Field Investigations of Laminitis-Problem Dairy Herds

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ABSTRACT

Field experience suggests that the two most common causes of laminitis in Wisconsin dairy herds are chronic, subacute rumen acidosis and excessive time standing on concrete. Diagnosis of subacute rumen acidosis is based upon clinical signs of the herd, needle aspiration of rumen fluid for pH determination, and ration analysis. In the author's experience, most acidosis problems result from a failure to adapt the dry cow to lactation rations, unrealistic dry matter intake (DMI) expectations in the peri-parturient period, and an overestimation of forage DMI in total mixed rations. Laminitis can result from excessive time standing on concrete surfaces. Poorly designed stalls can reduce resting time, thereby increasing the time cows spend standing. Stalls can be evaluated for surface comfort, adequate resting space based upon body size, availability of lunge room for rising, and proper positioning of neck rails to allow completion of the rising motion.

INTRODUCTION

Undifferentiated lameness is quite prevalent in our industry. A recent survey in Minnesota and Wisconsin indicated a prevalence of clinical lameness of approximately 15%, with a range from 0 to 33% (Wells et al., 1993). In this study, the actual prevalence was 2.5 times higher than the herd managers had estimated. Many dairy managers, veterinarians, and nutritionists share a tendency to underestimate, or perhaps tolerate, an abnormal prevalence of lameness in dairy herds and overlook its importance as a diagnostic sign of herd management problems. In reviewing my records of chronic acidosis herd investigations where lameness prevalence was critical to the diagnosis, less than 25% mentioned lameness as a herd problem in the referral request.

These same experiences indicate that when laminitis problem herds are investigated, it is common to find prior diagnostic work has been very narrowly focused. Often the sole diagnostic effort has been an analysis of the high lactation group ration, when the evaluation should include additional factors and age groups.

This paper will discuss field investigations of dairy herd problems where laminitis is a primary clinical sign. It will focus upon two causes of laminitis, rumen acidosis and excess standing time on concrete. The discussion will identify common management practices that create these problems.

CHARACTERISTICS OF LAMINITIS PROBLEM HERDS

Laminitis in cattle can be presented in four stages: acute, subacute, subclinical, and chronic (Weaver, 1988). It is unusual to find acute cases of laminitis in herd investigations, but "paintbrush" hemorrhages of the sole, characteristic of subacute laminitis, are occasionally found. Signs of chronic laminitis (Weaver, 1988) are common and include a reduction of the normal dorsal foot angle of about 55° to 35-45°, a concave dorsal wall surface, and horizontal lines continuing around the entire wall. The abaxial white line becomes widened and sometimes separates, leaving an opening for bacteria and foreign objects. Sometimes the tissue above the coronary band appears to be swollen. The laminitis problem herd usually reports problems with sole ulcers and abscesses, which are common sequelae to laminitis.

Hoblet (1993) characterizes a laminitis-problem herd as having greater than 5% of the herd showing lameness (not associated with infectious foot rot) within a year, more than 50% of all lameness occurs within 50 days of calving, or more than 25% of any lactation group showing signs of sole hemorrhages. In the author's opinion, an annual incidence of less than 5% is rarely achieved and may be optimistic.

In any herd investigation of laminitis, it is important to determine the herd management group in which the laminitis problem begins. If lactating heifers show signs of laminitis, the pregnant heifers need to be evaluated to see if the problem started there. If the pregnant heifers have laminitis, the investigation should include the growing heifers. The youngest age group with problems must be identified.

RUMEN ACIDOSIS AND LAMINITIS

Rumen acidosis is widely recognized as a cause of laminitis, although the physiological mechanism is not completely understood (Greenough and Vermunt, 1991). Research efforts focus upon histamine and endotoxin production (Peterse, 1985), which can produce laminitis.

Rumen acidosis can occur as the proportion of readily-fermentable carbohydrates, particularly starch, in the total ration increases. Amylolytic bacteria produce the normal end products including the volatile fatty acids (VFA). VFA's are absorbed through the ruminal mucosa. If VFA production exceeds absorption, rumen pH starts to decrease. A modest decrease in pH is necessary to begin the adaptation process and shift rumen microbial populations to groups appropriate for high concentrate rations. It is important that the pH drop is limited, as fiber-degrading, cellulolytic bacteria and protozoa populations decline dramatically if the rumen pH falls below 6.0 (Leedle, 1991). If rumen pH drops further toward 5.5, *Streptococcus bovis* has a competitive advantage and produces lactic acid rather than VFA's (Russell and Hino, 1985). Lactic acid utilizing bacteria do not function well at lower rumen pH values and cease altogether if pH reaches 5.2 . As lactic acid accumulates, the rumen becomes dominated by *Lactobacillus* sp. which produce lactic acid exclusively (Leedle, 1991). Rumen acidosis with all its clinical manifestations may result.

Diagnosis of Subacute Rumen Acidosis. It is uncommon for herds with substantial laminitis problems to have experienced acute clinical acidosis. The background is more typically chronic and subacute rumen acidosis which has been described (Kersting et al., 1993).

The diagnosis of subacute rumen acidosis has been difficult to make. Specific diagnostic tests have not been well developed. Veterinarians struggle to identify an emerging abnormal herd problem from the "normal" rates of disease in a herd. A surprising number of herd referrals that are finally diagnosed as chronic acidosis herds begin with a concern about "immunosuppression". This concern arises from a sense that the herd is experiencing an increase in a variety of health problems and that treatment response is poor.

Clinical signs of chronic rumen acidosis in a dairy herd are highly variable. At a minimal level, signs may include poor appetites, particularly in fresh cows, and occasional diarrhea and laminitis. If the rumen mucosa is damaged severely, rumen microorganisms can enter the rumen wall and portal circulation, resulting in rumenitis and abscess formation. In addition to liver abscessation, septicemia can produce lung abscesses, bacterial endocarditis, and sub-solar abscesses. At this stage, clinical signs can include thin and emaciated cows, poor response to therapy for peri-parturient infections, peritonitis, hemoptysis, and death. The prevalence of these signs depends upon the severity and duration of exposure to acidosis.

Many investigators look to the herd production records and assume that acidosis herds will show low milk fat percentages. While this assumption is sometimes correct, many acidosis herds have a history of normal fat production. Herds that suffer from an "adaptation acidosis" tend to have normal fat production because of the relatively small proportion of the herd at risk at one time.

Several tests of rumen fluid have been recommended for the diagnosis of rumen acidosis (Dirksen and Smith, 1987). These tests include pH, methylene blue reduction, sedimentation and flotation, and various chemical concentration tests. While tests such as the methylene blue reduction test and the microscopic exam for protozoa are useful in a diagnosis of acute acidosis, they have been of marginal value in investigations of subacute acidosis in modern dairy herds where almost all cows are fed a significant level of concentrates.

In the author's experience, pH appears to be a valuable test for subacute rumen acidosis. However, the recommended procedure of recovering rumen fluid by stomach tube or Dirksen probe (Dirksen and Smith, 1987) has proven to be less than satisfactory. The samples become contaminated with variable amounts of saliva, which is alkaline. To eliminate this source of variability, the author began collecting rumen fluid via simple needle aspiration. On one farm, samples were recovered simultaneously by Dirksen probe and needle aspirate. The results from six cows are reported in table 1. The mean difference in pH between the two methods was 1.1 pH units with a standard deviation of .42 units among the six sets of samples. This difference is considerably greater than that reported by Dirksen and Smith (1987) using fistulated cows. Research work is ongoing at the University of Wisconsin to validate the safety and variability of a needle aspirate method in cows in four different ration groups.

For reasons of confidence and convenience, the author prefers the needle aspirate method to collect rumen fluid. Samples are collected at an estimated two to five hours following feeding. Narrow range (5-7) pH paper (S/P pH Indicator Strips, Scientific Products, Inc., Minneapolis, MN) has been acceptable, but an electronic pH meter (Cardy Twin Soil and Water pH Meter; Spectrum Technologies, Inc., Plainfield, IL) provides greater precision. Our investigative service considers pH measurements of 5.9 and greater to be normal, 5.6 to 5.8 a marginal zone where ration management should be modified, and values of 5.5 and less a crisis zone. At the current time, a ration is not judged to be "marginal" or "in crisis" on the basis of only one cow. At least one third of the animals sampled from a feeding group should fall well into the marginal or crisis range before the ration is categorized as needing modification.

Feeding Management Problems that Produce Acidosis. Much research work on rumen acidosis emphasizes the adaptation of the rumen microbial population to increased levels of concentrates. Bacteriological studies suggest that about 21 days are required and that concentrate levels should be increased at 5 to 7 day intervals throughout the period (Mackie and Gilchrist, 1979).

Dirksen et al. (1985) have emphasized the importance of adaptive changes of the rumen mucosa in the prevention of acidosis. Mean surface area of rumen papillae will increase from 0.3 mm^2 to 1.2 mm^2 when exposed to high concentrate rations, but the process takes from four to six weeks. The larger surface area of adapted papillae was related to VFA absorption rates three times greater than rates for unadapted, smaller papillae.

Table 1. Comparison of pH measurements by collection method.

Cow ID	Dirksen probe	Needle aspirate
3	6.5	5.7
31	6.2	5.3
33	6.8	5.3
44	7.0	5.3
67	6.8	6.2

While acidosis researchers emphasize the adaptation of microbial populations and rumen papillae, nutrition advisory services in the field appear to be concerned primarily with excess rapidly fermentable carbohydrate levels in the rations. Recommendations for fiber content of dairy rations have been developed by the National Research Council (1988). Modifications of these recommendations for typical dairy rations in the midwest are found in table 2 (Shaver, 1993). Fiber guidelines should be modified by factors such as the fiber type, particle size and distribution, total dry matter intake, bulk density of ration, buffering capacity of the forage, feeding frequency, and body condition and production level of the animal (National Research Council, 1988).

Three of the most common nutrition management problems that produce acidosis are a failure to adapt the dry cow for lactation rations, unrealistic dry matter intake (DMI) expectations in the peri-parturient period, and an overestimation of forage DMI in total mixed rations.

Adaptation of dry cows to lactation rations. As total mixed rations (TMR) have been increasingly adopted by smaller dairy herds in the upper midwest, it has become a common practice to prepare one ration for the entire lactating herd. The single lactation TMR has made difficult the gradual introduction of concentrates to individual fresh cows in the weeks after calving. The single TMR can create acidosis problems for unadapted fresh cows and is necessitating the creation of transition rations between the dry cow and lactation ration.

Guidelines as to the maximal acceptable change between rations are scarce. Elam (1976) has recommended that the net energy of a ration can be safely increased about 10% at a time. For example, a change from an energy density of 0.70 mcal/lb to 0.77 mcal/lb would be viewed as safe. Many dry cow rations are estimated to have 0.58 mcal/lb and many lactation TMR rations have 0.78 mcal/lb. Observation of the 10% guideline would require two intermediate rations. Practical experience suggests that most cows can adapt with a single intermediate ration.

Table 2. Fiber guidelines for diets of lactating dairy cows.

Fiber analysis	Minimum fiber as a % of dry matter
Crude fiber	15-17
Acid detergent fiber	19-21
Neutral detergent fiber	27-30
Neutral detergent fiber from forage	21-22

Unrealistic DMI expectations in the peri-parturient period. The traditional prediction equations for DMI have not addressed the dynamic changes in intake in the immediate post-parturient period. Recently, DMI prediction equations for cows at each week post-partum have been published (Kertz et al, 1991). Table 3 lists daily DMI for two example cows. Dairy operators have been told for years to minimize the "negative energy balance" of early lactation and have attempted to maximize concentrate intake in early lactation. Field recommendations for the feeding of component-fed concentrates during the first three weeks are commonly excessive. For example, it is common to find cows fresh 7 days consuming 20 lbs of dry matter from concentrates. Rations like these rarely meet the NRC (1988) fiber guidelines for early lactation cows at zero to three weeks in milk.

Table 3. Dry matter intake predictions by week post-partum.

Week post-partum	First lactation, 1200 lbs BW, DMI lb/day	Later lactation, 1350 lbs BW, DMI, lb/day
1	29	33
2	32	37
3	35	41
4	36	43

Occasionally, the same situation applies to component-fed "steam-up" rations for dry cows. Bertics et al. (1992) have shown significant reductions in DMI in the last few days prior to parturition. If component-fed concentrates are consumed and "free-choice" forages refused as DMI drops, the dry cow may experience acidosis prior to calving.

Overestimation of forage DMI in total mixed rations. Field experience suggests that a minority of TMR operators monitor moisture of forages on an at-least weekly basis. The majority of dairy operators do not monitor moisture, but observe the rate at which cows clean up the bunk and adjust the forage weight of the next batch. In the upper midwest, the predominant forage is alfalfa haylage. If cows clean up the TMR feeding quickly, the weight of as-fed haylage is increased next time. Conversely, if TMR is left, forage is reduced in the following batch.

The practice is conceptually correct if the observed change in consumption is due to dry matter changes in the forage. However, if the change in consumption is due to anything other than the forage dry matter, the subsequent adjustments are incorrect. If the group of cows reduces its DMI and the dairy operator subsequently reduces haylage in the TMR, the ration usually becomes fiber deficient. Routine monitoring of dry matter of feed ingredients is an important task of TMR management.

The usual objection to monitoring forage dry matter is the time required to perform the test. Dairy extension services commonly recommend the use of a microwave oven for the determination. Oetzel et al., 1993 have compared a variety of methods. The use of an electronic meter (1210 Silage Tester; Farmex Inc., Aurora, OH) required the least operator skill and time, and accuracy was acceptable for haylage and high-moisture shelled corn. The electronic tester can help overcome objections to performing the test and reduce the risk of inappropriate TMR adjustments.

There are other feeding management problems that create acidosis and laminitis, but appear to be less common. Formulation errors, fine chopping and grinding, excessive mixing of the TMR, and slug-feeding of components remain as occasional problems. Again, all of these problems occasionally create laminitis in replacement heifers as well as mature cows.

STANDING TIME ON CONCRETE SURFACES

Veterinarians have speculated for years that exposure to hard surfaces is a contributing factor in laminitis. Greenough and Vermunt (1991) have used the term "overloading laminitis" to describe the phenomena. While several anecdotal reports can be found, a report by Colam-Ainsworth et al. (1989) is particularly compelling. One farm in England built two 130-cow dairy units. Both facilities were identical with the exception of manure handling systems. The facility constructed last had a liquid manure handling and storage system. Replacements were raised in common facilities. Feeds were produced on common fields. Ration management was identical. Yet the second barn experienced annual laminitis rates in lactating heifers of 47 to 70% during its first four years of operation, while the first barn experienced no lameness. When moved from the problem barn to the normal barn, lame heifers usually recovered.

Thorough comparative analyses are reported (Colam-Ainsworth et al., 1989). A summary of animal behavior parameters is presented in table 4. Stalls, ventilation, and feeding facilities were essentially identical. Because of the manure handling system, the problem barn used one-fourth the volume of bedding per stall as the normal barn. The report notes that bedding usage met "ADAS" quantity recommendations in the problem herd, but did not exceed such recommendations. Identification of the bedding and stall usage behavior differences convinced the manager to increase the bedding usage to equivalent amounts. The laminitis problem disappeared from the problem barn.

Table 4. Behavior during repeated 2 hour observation periods.

Behavior	Problem Barn	Normal Barn
% standing at observations	50-55	25-35
% lying down for entire period	32	65
% lying within 10 minutes of entering barn	29	70

Field investigations by the author have never found control groups like the report cited above, but several herd laminitis problems have been resolved following the correction of stall maintenance problems. The clinical issue appears to be the proportion of time spent standing on concrete. Access to bedded packs or earth exercise lots or increased time lying down appear to resolve the problem.

Because of mud and manure handling problems in open lots, total confinement operations are common in the upper midwest. In these facilities, proper stall design is a critical determinant of adequate resting time. Dairy cow stall design should provide for four functions: a comfortable surface to lie on, adequate platform space for the resting cow's body, lunge room during rising, and adequate neck room to complete rising.

Stall surface. The stall surface must be comfortable enough to attract a cow to lie down. Surface cushion makes a difference. Mean dairy cow resting times of 14 hours per day have been reported for deep straw and 7 hours per day on unbedded concrete (Cermak, 1986). Unpublished work in Wisconsin confirms 7 hours per day on concrete, with increases to about 9 hours per day with the installation of an unsatisfactory straw-filled mattress that compressed quickly.

The stall should have a soft, moldable surface from front to rear. The bedding should be dry and deeper than 4 inches. Because of minimal opportunity for bacterial growth, sand is preferred, followed by shavings and sawdust, sunflower hulls, chopped straw, shredded newspaper, and long straw.

Bedding placed on top of a flat platform gets dragged off, making the rear platform hard, uninviting, and a factor in development of hock calluses and crushing teat injuries. Sand has recently been approved as a bedding material in stanchion and tie-stall milking barns in Wisconsin. A PVC pipe is mounted toward the rear of the platform to help retain the sand. If a bedding-retaining curb is not an option, a mattress or bedding filled fabric bag will be useful. Preferred fillers are sand or shredded rubber. Mattresses must have some modest amount of dry loose bedding on top to absorb moisture. Bare concrete and unbedded rubber mats are unacceptable surfaces for the humane housing of cows.

Resting space. The platform from the rear edge of the stall to the area of the brisket board (or stanchion) must accommodate the cow's body. This length should be based upon cow size rather than breed, because of wide variation within breeds. Where cows choose their stalls, the stalls should be sized to fit the average of the biggest 25% of the group using the stalls. The length needed is approximated by the body length measured from the ischiadic tuber (pin bone) to the major tubercle of the humerus (point of the shoulder) (Cermak, 1986). Body length (BL) can be calculated (Cermak, 1986) from chest girth (CG) with the following equation: $BL_{cm} = 18.605 + (0.70175 \times CG_{cm})$. Alternatively, body length (BL) can be calculated from body weight (BW) as follows: $BL_{cm} = 18.605 + (0.70175 \times (23.292 \times BW_{kg}^{0.333})$. Calculated body lengths for animals of various weights and girths are presented in table 5.

Stall width should be 48 inches (1.2 meters) for cows greater than 1050 lbs (475 kg). If cows weigh less than this, the stalls should be 44 inches (1.1 meters) wide (Cermak, 1986).

Lunge room when rising. Photographic analysis of cows rising on pasture indicates that a forward lunge space of 27-39 inches (0.7 to 1.0 meters) is used in the rising movement (Cermak, 1986). The area in front of the brisket board should allow for this forward lunge and be free of low obstructions to allow the head to "bob" during the lunge.

Total stall length should accommodate the body space requirement for the cow plus the head space required for rising. For example, a mature 1500 lb (680 kg) Holstein cow would need 64 in (162 cm) resting area plus 27-39 in (70-100 cm) lunge area for a total stall length of 7.5-8.0 ft (91-103 cm). The high end of the range allows a full lunge space for all cows.

Many dairy barns with stalls of inadequate length also have interior dimensions that preclude lengthening the stalls. Free stalls of deficient length can be modified in two ways. Openings can be cut in the front of some stalls so cows extend their heads forward through the barrier. More commonly, the stall dividers are replaced with a divider where the lower bar is eliminated or bent upward (Michigan-style, DaSilveira, Dutch comfort, etc.) in the anterior area of the stall, allowing lunge-space into the next stall to the side.

Neck room to rise without obstruction. The neck rail should be located at a height 6-10 inches (15 to 25 cm) below that of the withers (Cermak, 1986) so that a cow can rise without hitting it. It should be positioned directly above the brisket-board. As measured from the rear curb of the stall, the neck rail should be positioned forward and above a distance equal to the resting body length as described above. Average cow wither height ranges from 47 in (120 cm) to 62 in (157 cm) and greater. While the neck rail limits forward movement of the cow, it is less effective than the brisket board in controlling fecal and urine contamination of the stall.

Table 5. Estimated relationship between body weight, girth, and required resting stall length.

Body Weight, Kg (lb)	Chest Girth, Cm (In)	Body Length, Cm (In)
408 (900)	172 (68)	140 (55)
454 (1000)	179 (70)	144 (57)
499 (1100)	184 (73)	148 (58)
544 (1200)	190 (75)	152 (60)
590 (1300)	195 (77)	155 (61)
635 (1400)	200 (79)	159 (63)
680 (1500)	204 (80)	162 (64)
726 (1600)	209 (82)	165 (65)

CONCLUSION

Both chronic, subacute rumen acidosis and excess standing time on concrete can produce herd problems with laminitis. Many dairy farms have moderate problems in both areas and they may be additive in effect on the herd. Resolution of the herd problem may require resolution of relatively minor problems in both ration and stall management.

REFERENCES

Bertics, S.J., R.R. Grummer, C. Cadorniga-Valino, and E.E. Stoddard. 1992. Effects of prepartum dry matter intake on liver triglyceride concentration and early lactation. J. Dairy Sci. 75:1914.

Cermak, Jan. 1986. The design of cubicles for British Friesian dairy cows. Page 119 *in* Proc.: Influence of the design of housing systems for cattle on lameness and on behavior, Brussels.

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

Dirksen, G.U., H.G. Liebich, and E. Mayer. 1985. Adaptive Changes of the Ruminal Mucosa and Their Functional and Clinical Significance. The Bovine Practitioner 20:116.

Dirksen, G.U., and M.C. Smith. 1987. Acquisition and Analysis of Bovine Rumen Fluid. The Bov. Practitioner 22:108.

Elam, C.J. 1976. Acidosis in Feedlot Cattle: Practical Observations. J. An. Sci. 43:898.

Greenough, P.R., and J.J. Vermunt. 1991. Evaluation of subclinical laminitis in a dairy herd and observations on associated nutritional and management factors. Vet. Rec. Jan. 5, 1991:11.

Hoblet, K. 1993. Watch for laminitis problems. Dairy Herd Management. July, 1993:14.

Kersting, K.W., J.R. Thompson, and W.M. Wass. 1993. Diseases of the Ruminant Forestomach. Page 716 *in* Current Veterinary Therapy 3: Food Animal Practice, Howard, J.L. (ed), W.B. Saunders Co., Philadelphia, PA.

Kertz, A.F., L.F. Reutzel, and G.M. Thomson. 1991. Dry Matter Intake from Parturition to Midlactation. J. Dairy Sci. 74:2290.

Leedle, J.A.Z. 1991. Microbiology and Physiology of the Rumen and Its Response to Different Feeding Strategies. Proc. Am. Assn. Bov. Pract. 23:3.

Mackie, R.I., and F.M.C. Gilchrist. 1979. Changes in Lactate-Producing and Lactate-Utilizing Bacteria in Relation to pH in the Rumen of Sheep During Stepwise Adaptation to a High-Concentrate Diet. Appl. Envir. Microbiology 38:422.

National Research Council. 1988. Nutrient Requirements of Dairy Cattle, 6th Rev. Edition. National Academy Press, Washington, D.C.

Oetzel, G.R., F.P. Villalba, W.J. Goodger, and K.V. Nordlund. 1993. A Comparison of On-Farm Methods for Estimating the Dry Matter Content of Feed Ingredients. J. Dairy Sci. 76:293.

Peterse, D.J. 1985. Laminitis and Interdigital Dermatitis and Heel Horn Erosion. Vet. Clin. N. Amer. Food An. Prac. 1:83

Russell, J.B., and T. Hino. 1985. Regulation of Lactate Production in *Streptococcus bovis*. J. Dairy Sci. 68:1712.

Shaver, R. 1993. Troubleshooting problems with carbohydrates in dairy rations. Vet. Med., Oct. 1993:1001.

Weaver, A. D. 1988. Laminitis. The Bovine Practitioner, 23:85.

Wells, S.J., A.M. Trent, W.E. Marsh, and R.A. Robinson. 1993. Prevalence and severity of lameness in lactating dairy cows in a sample of Minnesota and Wisconsin herds. J. Am. Vet. Med. Assn. 202:78.