, Liverpool, 1990.
(54) Colam-Ainsworth P, Lunn GA, Thomas RC, Eddy RG. Behaviour of cows in cubicles and its possible relationship with laminitis in replacement dairy heifers. Veterinary Record 125, 573-5, 1989.
(55) Cermák JP. Cow cubicle design. Farm Buildings Digest 18, 7-9, 1983.
(56) Hughes J. The cow and her cubicle. Pp 276-7. Proceedings of the VIth International Symposium on Disorders of the Ruminant Digit, Liverpool, 1990.
(57) Gwynn PEJ. Improvement of cow cubicle systems. Pp 24-8. Proceedings of the Autumn Meeting of the British Cattle Veterinary Association, Bristol, 1992.
(58) Potter MJ, Broom DM. The behaviour and welfare of cows in relation to cubicle house design. In: Wierenga HK, Peterse DJ (eds). Cattle Housing Systems, Lameness and Behaviour. Pp 159-65. Martinus Nijhoff Publishers, Boston, 1987.
(59) Toussaint Raven E. Lameness in cattle and footcare. Netherlands Journal of Veterinary Science 5, 105-11, 1973.
(60) Weaver AD. Lameness in cattle: the interdigital space. Veterinary Record 95, 115-20, 1974.
(61) Arkins S, Hannan J, Sherington J. Effects of formalin footbathing on foot diseases and claw quality in dairy cows. Veterinary Record 118, 580-3, 1986.
(62) Cornelisse JL, Peterse DJ, Toussaint Raven E. Formalin foot baths in the prevention of interdigital dermatitis. Tijdschrift voor Diergeneeskunde 107, 835-40, 1982.
(63) Cornelisse JL. Some observations on the disinfection of ruminant claws. Pp 137-8. Proceedings of the IIIrd International

Symposium on Disorders of the Ruminant Digit, Vienna, 1980.
(64) Greenough PR. Laminitis in review. Proceedings of the IVth International Symposium on Disorders of the Ruminant Digit, Paris, 1982.
(65) Greenough PR, MacCallum FJ, Weaver AD. Lameness in Cattle. J.B. Lippincott Company, Philadelphia, 1981.
(66) Toussaint Raven E. Cattle Footcare and Claw Trimming. Farming Press Ltd., Ipswich, 1985.
(67) Andersson L, Lundström K. The Influence of breed, age, bodyweight and season on digital diseases and hoof size in dairy cows. Journal of Veterinary Medicine A 28, 141-51, 1981.

EFFECT OF OVERCROWDED HOUSING CONDITIONS ON FOOT LESION DEVELOPMENT IN FIRST-CALVED FRIESIAN HEIFERS

F. Leonard, J. O'Connell and K. O'Farrell

Teagasc, Moorepark, Fermoy, Co. Cork, Ireland

Two studies were carried out to investigate the role of lying time in the development of foot lesions. In the first study, autumn-calving heifers were housed with one cubicle for every two animals from time of calving in September/October until February. To examine the effect of overcrowding on foot lesions in the absence of a calving effect, spring-calving heifers were housed at a 2:1 heifer-to-cubicle ratio from the end of October until they calved in January/February, after which they were housed with one cubicle each. Behavioural activities were observed every 15 minutes for five consecutive days and nights each month of the housing season. Claw health was recorded at housing and monthly thereafter.

A wide range of individual lying times were observed in the animals studied. The autumn-calving animals fell into one of three groups; those that lay for an average of 10, seven or five hours per 24 hours. Peak claw lesions were observed at four months after calving and claw health was significantly worse ($P<0.001$) in animals that lay for five hours (median haemorrhage score = 31) than in animals that lay for 10 hours (median score = 8). Lying behaviour and claw health at four months post-calving were significantly negatively correlated ($r_s = -0.54$, $P<0.001$).

Spring-calving heifers with high total haemorrhage scores (>50 over five examinations) stood in cubicles for longer ($P=0.05$) than heifers with low haemorrhage scores (<25 over five examinations). Lying behaviour was not significantly different between the two groups but animals with low haemorrhage scores tended to lie longer. There was a correlation of $r_s = 0.45$, $P<0.01$, between standing time in cubicles and total foot lesion score. No correlation between lying behaviour and claw health was detected.

These studies indicate that changes in behavioural activities are important in the development of foot lesions in cattle but other

factors acting around time of calving are also of significance.

THE ROLE OF STOCKMANSHIP IN FOOT LAMENESS IN UK DAIRY CATTLE

W. Robert Ward

Liverpool University
Leahurst, Neston, S Wirral, L64 7TE, U.K.

A major study in UK dairy cattle identified several risk factors in foot lameness (Clarkson et al, 1993), but it was clear that other factors were also important. Clackson & Ward (1991) showed that farmers who allowed their cows to walk in single file had less lameness in the summer--one aspect of stockmanship.

On 14 farms in North-West England, cows were locomotion scored (Manson and Leaver, 1988) once or twice in summer (Mill & Ward, 1994). A score of three or more on the five-point scale indicates that the cow is lame, and the proportion of lame cows is a measure of prevalence. One locomotion score in midsummer correlates with the mean of several scores (Clarkson et al, 1993).

On each farm, the farmer was defined as the person who normally decided whether to treat a lame cow or call for veterinary attention, and might be the owner or an employee. Farmers were asked what forms of foot lameness they saw in their herd, then shown pictures of foot lesions, and scored on their knowledge. The more knowledgeable farmers had a lower prevalence of lameness in their herd ($P<0.001$).

The farmers' training relevant to foot lameness was scored, from a one-day course in claw trimming to a degree in agriculture. Farmers with more training had a lower prevalence of lameness in their herd ($P<0.001$).

Farmers' awareness of lameness was assessed by asking them how many lame cows they had. This figure was compared with the number with locomotion score three or more on the same day. No farmer over-estimated the number of lame cows: those with fewest lame cows knew how many there were. Those most aware of lame cows had the lowest prevalence of lameness in their herd ($P<0.001$).

A second study (Diamond, Ward & Murray, unpublished) of 14 dairy farms in North-West England,

including seven studied by Mill & Ward (1994) produced similar results.

Stockmanship is a very important factor in the prevalence of summer lameness in UK dairy herds. If veterinarians and others can help farmers improve their knowledge, training and awareness of lameness, then the prevalence should be reduced.

References:

Clackson, DA, Ward, WR (1991) Farm tracks, stockman's herding and lameness in dairy cattle. Veterinary Record, **129**, 511-512

Clarkson, MJ, Downham, DY, Faull, WB, Hughes, JW, Manson, FJ, Merritt, JB, Murray, RD, Russell, WB, Sutherst, JE, Ward, WR (1993) An epidemiological study to determine the risk factors of lameness in dairy cows. (Ref; CSA 1379). Final Report

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

Mill, JM, Ward WR (1994) Lameness in dairy cows and farmers' knowledge, training and awareness. Veterinary Record, **134**, 162-164

THE MEASUREMENT OF BEHAVIOUR OF UK DAIRY CATTLE BY 24-HOUR OBSERVATIONS

W. Robert Ward

Liverpool University
Leahurst, Neston, S Wirral, L64 7TE, U.K.

In a study of lameness in UK dairy herds, most cubicles were found to be too small (Clarkson et al, 1993). We therefore watched cows for 24-hour periods, noting every 15 minutes which were lying down, standing, etc., in cubicles, straw yards and at pasture.

In farm 1, with silage fed in front of the cubicles, mean daily lying time early in the housing period varied from 6.25 hours in heifers not previously trained to use cubicles to 8.39 hours in mature cows (Singh et al 1993a). Heifers that lay down less developed more foot lesions. Later in the housing period heifers lay for longer. At pasture the heifers lay for 9.35 hours, and mature cows for 10.2 hours: they spent a long time travelling to and from the milking parlour, and in the dispersal area.

On farm 2, cows in a straw yard lay for 9.6 hours, while cows in cubicles lay for only 6.8 hours (Singh et al, 1993b).

On farm 3, large Holstein cows with a history of lameness when in cubicles had very little lameness when transferred to straw yards. They lay for 10.8 hours in 24. Lying time was reduced when some cows were in oestrus (Singh et al, 1994).

On farm 4, large cubicles (2.4 x 1.2 metres) with low kerbs (120mm) and brisket boards were installed (Bowling et al unpublished). Cows lay for 12 hours in 24.

On farm 5, with similar large cubicles and soft rubber mats, cows lay for 12 hours, despite more cows than cubicles.

Cows provided with straw yards or large well-designed cubicles lie down for longer than cows in small cubicles, and are less likely to become lame.

Naturally - all cows like cows @ same time
- 1 ... - 5 ... cows is most popular lying time

References:

Clackson, DA, Ward, WR (1991) Farm tracks, stockman's herding and lameness in dairy cattle. Veterinary Record, **129**, 511-512

Clarkson, MJ, Downham, DY, Faull, WB, Hughes, JW, Manson, FJ, Merritt, JB, Murray, RD, Russell, WB, Sutherst, JE, Ward, WR (1993) An epidemiological study to determine the risk factors of lameness in dairy cows. (Ref; CSA 1379). Final Report

Singh, SS, Ward WR, Lautenbach, K, Hughes, JW, Murray, RD (1993a) Behaviour of first lactation and adult dairy cows while housed and at pasture and its relationship with sole lesions. Veterinary Record. **133**, 469-474

Singh, SS, Ward, WR, Lautenbach, K, Murray RD (1993b) Behaviour of lame and normal dairy cows in cubicles and on a straw yard. Veterinary Record. **133**, 204-208

Singh, SS, Ward, WR, Hughes, JW, Lautenbach, K, Murray RD (1994) Behaviour of dairy cows in a straw yard in relation to lameness. Veterinary Record. (In Press)

oestrus → ↓ lying time for all cows
→ ? ↑ lameness in early lact?
? early lact → eat more → lie less?

Straw yds lying + ruminating 8.7
" early 4.1
standing + ruminating 2.7
" + feeding 5.4
" early 6.1

BIOMECHANICS OF DAIRY COWS; EFFECTS OF HOUSING AND GRAZING

A. H. Herlin
Swedish University of Agricultural Sciences, Dept. of Animal Nutrition and Management, Animal Research Station, P O Box 59, S - 23053 Alnarp, Sweden.

The long-term influence of management systems on the locomotion of seventeen dairy cows was investigated by high speed cinematography (100 frames/s) and kinematic analysis. The cows had been subjected to the following management systems: Tie-stalls and kept indoors all the year round (TI), cubicles and indoors all the year round (CI), tie-stalls and grazing in summer (TG) and cubicles and grazing in summer (CG). At the recording, cows had been kept in the management systems for about two and a half year. TG and CG cows were recorded at the end of the grazing season. Temporal variables were determined from the film. Angular patterns and hoof trajectories of the left fore and hind limbs are presented and statistics made of occurring minimum and maximum angles.
A smaller maximum angle of the elbow of CI cows compared to TI, TG and CG cows. The hock joint was less flexed during the stance in CI cows compared to the TI and CG cows. The minimum angle during the swing phase of the hock joint was smaller of TG and CG cows compared to TI and CI cows. Pastured cows had a less thorough bending of the fetlock joint angle during the stance than cows kept indoors. In the hind fetlock joint curve, a local maximum was observed shortly after the hoof was observed flat on the ground. This Max_1 was greater in the CI cows than in the TG and CG cows.
It is suggested that kinematic methods are applicable to evaluate floor environment and management relative to hoof health and locomotion of cattle.

Does the claw trimming procedure affect milk yield and milk quality factors ?

Ch. Stanek, M.-M. Thonhauser, G. Schoder
Clinic of Orthopedics in Ungulates, University of Veterinary Medicine, Vienna, Austria

Summary

The influence of the claw trimming procedure on milk quality factors was determined in 45 dairy cows of one farm. Daily milk yield, somatic cell count, bacteriological examination and clinical udder status was evaluated. After a preliminary phase of 7 - 10 days, the claw trimming was carried out on 4 subsequent days, thereafter the parameter were recorded over further 22 days. A significant decrease of milk yield of 10 % was observed, whereas the increase of somatic cell count was not significant. A positive bacteriological finding was obtained in milk samples in the period after the claw trimming procedure in 12 cows. One cow exhibited an acute catarrhalic mastitis. Part of the milk depression was also caused by the disturbance in the stable and not only by the claw trimming procedure itself.

Introduction

Claw disease is next to sterility and udder disease the primary cause for early culling of dairy cattle. Regular claw care is an important factor for healthy limb management. Claw care is not only important from an economic and veterinary standpoint but also from the point of animal welfare. A great number of publications have dealt with the importance of claw trimming for prophylaxis and therapy of various diseases, situated

mainly in the digital region in cattle. A large number of papers also refer to the technical management of claw trimming. Although the available literature gave no informations on the effect of the claw trimming procedure itself, the fixation in a claw trimming crush and the disturbance of the animal, on udder status and milk quality factors. Connections between the digital status, the claw trimming procedure and the udder status were evaluated in 45 dairy cows of one herd. In a time span from one week before and three weeks after the claw trimming procedure the parameters "milk yield", "somatic cell count" and "bacteriological status of the udder" were recorded (THONHAUSER et al., 1994).

Literature

The terminus "digital diseases" is a very comprehensive, but not a precise one. Claw horn overgrowth in many cases is the first step for more complicated digital diseases. . Economic consequences are considerable. Even in 1960, in Austria, economic losses caused by neglected claw trimming were estimated at 40 Mio $ (KNEZEVIC, 1960). This amount has to underly an upward correction, if lameness as a whole is registered (WEAVER, 1983). A herd affected with claw problems can exhibit a reduction in overall productivity of 20 %, the profitability is not assured anymore (GREENOUGH et al., 1981). In Great Britain yearly losses due to claw problems are estimated as high as 36 Mio £ (RUSSEL et al., 1982). When comparing cattle kept in housing systems and on pasture, the same digital diseases with different focal points can be noted. Many claw diseases which developed during the housing period become apparent to the farmer during the first weeks of the

pasture period (STÖBER, 1983).

The importance of claw trimming and guidelines for the practical approach are covered in many publications. Claw trimming should be carried out at least two times a year in dairy cattle; in AI bulls and in animals with claw problems at least 3 to 4 times a year.

Milk ejection is influenced by massage, suction and milking stimuli, acustical and other stimuli are also involved (WENDT et al., 1986). The influence of different factors on milk ejection and secretion has been studied. Only a few papers, dealing with a certain situation of disturbance, can be mentioned in this short survey. Depending on the time of omitted milking, reduction of milk yield is reported with up to 80 %, with an increase of the somatic cell count up to 100 to 200 times. Both parameters reach after 1 to 2 weeks their original level (SCHODER, 1991). Very individual reactions due to stress, changing of the housing conditions, transport or pasturing are reported (KÖFER, 1976, HAMANN u. REICHMUTH, 1990). Claw trimming represents, without a doubt, a severe disturbance in a dairy herd, even though we could not find appropriate literature on influence of milk quality factors.

Animals, Material and Method

Investigations were carried out in November 1992 in 45 dairy cows of the experimental herd of the University of Veterinary Medicine Vienna (26 Braunvieh Brown Swiss , 7 Fleckvieh
Simmenthal , 7 Pinzgauer, 3 Holstein Friesian, mean age 3.5 yrs., 32 cows in first lactation). Animals with severe orthopedic and other diseases other than claw horn overgrowth

were excluded. The last claw trimming was performed about 6 months prior to the examination. The animals were kept on a tied housing system during the entire year. Average milking yield measured 5077 kg / year, no further experiments, especially no painful manipulations were carried out during the recording period.

37 cows were in the last third, 7 in the middle and one in the first third of lactation. Care was taken not to perform the claw trimming procedure in the last months of gravidity. From the economical point of view the whole herd had be trimmed within a few days, which corresponds to practical circumstances.

The claw trimming procedure was carried out in the 45 animals on 4 consecutive days by different claw trimming technicians according to the "farriers method". 12 animals were trimmed on day 1, 8 cows on day 2, 13 on day 3 and 12 on day 4. Claw trimming was carried out in the stable, the cows were leaded from their place to the claw trimming stand (KENEZEVIC 1962). Two diagonal limbs were treated simultaneously. The front limbs were fixed on a wooden block, while the hind limbs are lifted with a leather sling and trimmed in this position. The trimming began with the cleaning of the claws. Then the major part of the sole horn was removed using a paring knife on the front limb. On the hind limbs this step was carried out with a drawing knife. Then the distal end of the wall was shortened using snips, starting palmar/plantar. In a next step the bulbs and the sole including the axial excavation were trimmed precisely using the drawing knife. The trimming of the dew claws was the last step. Both limbs were downlifted, thereafter the other diagonal limb pair could

be trimmed in the same manner. Judgment of the types of claw horn overgrowth was performed by the second author in every case. The grade deviations were evaluated according to the amount of exuberant horn: low degree deformations representing an average of horn overgrowth of 2 months (1 - 1.5 cm), medium degree representing an average of horn overgrowth of 4 months, and high degree of 6 months and more (KNEZEVIC, 1960). Animals with an obvious lameness were not included in this study.

The study was divided in a preliminary stage (Evaluation of zero probes), the day of claw trimming and the post-trial stage (Evaluation of the effects of claw trimming on milk yield and udder status). Depending on the exact day of correction, the total recording time was between 30 and 33 days. Due to internal reasons, the claw trimming had to be done on 4 subsequent days before noon. For the statistical evaluation the day of claw trimming was designated as day 8, explaining the different length of the preliminary stage (7 - 10 days), herewith also the different length of the total recording time. The post-trial stage had a duration of 22 subsequent days. The influence of the disturbances in the stable itself, caused by the claw trimming procedure carried out in fellow cattle,was analyzed as a separate factor.

Milking was performed twice a day, between 4 and 8 a.m. and in the evening between 4 and 7 p.m., using a Duovac - milking system[1]. The individual milk yield was evaluated using a

a. ALFA LAVAL, Vertretung Wien

Waikatosystem. Milking times were not influenced by the claw trimming performed during the morning. During the entire duration of the study milk samples were taken from the p.m. milking for determination of indirect and direct cell count and milk yield. For the California Mastitis Test the first milliliters from milking were used.The next ten milliliters were put into glass tubes for the measurement of the direct cell count using the Fossomatic Cell counter. Every 3 days sterile milk samples were taken for bacteriological evalution, after this, samples for the California Mastitis Test and cell count were harvested. The milk samples were cultured on sheepbloodagar at 37°C for 24 hours. All examinations carried out are outlined in table 1.

phase / day	claw status	milk yield	CMT	cell count	bacteriol.exam.
preliminary phase day (-2) 1 - 7		daily	daily	daily	day(- 2), 1, 4, 7
claw trimming day 8	evaluated	daily	daily	daily	bacteriol. exam.
post trial stage after claw trimming. day 9 - 30		daily	daily	daily	bacteriol. exam. day 9, 12, 15, 18, 21, 24, 27, 30

Table 1: chronological sequence of the examination (CMT = California Mastitis Test, cell count = somatic cell count; bacteriol. exam. = bacteriological examination)

Results

A medium degree of claw horn overgrowth was the most common

observed deformity. High grade claw horn overgrowth was more often observed on the hind limb (Table 2). Six of the cows showed low grade claw horn overgrowth on all 4 limbs, 8 showed high grade overgrowth on all 4 limbs. All the other cows (n=31) showed middle grade overgrowth.

grade/ extremity	low	medium	high
front limbs, animals n	8	29	8
%	*17,8*	*64,4*	*17,8*
hind limbs, animals n	7	25	13
%	*15,5*	*55,6*	*28,9*

Table 2: Occurrence of claw horn overgrowth in 45 cows (scheme by KNEZEVIC, 1960)

In 29 cows pathological changes, like double sole formation, white line separations or interdigital hyperplasia were observed mainly in hind limbs during the claw trimming procedure. In 9 cows the horn of the sole was perforated during the claw trimming at one localisation, mainly due to pathological changes and their correction. This condition was not taken into further consideration.

When comparing the daily milk yield between the preliminary and post-trial stage, a significant reduction of the milk yield, beginning with the day of claw trimming was observed. In the Wilcoxon-test almost without any exception a significant , and in most cases a highly significant reduction in daily milk yield lasting from the last day of the preliminary stage through the entire study could be documented. As there was no normal distribution, the median values of the singular day values are presented in figure 1. As day of the claw trimming procedure, day 8 is accepted. The milk yield

reduction was seen in cows with average milk yield as well as in cows with an above average milk yield (early lactation period) . The highest reduction was seen on the day of claw trimming. During the following days the milk yield increased gradually but did not reach the original status. A further decline in milk yield was registered on approximately day 20 of the study.

Furthermore, four groups according to the definite day of claw trimming are formed. The decrease of milk yield in animals corrected on the first day of the claw trimming period was comparably low. In those groups trimmed in the following days, the decrease was higher. A certain decrease was also observable in the phase of disturbance prior to trimming of these animals.

Examining relationships between degree of hoof horn overgrowth and milk yield, animals with middle grade overgrowth were situated at a lower level than the group with low grade overgrowth and high grade overgrowth. The differences between groups could not be statistically verified. Cows with high grade overgrowth already showed in the preliminary stage an average milk yield reduction of 1 kg.

In contrast to the daily milk yield, the direct somatic cell count showed very high indiviual variations, but remaining on the same level overall. The direct somatic cell count showed the highest value on the day of the claw trimming, but no statistical significance was found.

The bacteriological examination of the sterile milk samples showed the following results: a negative result was obtained in 14 cows over the whole period of the investigation, 12 cows had a negative result in the preliminary period but showed on the day of claw trimming or at least at some time during the investigation period udder pathogenic bacteria i.e. hemolysing streptococci and *Staphylococcus aureus* in one quarter. 19 cows showed positive results before as well as after the claw trimming procedure. No

statistical verified connections could be found between bacteriological results and the grade of hoof horn. During the entire period of the examination none of the cows, with one exception, showed any clinical changes on the udder. Cow #45 exhibited on the third day after the claw trimming an increase of the somatic cell count from under 200,000 to over 1 million on day 7. On day 5 clinical symptoms of an acute catarrhalic mastitis became apparent. The cow was treated, bacteriological examination was negative during the entire time.

Discussion

The value of periodical orthopedic claw trimming performed by skilled technicians is very high, is stated by many authors and is not questioned by this publication. Carrying out this procedure certainly has an influence on the daily routine of the animal. The patient is fixated in standing or lying position and the claw trimming is performed in a time period of up to 30 minutes. This can be done manually - as in this study - or using angle grinders oder hydraulic pliers. A thorough and professional instruction of the technicians is absolutely necessary due to management and animal welfare (KNEZEVIC, 1960, STANEK, 1980).

Reaction of animals on stress varies considerably and adaption ability decreases with higher production rates. Partial drying over a period of some days induces a severe decrease in milk yield (up to 80 %) of the original yield and an increase in somatic cell count (SCHODER, 1991). Trials inducing transport and housing stresses revealed a very individual reaction in the elevation of somatic cell count and the decrease of milk yield (KÖFER, 1976). Accompanying measures are as important as the claw trimming procedure itself. Disturbances in the stable induce a milk yield decrease comparable to the claw trimming procedure. Reduction of milk yield was within an level as observed in cattle exposed to repeated noise (KRAKOSEVIC 1970, HEAD et al., 1993).

The movement of animals, the presence of persons not familiar to the cow, optical and acoustical phenomena should be taken into consideration. Cows, who underwent claw trimming on the last day, already showed milk yield decretion prior to claw trimming. A statistical significant milk decrerase of 1.2 kg/day was observed on the day of the claw trimming. It should be considered that the cows were in the last third of lactation during this study.

Choosing the right time for the semiannual claw trimming is important. The risk for the cow and the unborn calf is rather high a few weeks before parturition, the last third of the lactation is a more appropriate time. This means, although, that the next trimming would be near the highest point of the lactation. A correlation between the degree of claw horn overgrowth and decrease of milk production was not observed. This observance should not lead to neglection of milk losses caused by forgotten claw care.

Results indicating a negative correlation between milk yield and somatic cell count could not be verified in this study (KÖFER, 1976, SCHODER, 1991). No statistical significant increase of somatic cell count after claw trimming could be found, even though strong individual variations were observed.

As a whole, claw trimming does not cause as great a problem as the above mentioned situations. The mechanical insultation induced by the stock, the fixation mechanisms and injuries resulting from the cow trying to free itself obviously are of minor importance. These facts should be considered when designing claw trimming stocks. The influence on the udder status should not be neglected. This refers to the occurrence of udder pathogenic bacteria and acute mastitis. Herd control should focus on these criteria.

Literature

GREENOUGH, P., MAC CALLUM, F. J., WEAVER, A. D. (1981): Lameness in cattle. 2nd ed., Wright Scientechnica, Bristol, S. 105 - 112.

HAMANN, J., REICHMUTH, J. (1990): Exogene Einflüsse auf den Zellgehalt der Milch unter Berücksichtigung des Gesundheitszustandes der Milchdrüse. Milchwiss. **45,** 286 - 290.

HEAD, H. H., KULL, R. C., CAMPOS, M. S., BACHMAN, K. C., WILCOX, C. J., CLINE, L. L., HAYEN, M. J. (1993): Milk yield, milk composition and behaviour of Holstein cows in response to aircraft noise before milking. J. Dairy Sci. **76,** 1558 - 1567.

KNEZEVIC, P. F. (1960): Die Klauenpflege beim Rind. Wien. tierärztl. Mschr. **47,** 240 - 250.

KNEZEVIC, P. F. (1962): Ein transportabler Zwangstand und ein neues Schleifgerät zur Klauenkorrektur für Rinder. Wien. tierärztl. Mschr. **49,** 370 - 377.

KÖFER, J. (1976): Der Einfluß verschiedener Belastungen auf den Zellgehalt der Milch. Diss. Vet. Med. Univ. Wien.

KRAKOSEVIC, N. D. (1970): (Preventing industrial noise on farm animals) Veterinarija, Moskva, **47,** 33.

RUSSEL, A. M., ROWLANDS, G. J., SHAW, S. R., WEAVER, A. D. (1982): Survey of lameness in British dairy cattle. Vet. Rec. **111,** 155 - 165.

SCHODER, G. (1991): Der Einfluß von Stressoren auf den Zellgehalt der Milch. Diss. Vet. Med. Univ. Wien.

STANEK, Ch. (1980): Beitrag zur maschinellen Klauenkorrektur mittels Winkelschleifer. Wien. tierärztl. Mschr. **67,** 138 - 145.

STÖBER, M. (1983): Klauenkrankheiten des Weiderindes. In: KNEZEVIC, P.F. (Hrsg.): Orthopädie bei Huf- und Klauentieren. Schlüter, Hannover, S. 91 - 92.

THONHAUSER, M.-M., STANEK, Ch., SCHODER, G. (1994): Zur Beeinflussung von Milchmenge, Zellzahl, und bakteriologischem Befund der Milchproben durch eine nach der "Schmiedemethode" durchgeführte Klauenkorrektur beim Milchrind. Wien. Tierärztl. Mschr. **81,** 66 - 72.

WEAVER, A:D. (1983): Wirtschaftliche Bedeutung von Klauenkrankheiten beim Rind. In: KNEZEVIC, P.F. (Hrsg.): Orthopädie bei Huf- und Klauentieren. Schlüter, Hannover, S. 100 - 104.
WENDT, K., MIELKE, H., FUCHS, H. W. (1986): Euterkrankheiten. Fischer, Jena, S. 13 - 288.

Adress of authors: Univ. Doz. Dr. Ch. Stanek, Linke Bahng. 11, A 1030 Wien, AUSTRIA
Tel. Austria (1) 71155/372 FAX Austria (1) 714 91 06

SOME HOUSING AND MANAGEMENT CONSIDERATIONS RELEVANT TO DAIRY COW WELFARE AND STRESS RELATED LAMENESS

Jan Čermák
ADAS Guildford, 98 Epsom Road, Guildford UK GU1 2LD

Many veterinary practices which specialise in large animal work provide services mainly concerned with the treatment of reproductive metabolic disorders. There is however much interest among farmers on the practical aspects of dealing with housing related lameness and upper leg injuries which, in some dairy herds, can cause significant economic losses.

This contribution aims to highlight some of the background information which is relevant to the broader aspects of welfare and lameness of housed dairy cows. Many farmers are interested to hear how they could improve the overall standard of health in their herds. It seems that housing related health problems often attract much less attention than husbandry topics and that there appears some scarcity of proven guidance on the subject.

Veterinarians are well placed to advise farmers on what can be done to reduce the underlying level of aggression among cows, enhance animal comfort, reduce incidental injuries and respiratory problems and how to alleviate food and upper leg lameness.

While it would be misleading to introduce some anthropomorphic sentiments we can safely postulate that by housing and managing animals man has taken on the responsibility of the animals' well being. One possible approach is

to try to ensure that animals in our care are free from fear, hunger, thirst, adverse climatic environmental conditions and that they are free to manifest their natural behaviour.

On many farms cow housing and built environment fails to provide the conditions which would ensure that the above aspects can be properly satisfied. Subsequently animals suffer various reproductive and metabolic disorders, injuries, display aggression and bullying and deviant rising movement. In some housed herds cows even tend to alter their feeding and resting behavioural patterns.

Apart from the moral standpoint of perceived exploitation of animals sometimes there can be substantial economic penalties in terms of loss of milk yield, body weight, excessive culling rates or missed oestrus and delayed calving periods. Handling of animals suffering from acute forms of lameness can also delay management routines especially collecting animals for milking.

Looking ahead it is likely that economic pressures on producers will firm up as milk quotas and production subsidies are being questioned both in North America and in Western Europe. Farmers will need to concentrate more on the cost of production rather than just on the need to meet their milk quota allocations or other output indicators in order to achieve the expected profit margins.

Animal welfare - target parameters.

From the zoocentric angle the key factors which influent the welfare of animals include:

- the extent of space that is available for animals to move around and to lie down.
- the provision of a dry resilient lying area.
- non-skid floor surfaces.
- the absence of prolonged exposure of the hoof horn to slurry and wet underfoot.
- the design of mangers which can truly accommodate anatomy and morphology of the cow's body, thus easing the animal's reach for feed.
- climatic environmental conditions appropriate for large ruminant animals.

Lameness, in its acute form, certainly causes much pain to an animal and since it is such a multifactorial disorder its prevention requires a joint effort on the part of farmers, nutritionists, geneticists, building designers and veterinary practitioners. This paper examines some aspects of the design of resting and feeding areas and points out some principals which could help to reduce the incidence of lameness on dairy units.

Cubicle housing and organisation of space
Circulation Areas

Cubicles (free stalls) have become a dominant housing system for dairy cows in Britain and in most large scale dairy units in Northern America. Cows are allowed to move within buildings freely, circulating between their resting, feeding and milking areas.

The extent of space provided by building designers needs to accommodate the cow's requirements for turning around, passing each other or other animals which are eating or drinking. The observation of cows' interactive behaviour indicates that they respect "personal space", the extent of which is defined with the flight distance exhibited during aggressive encounters between cows of varying social hierarchy.

There is also the space which a cow needs in order the lie down and to rise correctly. The extent of this space could be referred to as the cow's own "space envelope". Those cubicle designs which do not respect this "space envelope" can case injuries and damage to animals especially damage to the rib cage, knees, the nape part of the neck and foot ligaments, as the cow struggles to get up, often undertaking "press ups" rather than a fluent forward swing to accomplish the rising movement.

Potter and Broom (1) studied the use of space in a cubicle unit and concluded that the space is used very competitively, particularly around clearways between rows of cubicles, drinking troughs, milking collecting yards and entries and exits to other parts of the

building. The underlying level of aggressive social encounters rises as animals are confined to a limited space and are thus forced to invade each others "personal space". This can lead to sudden avoidance actions and rapid movements causing animals to slip and even fall on slurry-covered surfaces. This can lead to upper leg lameness.

The width of the circulation space can be conveniently defined as multiples of approximately one half of the cow's body length. The overall shape of available space is normally governed by linear layouts, initially conceived to accommodate the movement of tractors, forage wagons and slurry scraping machinery. These, of course, are not animal centred solutions and thus it is important to ensure that adequate space for the animal is provided, especially around the "strategic sites" mentioned above.

Resting areas - cubicle system

The advent of the cubicle system of housing dairy cows has lead to an increase in labour productivity and to savings in the use of bedding materials. From the animal's standpoint, however, there is an increased environmental challenge to their welfare and well being. For example, prolonged exposure of the hoof horn to slurry on mostly slippery concrete surfaces leads to the softening of hooves and subsequently to an increased rate of abrasion of load bearing hoof wall and subsequent problems.

Widespread adoption of the cubicle system led to the proliferation of the designs of cubicle partitions. There are many types on the