DAIRY HERD HEALTH

PROGRAMMING CONFERENCE

JUNE 6-7,1984

EARLE BROWN CONTINUING EDUCATION CENTER

UNIVERSITY OF MINNESOTA

ST. PAUL CAMPUS

TABLE OF CONTENTS

TABLE OF CONTENTS (continued)

PRACTICAL ASPECTS OF METABOLISM AND METABOLIC PATHOPHYSIOLOGY IN DAIRY COWS

Thomas H. Herdt DVM, MS
Diplomate, American College of Veterinary Internal Medicine
Michigan State University

The purpose of this outline is to review the current understanding of the pathophysiology of some metabolic diseases of dairy cows. The intention is to increase the practitioner's awareness of pathophysiology and to point out the role of dietary control in the prevention of metabolic disease. The outline covers calcium and phosphorus, lipid and energy, and protein digestion and metabolism.

CALCIUM METABOLISM AND HOMEOSTASIS

The calcium pool

About 1% of the calcium in the cow's body is contained in a dissolved or readily soluble state. This is known as the soluble calcium pool. Serum calcium forms part, but not all, of this pool. Anatomical locations for other portions of the calcium pool include the extracellular fluid, intracellular fluid and probably a readily soluble portion of newly formed bone. Calcium continuously enters and leaves the pool from the gut and bone. It also leaves into the urine, milk and fetal tissues. Most research concerning the susceptibility of cows to hypocalcemia has dealt with movement of calcium into and out

of the pool. The total size of the pool has not been as well studied, although it may also have an influence in the cows suceptibility to hypocalcemia.

Factors controlling pool size and the rates of calcium transport into and out of the pool.

Parathyroid hormone (PTH)

1) Increases the inflow of calcium from bone
2) Decreases the loss of calcium into the urine
3) Promotes phosphorus loss into the urine, during times of adequate body phosphorus stores
4) Promotes the formation of calcitriol from vitamin D

Calcitriol

1) Increases the efficiency of calcium absorption from the gut
2) Together with PTH, increases the flow (absorption) of calcium from bone

Changes in the calcium pool size and transport rates (rates of flow into and out of the pool) at parturition.

At parturition calcium flows rapidly from the calcium pool into the udder. In cows which have been in positive calcium balance during the last portion of the dry period, the calcium pool size shrinks and the flow of calcium from the gut increases after calving.

Approaches to the prevention of milk fever by the manipulation of dietary calcium.

Oral calcium prophylactic therapy has been used with apparent success. It consists of giving large doses (125g) of calcium chloride (a very soluble calcium source) just prior to calving. This method takes advantage of the natural increase in intestinal calcium absorption that occurs around the time of calving. To be successful, animals have to be observed closely so as to properly time the administration of calcium.

Low calcium prepartum diets

This method relies on placing the cow in negative calcium balance prior to calving. This stimulates PTH secretion and activates bone resorption, thus there is rapid flow of calcium from the bone to the calcium pool at the time when milk secretion is initiated. PTH secretion is normally stimulated by the drop in serum calcium that accompanies the initiation of lactation; however, in many cases the bone is not prepared to respond maximally to PTH secretion and thus calcium absorption from the skeleton does not occur, in spite of PTH stimulation. With low calcium prepartum diets PTH secretion precedes the high calcium demands of lactation by several days or weeks. This gives the bones, particularly the osteoclasts, time to become sensitized to the action of PTH. This allows rapid bone resorption to occur simultaneously with the initiation of milk secretion.

This method of milk fever prevention has been extensively tested in both laboratory and field trials, and appears to be very effective. However there are field reports that it is sometimes does not work. There are a number of dietary factors, in addition to calcium intake which can influence bone mobilization. These may interact with calcium intake to determine the total effectiveness of the diet in preventing hypocalcemia.

Calcium content and duration of feeding

One potential problem with the concept of low calcium prepartum diets is that "low" is a relative word. The NRC recommends 37g of calcium intake daily for the average Holstein dry cow. This amount of calcium is lower than that naturally present in many forage diets, and thus would appear to many people to be a "low calcium" diet. However, a diet containing 37g of calcium may not, and indeed should not, place the cow into negative calcium balance. Most successful low calcium dry cow diets have contained less than 30g calcium, and in many cases less than 25g.

The optimum duration of feeding for calcium deficient diets appears to be one to two weeks. Most experiments utilizing these diets in the United States have not involved feeding periods longer than 30 days. In one German experiment dry cows were fed calcium deficient diets for the entire dry period with no apparent ill effects. Still, the amount of mobilizable bone is limited

and it would appear reasonable to not make the period of calcium deficiency any longer than necessary. I have observed that in one Jersey herd, feeding a calcium deficient diet for the last month of lactation and throughout the dry period resulted in a 100% incidence of milk fever. This may have resulted because the cows supply of readily mobilizable bone was depleted.

Hormonal factors influencing bone calcium mobilization

The role of sex hormones in milk fever has been debated for some time. It is known that the serum concentration of estrogen rises and that of progesterone declines in the cow just prior to parturition. It is also known that estrogens tend to inhibit resorption of bone. While some experiments have discounted the role of sex hormones as a factor in the pathogenesis of milk fever, recent observations on cows with spontaneously occurring milk fever demonstrated that they had higher concentrations of estrogen than cows on the same diet that did not develop milk fever. While differences in estrogen concentration may account for some of the individual variation in milk fever occurrence on a given diet, it is likely that a diet sufficiently low in calcium will over come most of the estrogenic effects on calcium mobilization.

Magnesium deficiency

Magnesium is known to play some role in calcium mobilization. It has been known for some time that

magnesium deficient rats do not readily mobilize calcium from bone. Recently this has also been demonstrated in cattle. The degree of hypomagnesemia associated with impaired calcium mobilization in cows is similar to that occurring on some dry cow diets that are unsupplemented with magnesium. The amount of magnesium in dry cow diets should be at least .2% of the dry matter and the serum magnesium concentration of dry cows should be greater than 2 mg/dl. In addition to monitoring the diet and serum, evaluating urinary concentrations of magnesium is also useful. Magnesium is absorbed from the gut in amounts proportional to the dietary concentration; the amount in excess of need is excreted, primarily, in the urine. Thus, if there are reasonable concentrations of urinary magnesium, the dietary supply is more than adequate. Unless, for whatever reason, the urine is very dilute, the magnesium concentration should be 15 to 20 mg/dl or above.

Improper phosphorus supplementation

Opinions abound on the proper use of phosphorus supplements for dry cows. Before discussing this issue directly I would first like to review some aspects of phosphorus digestion and metabolism in ruminants.

Phosphate absorption and metabolism in ruminants

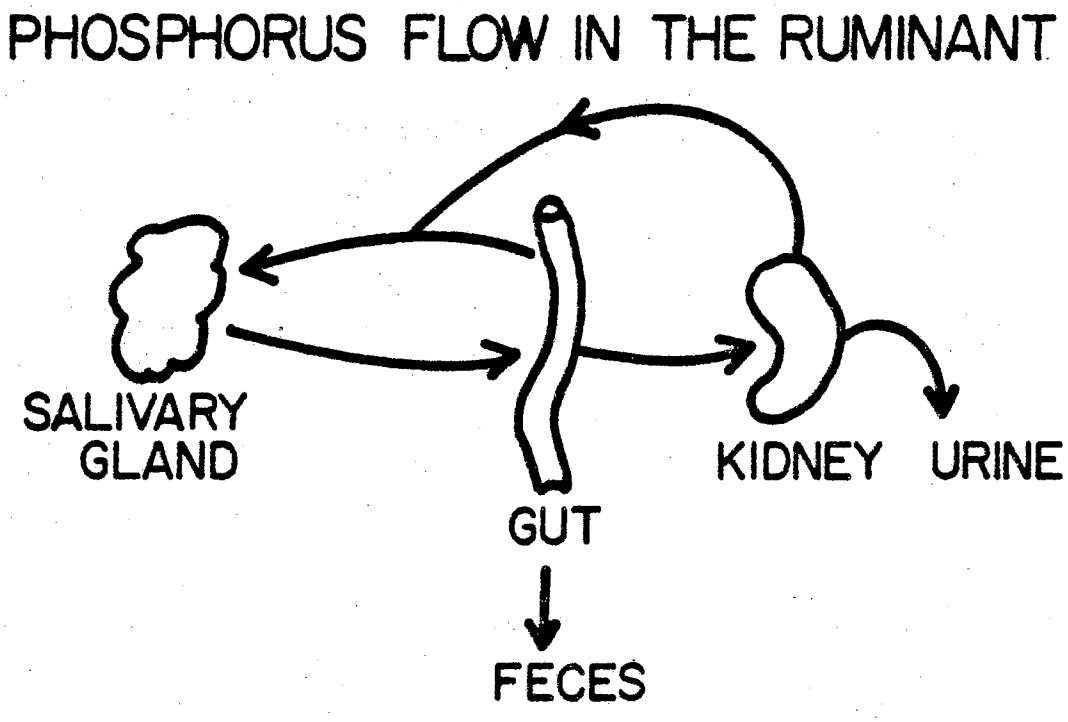
1) Chemical effects of the dietary calcium:phosphorus ratio

The fact that dietary calcium and phosphorus influence the retention of one another is well established; the exact mechanism of this interplay is not

as well understood. Frequently it is suggested that increasing the dietary concentration of one mineral decreases the availability of the other. This suggestion is based on the fact that in order for the minerals to be absorbed they must be in solution. None of forms of calcium phosphate are very soluble in water at a neutral pH. When the gut water is saturated with respect to calcium and phosphate, the product of the concentrations of the ions is expected to be a constant. Therefore, if the concentration of one ion in the solution is increased, the concentration of the other is expected to decrease. In fact, this relationship has been demonstrated in ileal contents from calves. However, the solubility of the calcium phosphates increases markedly as the pH decreases. While the pH of the ileum is near neutrality, that of the upper gut, where most calcium and phosphate absorption takes place, is below 7. At the pH of the upper gut (duodenum and upper jejunum) the reciprocal effects of calcium and phosphate ions on each other's solubility cannot be expected to have a great effect on dietary availability.

2) Salivary phosphate recycling in ruminants

The phosphate concentration of ruminant saliva is much higher than other species (approx. 10 mg/dl). In addition, cattle secrete phenomenal quantities of saliva (as much as 200 liters per day). Thus, most of the phosphorus presented to the intestine for absorption is


PHOSPHORUS FLOW IN THE RUMINANT
SALIVARY GLAND
GUT
KIDNEY
URINE
FECES

endogenous, i.e. from saliva. Therefore the ratio of calcium to phosphorus at the absorption site is considerably different than it is in the diet.

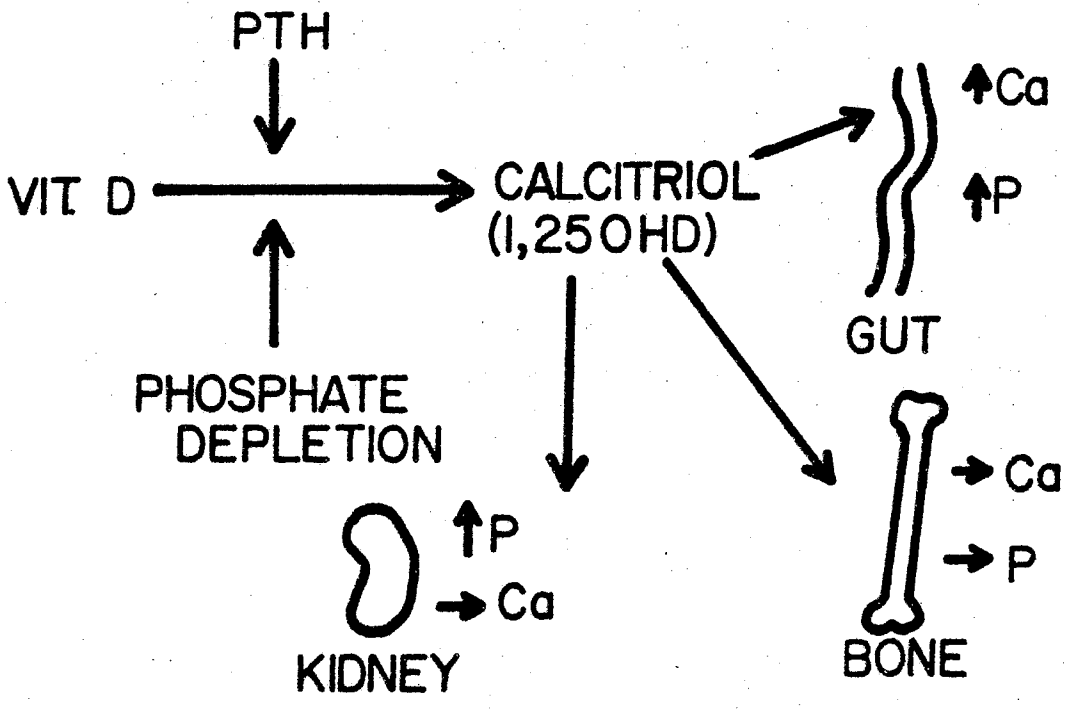
Regulation of phosphate homeostasis

1) Monogastrics

In the monogastric, and apparently in the ruminant under certain conditions, the primary regulation of phosphate homeostasis is exerted at the renal level. In general, in both monogastrics and ruminants, phosphorus is absorbed from the gut in direct proportion to dietary supply. If phosphorus is absorbed in excess of the body needs, the extra amount is excreted in the urine.

Calcitriol (activated vitamin D or 1,25-dihydroxy vitamin D) is one of the factors regulating phosphorus homeostasis. It increases the efficiency of phosphate absorption from the gut, aids in stimulating bone resorption and, in the absence of PTH, decreases phosphorus loss into the urine. The synthesis of calcitriol is stimulated by either PTH or low phosphate concentration in the renal cortex. Thus, calcitriol's formation can be stimulated either indirectly, via PTH, during times of calcium deficiency, or directly during times of phosphorus deficiency.

A poorly defined mechanism is also present that stimulates renal conservation of phosphorus, even in the face of high serum PTH concentrations. This mechanism allows for renal conservation of both calcium and


PTH
VIT D
CALCITRIOL
(1,25OHD)
↑Ca
↑P
GUT
PHOSPHATE
DEPLETION
↑P
→Ca
KIDNEY
→Ca
→P
BONE

phosphorus at times when both ions are in short supply.

2) Ruminants

The renal mechanisms for the regulation of phosphorus homeostasis outlined above are probably also present in ruminant animals, although they have been best described in monogastric species. However, by virtue of the large amount of salivary phosphorus that enters the gut in ruminants, intestinal reabsorption of phosphorus may play a larger role in ruminant phosphate homeostasis than it does in monogastrics. Small changes in the efficiency of intestinal phosphorus absorption could be expected to have a large influence on ruminant phosphorus balance because such a large quantity of the total phosphorus pool is repeatedly circulated through the ruminant gut.

Dietary phosphorus and milk fever

The phosphorus consumption of the dry cow appears to have an important influence on the incidence of milk fever. In an Ohio study performed on 17 commercial dairy herds, feeding either phosphorus or calcium in amounts above NRC recommendations to dry cows resulted in an increased incidence of milk fever. In this study the serum inorganic phosphate concentrations (the usual measurement of serum phosphorus) of the dry cows were positively correlated to both the dietary concentration of phosphorus and the incidence of milk fever. An Iowa experiment was designed to study the influence of various combinations of dietary calcium and phosphorus during the

dry period on vitamin D metabolites and hypocalcemia at parturition. Dry cows were fed diets containing either low calcium-low phosphorus, low calcium-high phosphorus, high calcium-low phosphorus or high calcium-high phosphorus. The investigators hypothesized that high levels of phosphorus feeding may suppress vitamin D activation (calcitriol formation) and thus predispose cows to milk fever. Although no variation in serum calcitriol concentrations among the treatments was observed, the cows receiving the high calcium-high phosphorus diet were markedly less able to mobilize calcium at the initiation of lactation. Cows in this group became significantly more hypocalcemic, and remained hypocalcemic longer after freshening than cows in the other feed groups. Cows fed the high calcium-high phosphorus dry cow diet were the only ones in the study to develop milk fever. In the many experiments that have been conducted since the early 1950s, almost no cases of milk fever have been recorded in cows that have received less than 20 g of calcium and less than 40 g of phosphorus daily through the last weeks of the dry period. This is especially remarkable when one considers the general lack of consistency that exists in the results of many of these experiments. It is thus apparent that the phosphorus, as well as the calcium, intakes of dry cows must be monitored and that phosphorus supplementation of dry cow diets is frequently unnecessary and may well be detrimental.

Vitamin D and milk fever

Vitamin D deficiency

It appears that vitamin D deficiency during the dry period can predispose cows to milk fever. Since many dry cows are receiving forages of less than high quality, it is important to provide adequate vitamin D supplementation to assure that deficiencies will not occur. Supplemental levels of 25,000 units per head per day are ample.

Vitamin D prophylactic therapy

Vitamin D, and its various metabolites, have been tested for their usefulness in prophylactic therapy for milk fever. In general the metabolites of vitamin D: 25-hydroxy vitamin D and 1,25-dihydroxy vitamin D, have appeared effective in preventing milk fever. They are, however, not available at a reasonable price for use in dairy cattle. Vitamin D therapy has successfully prevented milk fever in some instances, but not in others. If vitamin D is to be used in milk fever prevention, the following guidelines should be used:

1) Administer 10×10^6 approximately one week before calving.
2) If the cow does not calve in the week following administration, another dose may be given.
3) High phosphorus diets may limit the effectiveness of vitamin D in preventing milk fever.
4) Vitamin D is potentially toxic at doses close to those recommended for milk fever prophylaxis and it is more toxic in dry cows than fresh cows.

Calcium digestion and metabolism in the fresh cow

Most cows are in negative calcium balance for the first several weeks, or even months, of lactation. The National Research Council states that their recommendations for calcium consumption will not meet the calcium requirements of cows in early lactation. This means that most cows must mobilize skeletal calcium to meet the demands of milk production. Most research has indicated that this is not detrimental so long as the animals can replace the lost skeletal calcium during the final stages of lactation.

Serum calcium concentrations in fresh cows

During the period of negative calcium balance most cows are able to maintain their serum calcium concentration in the normal range. This has lead to the general statement that serum calcium measurement is of little or no value in assessing the adequacy of calcium nutrition. However, this does not mean that serum calcium measurements are of no value in assessing calcium homeostasis. Herds of dairy cows can be found in which the serum calcium concentrations of many cows in early lactation are below 9.0 mg/dl. If we assume that the cows are in negative calcium balance, a reasonable assumption for fresh cows, then these serum calcium concentrations should be associated with intense parathyroid activity and skeletal calcium mobilization. In other words, the cow's endocrine system is scrambling to maintain normal serum calcium values and not making it. While negative calcium

balance in itself may not cause a problem, low serum calcium concentrations may. The strength and frequency of rumen contractions appears to be linearly related to serum calcium, even in the normal range of serum calcium concentrations. Abomasal contractility may also be reduced by marginally low serum calcium.

In response to finding low serum calcium concentrations in fresh cows, several questions should be asked:

1) Were an adequate number of samples taken? At least seven cows in early lactation should be sampled.

2) Were the cows apparently healthy and eating the feed presented to them? Anorexia will reduce serum calcium in nearly all milking cows.

3) Does the diet appear to be balanced? The entire diet, in addition to the calcium supplementation, should be evaluated.

After these things have been examined, raising the calcium content of the diet is the appropriate response to make to low serum calcium values.

Serum phosphorus concentrations in fresh cows

Serum inorganic phosphorus concentrations are, in general, more responsive to dietary phosphorus levels than are serum calcium concentrations. However, in addition to dietary phosphorus, several other factors must be considered in the interpretation of serum phosphorus.

1) Sample handling - Many organic phosphorus compounds within the red blood cells tend to dephosphorylate with time after the blood sample is taken. The resultant phosphorus then leaks from the cells into the serum, raising the serum phosphate concentration. Serum samples for phosphorus determination should be removed from the clot as soon as possible.
2) Calcium deficiency with adequate dietary phosphorus - Bone mobilization releases phosphorus as well as calcium. If dietary phosphorus is adequate to meet the production demands, and bone is being mobilized to supply calcium, serum phosphorus may be increased.
3) Time of day - Daily fluctuations in serum phosphorus concentrations usually exceed those of serum calcium. Phosphorus values appear to peak shortly after eating and then decline. The drop in concentration may be due to the flow of saliva into the rumen or the phosphorylation of absorbed carbohydrates. The fluctuation may be as large as 2.0 mg/dl and appears to be the greatest on diets which result in the highest serum phosphorus values.

After considering the above points, and thoroughly evaluating the ration, some dietary changes may need to be based on serum phosphorus values. It has been my experience that diets unbalanced enough to result in abnormal serum phosphorus concentrations, usually will be discovered by simple mathematical evaluation of the ration. However, the serum values may add one more piece to the puzzle during problem herd investigations.

FAT MOBILIZATION AND ITS ASSOCIATED PROBLEMS

Nearly all dairy cows are in negative energy balance for the first several weeks after calving. This situation may be unavoidable in high producing cows and it may in fact be desirable for the production of maximum butterfat yields. During the period of negative energy balance body fat is mobilized and cows lose weight. Some metabolic problems can be associated with rapid fat mobilization and weight loss.

Fat cow syndrome

The so called "fat cow syndrome" is the extreme degree of a fat mobilization associated health problem. Several clinical and pathological conditions characterize this syndrome:

1) Obese prepartum cows

2) Mild to severe ketosis pre and or postpartum

3) High death loss

4) Liver floats in water

Fat mobilization syndrome

Fat cow syndrome with its high death loss may in fact be the tip of an iceberg. Nearly all cows, even those in desirable body condition, mobilize fat in late gestation and early lactation. This frequently results in an increase in the concentration of liver fat. The effects of fat mobilization and fatty liver infiltration on animal health and productivity are probably a matter of degree. In contrast to the American description of "fat cow syndrome," British investigators have demonstrated fatty liver infiltration to be extremely common in fresh cows. In the British studies, severe illness or death loss has not been observed to be a frequent consequence of fatty liver. In their clinical studies, reduced fertility without other clinical signs has been the health problem most commonly associated with fat mobilization and fatty liver.

The physiology of fat mobilization and pathophysiology of fatty liver

Adipose tissue

Fat is liberated from adipose tissue in response to hormonal and perhaps nervous stimuli.

The catecholamines (epinephrine and norepinephrine) are probably the most potent stimulants of adipose fat release. Other hormones such as glucagon, growth hormone and possibly prolactin, also stimulate adipose fat release, at least in some species. The glucocorticoids potentiate the response of adipose tissue to the fat mobilizing hormones.

In addition, low serum glucose concentrations may directly stimulate adipose fat release. Insulin counteracts the effects of most of the above mentioned hormones and inhibits fat mobilization.

Probably because of some combination of changes in hormone concentrations which occur in preparation for calving, cows start to mobilize fat about two weeks before freshening. This prepartum increase in fat mobilization is then followed by a further increase in fat mobilization after freshening. The second increase is in response to the hormonal alterations brought about by negative energy balance.

Adipose fat release, or lipolysis, appears to be a poorly regulated process. The rate of release of fat from the body fat stores may be proportional to the amount of adipose tissue. This would explain why obese cows are more prone to develop fat mobilization associated problems than cows in more desirable body condition.

Serum free fatty acids (FFA)

Fat is released from the adipose in the form of free fatty acids (FFA). FFA may have some direct effects on cow health. First, they combine with calcium to form calcium salts in the adipose tissue. If calcium mobilization from the bone is inadequate, then rapid adipose fat release may contribute to reduced serum calcium at freshening. Second, in the blood FAA are bound to albumin. There are two FFA binding sites per albumin molecule. When the concentration of FFA molecules exceeds twice the concentration of albumin molecules, then FFA can exist free in the serum and cause toxic reactions. Thus diet, management and therapy of dairy cows should be aimed at keeping the degree of FFA release at a moderate level.

Once released from the adipose, FFA can travel in one of several metabolic directions. First, they may be used directly by the tissues of the body for energy. If fact they probably supply a large portion of energy for the skeletal muscles of the cow. Second, they may be converted in the liver to ketones, which are then released into the blood to be used by the body for energy. Third, they may be packaged in the liver to lipoproteins. Lipoproteins may also provide energy to the body, but more importantly, in early lactation they also provide a large portion of the fat for butterfat synthesis.

Hepatic lipid metabolism

The liver takes up a large portion of the circulating FFA. A portion of the FFA taken up by the liver are converted to ketone bodies. This is a normal process and ketone bodies make up and important part of the energy supply to cows is early lactation. Extremely high concentrations of ketone bodies are associated with ketosis. Ketosis occurs under conditions in which there is rapid fat mobilization and a large portion of the fat is used for ketone body synthesis.

Much of the FFA that is not metabolized to ketone bodies, is converted back into triglyceride, the same type of fat found in the adipose tissue. Normally the triglycerides are then packaged into lipoproteins and secreted from the liver into the blood. The lipoprotein package consists of a core of triglyceride surrounded by a coat of protein, cholesterol and phospholipid. The ability to produce lipoproteins appears to be insufficient to meet the demand of rapid hepatic FFA uptake in cows that develop severe fatty livers.

The prevention of fat mobilization associated problems

1) Avoid obese dry cows.

Under many management systems today cows become too fat while in late lactation. This is especially true when there have been breeding problems in the herd, resulting in cows having long

lactations. Cows should be feed to go into the dry lot at a condition score of 3.0 to 3.5 on a scale of 0 to 5. They should neither gain nor lose body condition in the dry lot.

2) Reduce adipose fat release

A) Avoid feed restriction during the dry period.

Adipose fat is released anytime an animal is loosing weight. The problem with fat dry cows is not that they are fat, but rather that they are likely to mobilize fat rapidly and achieve high serum FFA concentrations. The objective of dry cow feeding should be to keep fat mobilization to a minimum. Therefore, you should not try to put dry cows on a "diet" since this would only increase FFA mobilization. Dry cows should be fed to maintenance requirements. In most cases a total ration of approximately 50% TDN will be sufficient to maintain the body weight of dry cows. Many dairymen have been lead to believe that the worst feed available is the best dry cow feed. This is not necessarily true since the worst feed available often contains less than 50% TDN. Feeding insufficient energy to dry cows will result in early FFA mobilization that will be superimposed on the natural increase in FFA mobilization that occurs just before calving.

B) Increase energy and starch intake rapidly after freshening

The appetite of all cows, and especially older cows, decreases prior to calving, remains low for the first few days of lactation, and increases gradually afterwards. This is probably a natural phenomenon that cannot be totally avoided. However, keeping the dry cow a "little hungry" will help stimulate her appetite after freshening. In addition feeding palatable feeds at frequent intervals to fresh cows and using high quality forages will allow the energy intake of cows in early lactation to be increased at the maximum possible rate without creating rumen acidosis.

C) Avoid stress during the late dry period and early lactation

It is impossible to totally avoid stress during this period, but anything that can be done to reduce the degree of stress will probably also reduce the degree of FFA mobilization. Sanitary measures to aid in the prevention of infectious diseases at freshening are particularly helpful.

D) Feed pharmacological doses of niacin during the late dry period and early lactation?

The B vitamin, niacin, at pharmacological doses, partially blocks the catecholamine stimulation of beta adrenergic receptors, such as those found on adipose tissue cells. Feeding 6 to 12 grams of niacin per head per day during the late dry period may reduce FFA release.

3) Increase hepatic lipoprotein output

A) Balance diet, especially for protein

C) Feed choline?

D) Feed inositol?

Protein and phospholipids are essential components of lipoproteins. If they are in deficient supply the mobilization of fat from the liver will be impaired. Choline and inositol are essential components of phospholipids and as such they must be present in sufficient supply for efficient mobilization of fat from the liver. Both of these substances can be synthesized in the cow's body, however it is possible that the rate of synthesis may not be rapid enough to provide all the phospholipid needed to respond to the rapid rate of fat mobilization which occurs in early lactation. If this is true, then feeding choline or

inositol to cows in the dry period and during early lactation may be beneficial; however, to date there is no good evidence that these substances are in fact deficient in the diets of fresh dairy cows. If they are not deficient, there is no reason to believe that additional amounts of choline or inositol added to the diet will enhance hepatic lipoprotein production. In addition, most available evidence indicated that preformed dietary choline is distroyed in the rumen and that ruminants have no dietary choline requirement.

Treatment of fat mobilization associated diseases

1) Reduce adipose fat release

A) Intravenous glucose therapy

Increasing the blood glucose concentration with the use of IV glucose therapy can be expected to decrease adipose FFA release directly and indirectly, through the stimulation of insulin secretion and the inhibition of glucagon secretion. Unfortunately, in the field administration of glucose usually consists of giving small quantities (250 grams) once or twice per day. This amount of glucose, administered IV as a bolus, can be expected to maintain the blood glucose concentrations above normal for only about three hours after administration.

B) Insulin therapy

Insulin is a strong direct suppressor of adipose FFA release. Doses of 150 to 200 IU of a long acting form of insulin (NPH or protamine zinc insulin) given subcutaneously appear beneficial in treating cows with severe fatty livers and with nonresponsive ketosis.

C) Force feeding

Probably the most desirable way to reduce adipose FFA release is to reduce the negative energy balance that created it. Under some situations, usually in large animal hospitals, force feeding via gastric intubation is possible and appears very beneficial.

2) Increase hepatic lipoprotein output

A) Glucose therapy

Glucose therapy, by reducing the need for hepatic gluconeogenesis, can be expected to reduce the destruction of amino acids and make them more available for the synthesis of protein, such as lipoprotein.

B) Insulin therapy

In addition to suppressing the release of FFA from adipose tissue, insulin promotes protein synthesis, potentially including lipoprotein synthesis.

C) Corticosteroid therapy

Corticosteroid therapy is controversial in the management of bovine fat mobilization problems. Corticosteroids enhance the fat release response of the adipose tissue to other stimuli. This effect can be expected to be detrimental to the treatment of fat mobilization problems. Corticosteroids, however, have other effects that may be beneficial. Perhaps most important, they stimulate appetite and suppress milk production, thus they may reduce the degree of negative energy balance. In addition, in some species, they directly stimulate the production of lipoproteins. This has not yet been demonstrated in cattle, but may occur. I personally feel that one or two doses of corticosteroids, in combination with other forms of therapy, probably has an overall beneficial effect in cows with severe fatty livers.

Diagnosis of fatty liver

Most of the clinical signs associated with fatty liver are vague and nonspecific. In addition, other diseases usually exist concurrently with fatty liver, clouding the diagnosis. These factors make the diagnosis of pathological fatty liver difficult to make with confidence. It is my feeling that severe fatty liver is probably diagnosed too often and without sufficient justification.

1) Urine ketones

Cows with ketosis invariably have some degree of fatty liver. Likewise, cows with severe fatty livers always have at least a detectable amount of ketones in the urine. Although the diagnosis of severe fatty liver cannot be made on the basis of ketonuria, the absence of ketonuria effectively rules out severe fatty liver.

2) Complete Blood Count

The white blood cell count is frequently reduced in cows with fatty livers. Although this is too nonspecific to be used diagnostically, it probably is related to the frequent observation that cows with fatty livers appear to be immunosuppressed.

3) Serum chemistry

No serum chemistry measurement is adequate for the diagnosis of fatty liver in dairy cows. When large numbers of cows with fatty livers are compared to normal cows, the serum total bilirubin concentrations are elevated and the serum albumin concentrations are reduced, on the average, in the cows with fatty livers. However, these abnormalities are usually very small in degree and are too variable and nonspecific for diagnostic use. Serum FFA are consistently elevated in cows with fatty livers, but this test is not available to most veterinarians in clinical practice.

4) Serum enzymes

Measuring serum activities of liver enzymes is not a sensitive way of assessing liver fat content. Of the serum enzymes which I have evaluated, serum aspartate aminotransferase activity (AST, formerly SGOT) is the one most consistently elevated in cows with fatty livers. This enzyme is not, however, liver specific and other conditions, such as ischemic or traumatic muscle damage, both common in fresh dairy cows, can cause elevations in serum AST activity. Sorbitol dehydrogenase (SDH), a liver specific enzyme in cattle, is not as sensitive to fatty infiltration of the liver as is AST. Some practitioners use serum total lactate dehydrogenase activity in the evaluation of suspected fatty liver cases. I have not evaluated this enzyme, but some Italian investigators have. They indicate that it is frequently elevated in cows with fatty livers. Serum total LDH activity is the combined activity of five isoenzymes, including those from skeletal muscle, cardiac muscle and red blood cells. For this reason care must be taken in the interpretation of serum LDH activity.

5) BSP clearance

BSP is a dye that is eliminated from the body via bile secretion. A reduced rate of BSP clearance is an indication of impaired liver function. This is a sensitive test that will detect biliary obstruction as well as abnormalities in liver blood flow or hepatocyte

function. Mild elevations in BSP clearance times are usually observed in fresh dairy cows, regardless of liver fat content. Extremely prolonged BSP clearance does not occur in cows with fatty livers until there is a marked degree of fat infiltration.

6) Liver biopsy

Liver biopsy is probably the only way to definitively diagnose fatty liver. Histopathological evaluation of liver biopsy samples can be helpful, but the quantitative evaluation of liver fat content is too subjective to be of value in many cases. Recently, a quick field test for the estimation of fat content in liver biopsies has been developed. The method for doing this test is in the attached reprint. Anyone wanting to use this test can obtain the appropriate copper sulfate solutions by requesting them from me. Write to:

Dr. Thomas Herdt
Veterinary Clinical Center
Michigan State University
East Lansing, Michigan 48823

Care must be taken in the interpretation of liver fat contents. Many cows develop liver fat concentrations as high as 25% with no outward sighs of illness. At this time, it is difficult to determine the clinical significance of liver fat concentrations in the range of 13 to 25%. Clinically ill cows with liver fat concentrations in this range should probably be treated

for fatty liver, but it must not be assumed that the fatty liver is the only condition preventing their recovery. Other diseases may be causing the degree of negative energy balance necessary to create the fatty liver. Cows with liver fat concentrations above 35% (liver floats in water) can be expected to have severely reduced liver function, as judged by BSP clearance. These cows have a poor prognosis and should be treated vigorously.

RUMEN PROTEIN METABOLISM AND ITS POTENTIAL INFLUENCE ON ANIMAL HEALTH

Nearly everyone familiar with ruminant nutrition is aware that rumen microbes can synthesize protein from ammonia nitrogen. This is the basis for nonprotein-nitrogen feeding, a practice that has been used for approximately 30 years. Although the qualitative aspects of this phenomenon have been known for along time, some of the quantitative aspects have been more recently illuminated and may not be well known to some veterinarians in cattle practice. Some of this information may help to resolve the frequently asked question as to whether high protein diets are detrimental to health and fertility in dairy cows.

A. Ruminal protein degradation

Some of the protein entering the rumen is rapidly acted upon by bacterial proteases to yield small peptides and amino acids. Some of these are directly taken up by growing bacteria and converted into bacterial protein. A much larger portion of the peptides and amino acids are further degraded into ammonia and ketoacids. Ketoacids are the carbon skeletons remaining from the amino acids after the nitrogen is removed. The portion of protein not degraded in the rumen passes on into the abomasum and is digested there and in the intestine in a manner similar to the monogastric. The factors regulating the relative proportions of protein that are degraded or not degraded are of central importance to our discussion.

Most of the bacterial proteases are located outside the bacterial cells, either on their surfaces, or free in solution. The activity of these enzymes is not regulated. Therefore the degradation of protein proceeds at a rate that is not greatly dependent on the status of the microbial mass of the rumen. The growth rate of the bacteria has a relatively small effect on the rate of protein degradation. It follows that the microbial need for ammonia is not tied to the rate of ammonia generation. Protein degradation and ammonia generation proceed at approximately the same rate whether or not the bacteria need or can use the nitrogen for microbial protein synthesis.

The major factors regulating the rate and extent of protein degradation are the nature of the protein and the amount of time it spends in the rumen. One of the factors associated with the nature of the protein is solubility.

Solubility is frequently associated with degradability, more soluble proteins being more rapidly degraded. Since protein solubility is easily measured, some ration balancing systems use it as an approximation of degradability. This may be a practically useful, but it should be remembered that solubility does not always predict degradability. Besides the nature of the protein, the other determinant of protein degradability is the amount of time it spends in the rumen. This is not only determined by the protein itself, but also by the nature of the rest of the diet and the rate of intake.

Thus, we can see that rumen ammonia generation (protein degradation) and ammonia utilization (bacterial growth) are disjointed; one is not tied to the other. For efficient protein utilization by the ruminant animal, bacterial growth must be matched to ammonia generation. Let us now examine the factors regulating bacterial growth and ammonia utilization.

B. Ruminal protein synthesis

The rate of ammonia utilization is essentially dependent on bacterial growth rate, which is in turn dependent on the energy supply and the rate at which bacteria are removed from the rumen. The energy supply is dependent on the energy density of the ration and intake. The rate of bacterial removal from the rumen is dependent on feed intake and other factors associated with the diet. High rates of bacterial removal are associated with rapid bacterial growth and large feed intakes.

Mismatches between rumen ammonia generation and bacterial growth rates can occur in two ways. First, ammonia may not be generated fast enough to meet the bacterial demand. In this case bacterial growth will be suppressed and therefore rumen digestion will be less efficient. In this case fiber digestion and energy utilization could be impaired. Second, ammonia may be generated faster than it can be incorporated into bacterial protein. In this case the excess ammonia will be absorbed into the portal blood and converted to urea in the liver.

C. Matching rumen protein metabolism to animal needs

From the above discussion it can be seen that high levels of energy intake are conducive to efficient rumen protein utilization. However, it is at this point that the quantitative aspects of the process must be introduced. Since there is a limit to intake, it follows that there must be a limit to bacterial protein synthesis. At the maximum intakes achieved by most dairy cows, the amount of crude protein synthesized by the rumen bacteria is in the range of six pounds. High producing dairy cows at peak lactation may require 8 to 10 pounds or more of crude protein. The difference between the 6 pounds of bacterial protein and the 8 to 10 pounds of crude protein required must be made up by nondegraded, or rumen-bypass protein. To match rumen protein metabolism to animal needs, during times of high protein requirements, the proportion of nondegraded protein must vary

inversely with energy intake. In practical terms, this means that cows in early lactation, when milk yields are high but before appetite has peaked, a high proportion of nondegradable protein is necessary to meet protein requirements without creating a large excess of rumen ammonia.

D. Effects of excess rumen ammonia on animal health

Much controversy centers on the relationship of rumen ammonia, and thus dietary protein, to animal health. A large body of evidence exists indicating that high levels of protein feeding, or proper use of nonprotein-nitrogen, are not detrimental to the health or fertility of dairy cows. However, there is an accumulating number of reports which incriminate high rumen ammonia concentration as a negative factor for dairy cow health and fertility. Since high rumen ammonia concentrations reflect a waste of dietary protein, and since there is some indication that they may be associated with reduced fertility and ill health, I believe it behooves us as veterinarians to advise our clients to take steps to match rumen protein degradation to bacterial protein synthesis.

E. How can by-pass protein be used to match ammonia production and microbial growth rates.

The major mismatch problem in dairy rations occurs with high protein diets feed in early lactation. The problem arises because cows during this period have high protein requirements, but have not reached peak dry matter intake. If the diet is

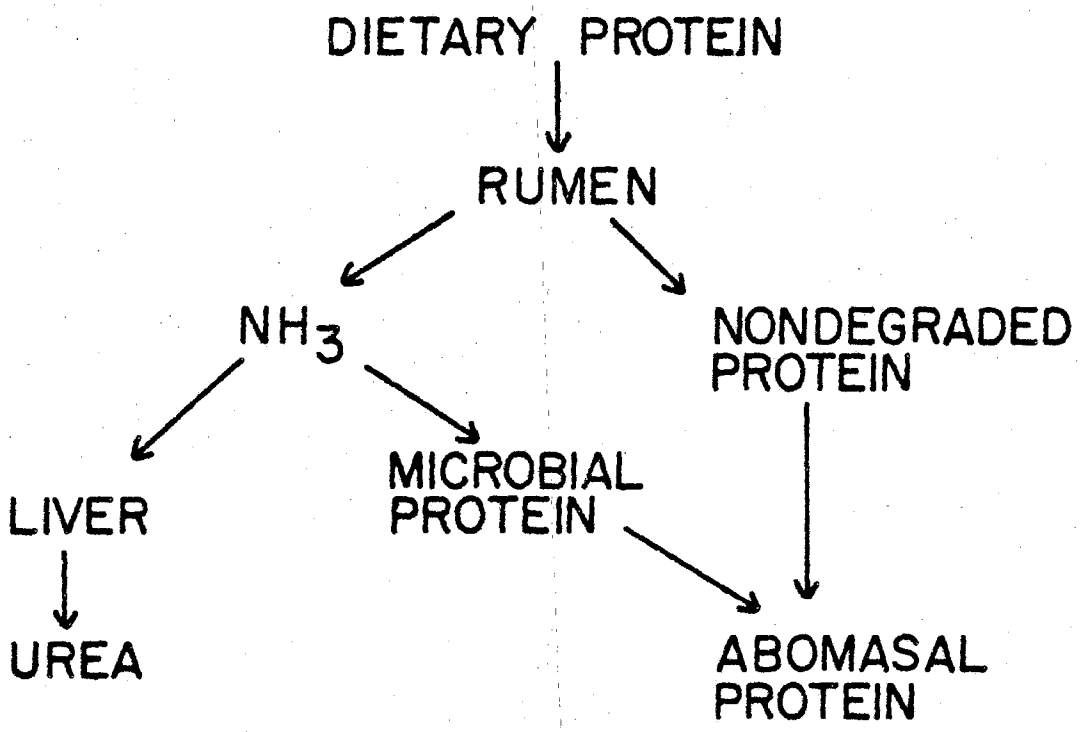
balanced for protein during this time, the protein concentration must be high due to the high absolute requirement and the relatively low total intake. The relatively low total intake means that energy delivery to the rumen is not at maximum and thus neither is the bacterial growth rate. There is relatively too much ammonia being produced compared to the amount of energy being delivered to the rumen. If the proportion of protein that is degraded in the rumen is reduced two beneficial effects will result. First, the amount of ammonia produced will be reduced and thus more nearly match the bacterial growth rate. Second, more total protein will reach the lower digestive tract because less is wasted through excess ammonia production. Protein sources with a high proportion of non-rumen-degradable protein are referred to as "by-pass" protein sources. Such products as corn gluten meal and brewers grains are popular sources of by-pass protein for dairy cows.

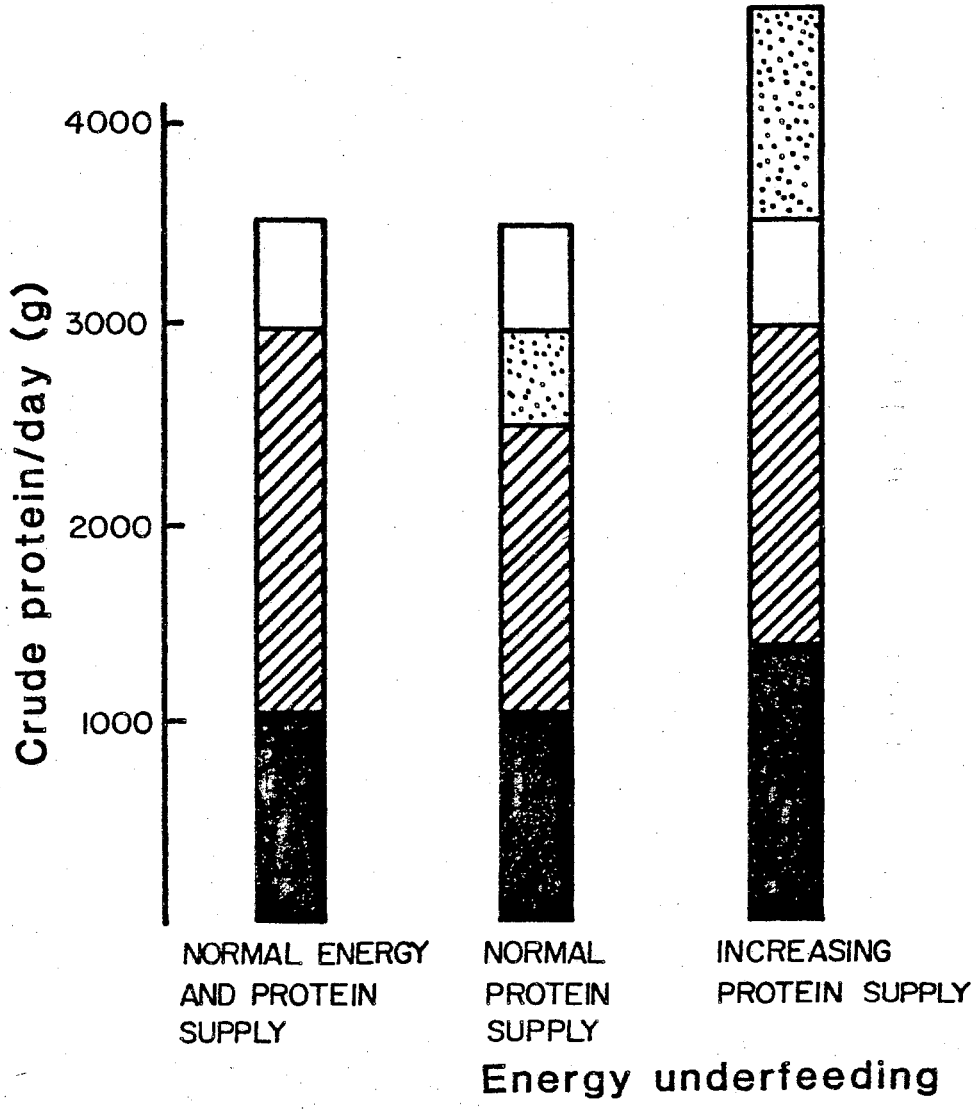
Various systems have been developed in attempt to quantitatively predict when by-pass protein sources will be beneficial to cows and to estimate how much of the total protein should come from by-pass sources. All of these systems have draw backs, the most frequent one being the inability to predict the proportion of protein that will be degraded. As a rule of thumb, cows producing over 60 pounds of milk and receiving more than 13% of their ration as crude protein may benefit from by-pass protein feeding. In these cases up to one half of the total protein may come from by-pass sources. Cows receiving less than a 13% crude protein ration should not

receive by-pass proteins because this may result in rumen ammonia concentrations that are too low to support optimal rumen fermentation.

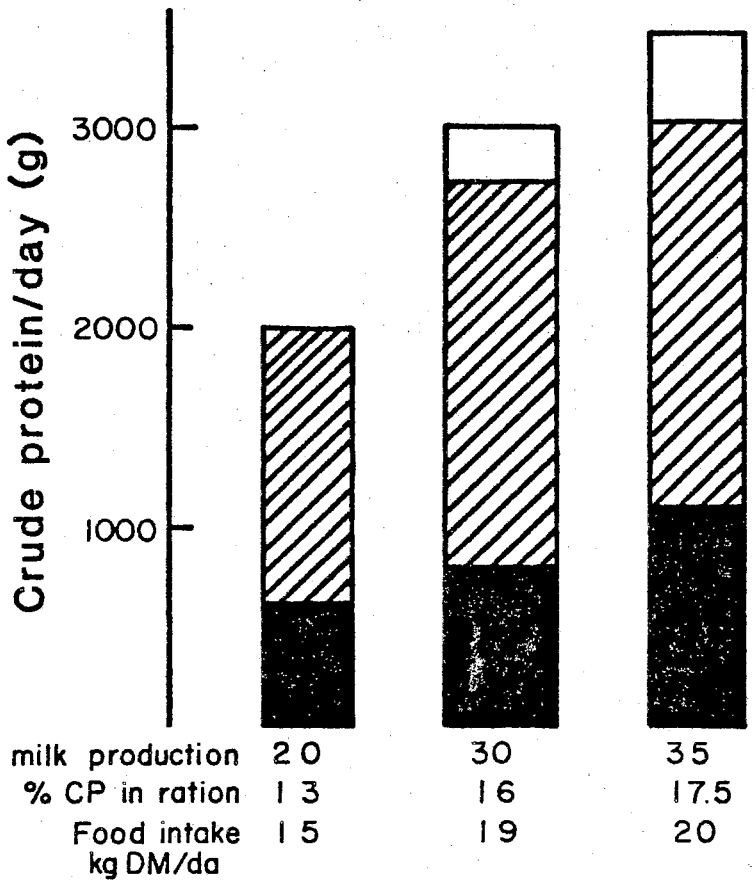
F. The use of blood (or serum) urea nitrogen (BUN) in the assessment of ruminant protein nutrition.

Serum urea nitrogen concentrations in healthy cattle vary in proportion to protein intake. The values probably reflect the rumen ammonia concentrations, since excess ammonia produced in the rumen is absorbed into the portal blood and rapidly converted to urea in the liver. Thus, BUN concentrations may be an indirect way to monitor the matching of rumen ammonia production and utilization. Cows on well balanced diets will have BUN concentrations between 10 and 17 mg/dl. Values below 10 mg/dl usually indicate insufficient total protein, but in some instances may indicate insufficient degradable protein (too much by-pass). Values above 18 or 19 mg/dl are associated with high protein feeding and may indicate excessive rumen ammonia concentrations. In these cases subclinical ammonia toxicity may be occurring and an increase in the proportion of by-pass protein sources being fed is indicated.


DIETARY PROTEIN
RUMEN
NH_3
NONDEGRADED PROTEIN
LIVER
MICROBIAL PROTEIN
UREA
ABOMASAL PROTEIN



H. HAGEMEISTER, ET AL, 1980



H. HAGEMEISTER, ET AL, 1980

Reprinted from the JOURNAL OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, Vol. 182, No. 9. Pages 953-955.

Test for estimation of bovine hepatic lipid content

T. H. Herdt, DVM, MS; L. Goeders, DVM; J. S. Liesman; R. S. Emery, PhD

SUMMARY

The lipid content of bovine liver was found to be highly correlated with its specific gravity. This observation was used as the basis for a clinical test to estimate the liver lipid content in dairy cows. The test consisted of submerging needle biopsy specimens of liver into water and copper sulfate solutions with specific gravities of 1.025 and 1.055. On the basis of buoyancy in these liquids, bovine liver samples were classified accurately as containing greater than 34% lipid, greater than 25% lipid, greater than 13% lipid, or less than 13% lipid.

HEPATIC LIPIDOSIS is a prevalent condition in periparturient dairy cows.[1,a] When severe, it appears to result in clinical disease,[2] but clinical diagnosis is difficult because signs are vague and nonspecific, and perhaps more important, because concurrent disease is a frequent complication. In this report, we describe a rapid and practical, semiquantitative test for bovine hepatic lipid content. The test is suitable for field application in the diagnosis of bovine hepatic lipidosis.

Materials and Methods

Liver biopsy specimens weighing 10 to 15 g were taken from 49 dairy cows admitted to the Michigan State University Veterinary Clinical Center for surgical correction of displaced abomasum. The total lipid content of each specimen was determined on a wet-weight basis by the method of Hara and Radin.[3] Subsamples for buoyancy testing were taken from biopsy specimens with selected fat concentrations. A needle biopsy instrument was used to take the subsamples.[b] Additional liver specimens taken percutaneously from 16 dairy cows prior to slaughter also were tested for total lipid content, as described.[3]

The buoyancy of the needle biopsy samples was observed immediately after the samples were submerged into copper sulfate solutions or water. When it was necessary to measure the specific gravity of a sample as accurately as possible, subsamples repeatedly were obtained and submerged into copper sulfate solutions. The concentration of each solution was adjusted after each subsampling, until the solution of the lowest specific gravity that would float the sample was determined. The specific gravity of the sample was then assumed to approximate that of the solution. When it was necessary only to determine the specific gravity of a sample within a range, subsamples were submerged into each of 3 liquids: copper sulfate solutions with specific gravities of 1.025 and 1.055 and water. The buoyancy of each sample in each liquid was noted.

Copper sulfate solutions were made with hydrated copper sulfate[c] and distilled, deionized water. Appropriate tables[4] were used to determine the copper sulfate concentration necessary to make aqueous solutions of the specific gravities desired. The specific gravity of solutions made according to these tables usually varied slightly from the expected value, so small adjustments were made when necessary by the addition of water or a concentrated copper sulfate solution. The temperature of the solutions was maintained within a range of 20 to 23 C. The specific gravity of the solutions was determined from tables[4] that relate refractive index to specific gravity. The refractive index was read directly from a refractometer.[d]

Technique

A site is selected on the right side of the cow, at the point where the 10th intercostal space intersects an imaginary line from the tuber coxa to the olecranon. A 10-cm square area at this site, including the adjacent 9th intercostal space, is prepared as if for surgery. A stab incision is made through the skin at the intersection of the imaginary line and the 10th intercostal space. A biopsy needle is then inserted in a direction perpendicular to the median plane. The instrument is passed through the pleural cavity and diaphragm and into the liver, where a specimen is obtained. A specially designed cutting biopsy instrument is recommended.[b] Occasionally, liver will not be

From the Department of Large Animal Surgery and Medicine, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824.

Published as Michigan State University Agricultural Experimental Station Journal Article No. 10641.

[a]Jasper DE: Hepatic changes in the bovine during pregnancy and ketosis. PhD thesis, University of Minnesota, St Paul, Minn, 1947.

[b]Tru-Cut 6-in biopsy instrument, Travenol Laboratories, Deerfield, Ill.

[c]Cupric sulfate, pentahydrate, $CuSO_4 \cdot 5 H_2O$, Mallinckrodt, Paris, Ky.

[d]TS Meter, American Optical Co, Buffalo, NY.

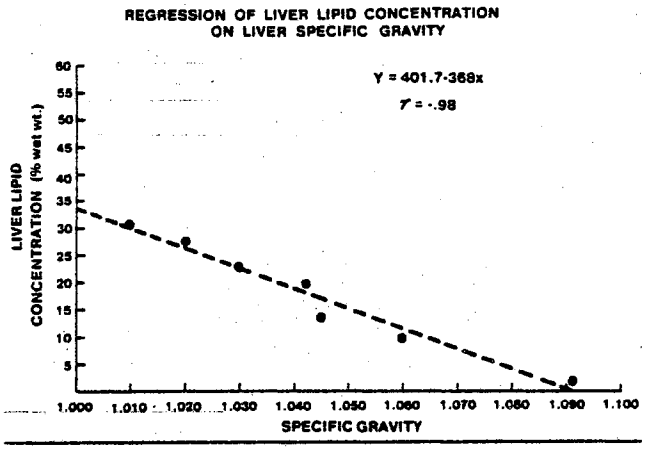


Fig 1—Regression of liver lipid concentration on liver specific gravity.

TABLE 1—Lipid concentration and specific gravity range of selected bovine liver samples

Lipid concentration (%)	Buoyancy according to specific gravity of solution		
	1.000	1.025	1.055
42	f	f	f
39	f	f	f
30	s	f	f
28	s	f	f
27	s	f	f
27	s	f	f
25	s	f	f
21	s	s	f
19	s	s	f
16	s	s	f
12	s	s	s
12	s	s	s
10	s	s	s
7	s	s	s

f = floated; s = sank.

procured from pregnant cows from the site described. In such a case, the needle should be reinserted in the 9th intercostal space, 2 to 4 cm above the line from the tuber coxa to the olecranon.

A piece of liver 2 mm in diameter, 2 cm long, and usually weighing approximately 50 mg is obtained by this procedure. The tissue is divided into 3 pieces of equal size, and each piece is placed into 1 of 3 vials containing either water or copper sulfate solution. The specific gravity of the 3 liquids is 1.000, 1.025, or 1.055. The specimen is submerged in the solution and its buoyancy noted.

Results

Liver lipid concentration and specific gravity were closely correlated ($r = -0.98$). The regression of liver lipid concentration on specific gravity is presented in Figure 1. The regression appears linear in the specific gravity range between 1.000 and 1.100. The buoyancy of 14 liver specimens, with a broad range of lipid contents, in solutions with specific gravities of 1.000, 1.025, and 1.055 is shown in Table 1. Each sample floated or sank in accordance with its lipid content, as predicted by the regression.

In all 16 of the preslaughter cows, the liver specimens sank in the 1.055 specific gravity solution. The liver lipid concentrations were determined in 8 of the cows. They were all less than 4%. At slaughter, evidence of hemorrhage was not observed on the surface of the liver. A small hemorrhagic area roughly equal to the size of the biopsy specimen was observed in the liver parenchyma.

The biopsy technique described has been used in this laboratory approximately 200 times (often in conjunction with other methods of liver lipid determination). Liver tissue could not be obtained in 15 cases. There have been no adverse consequences.

Discussion

The number of copper sulfate solutions and their specific gravities may be selected on the basis of accuracy desired for the estimation and the range of liver lipid concentrations to be detected. It is suggested that, for routine clinical diagnosis, 3 solutions be used with specific gravities of 1.000, 1.025, and 1.055. Depending on buoyancy or lack of buoyancy in these solutions, the liver samples may be divided into 4 lipid concentration categories. Those that float in all 3 solutions are greater than 34% lipid. Those that sink in water but float in 1.025 and 1.055 specific gravity solutions are less than 34% lipid but greater than 25% lipid, whereas those that float only in 1.055 specific gravity solutions are less than 25% but greater than 13% lipid. Samples that sink in all 3 solutions are less than 13% lipid. Limited research[5] has indicated that cows with liver lipid concentrations above 34% are severely affected and may be expected to have clinical manifestations of hepatic insufficiency. Those with liver fat concentrations between 34% and 25% are moderately affected and might be suffering from hepatic insufficiency. Those with liver lipid concentrations between 25% and 13% are mildly affected. The clinical significance of liver lipid concentrations in the mildly affected range is difficult to assess. Many postpartum dairy cows develop this degree of hepatic lipidosis without apparent clinical disease.[1] Liver lipid concentrations below 13% probably are inconsequential.

Several precautions and limitations must be kept in mind when using this test. If the instrument suggested is used, the manufacturers directions should be followed carefully. It is imperative that the stylet remain stationary and the needle advanced as the sample is taken. If the needle remains stationary and the stylet is withdrawn, tissue will not be obtained.

The liver specimen should be submerged rather than just dropped into the solution, thus eliminating any effect of surface tension on buoyancy. The buoyancy should be noted immediately after the tissue is placed in the solution. Copper sulfate is used to coagulate surface protein and reduce the flow of water between the solution and the sample. This delays but does not prevent water moving into or out of (depending on the concentration of the solution) the tissue and altering its specific gravity, thus the need for immediacy. Also, care must be taken to prevent drying of the specimen prior to buoyancy testing. Small specimens can dry rapidly, which would alter their specific gravity.

The specific gravity scale on clinical refractome-

ters cannot be used to determine the specific gravity of copper sulfate solutions. The refractive index of the solution at 20 C must first be determined. The specific gravity can then be determined from tables relating the specific gravity of copper sulfate solutions to their refractive index.

References

1. Reid IM: Incidence and severity of fatty liver in dairy cows. *Vet Rec* 107:281–284, 1980.

2. Morrow DA, Hillman D, Dade AW, Kitchen H: Clinical investigation of a dairy herd with the fat cow syndrome. *J Am Vet Med Assoc* 174:161–167, 1979.

3. Hara A, Radin NS: Lipid extraction of tissue with a low-toxicity solvent. *Anal Biochem* 90:420–426, 1978.

4. Weast RC, Astle MJ (eds): CRC Handbook of chemistry and physics, Ed 62nd. Boca Raton, Fla, CRC Press Inc, 1981, p D-206.

5. Herdt TH, Gerloff BJ, Liesman JS, Emery RS: Hepatic lipidosis and liver function in 49 cows with displaced abomasums. XIIth World Congress on Diseases of Cattle, Proceedings. Netherlands, 522–526, 1982.

Don't Forget the Youngstock. Keep Eyes, Ears and Nose Sharp

Andrew P. Johnson, DVM
Valley Veterinary Clinic, S.C.
Seymour, WI 54165

As practicing dairy veterinarians we often get quite involved in herd health programs. The only problem is we often only consider the adult cows in our programs. There is a very important group of animals on everyone's farm called the young stock or forgotten flock. The young stock is a very important group to a herd health program as well because they are the "future" herd. In every herd you visit, a trip to the young stock area will usually be worth the time spent. By keeping our eyes open (if possible), our noses sniffing (it maybe unpleasant), and our ears sharp we can usually accumulate a lot of good and valuable information.

The findings in our practice have been that most dairymen are very receptive to total herd health which includes the young stock. By working with the young stock we can increase his profits and productivity as well as our own. Now more than ever as the economy tightens, it is so important that the young stock survive and remain healthy.

Our program looks at many different areas of young stock programs. We look at environment and management, ventilation, nutrition, genetics and disease control. I plan to break these down and discuss them individually.

ENVIRONMENT

This is probably one of the most important parts of a herd

health program. Many calves often start off behind the eight ball and never have a chance. We educate and educate to convince dairymen that environment starts at birth and goes from there. We try to avoid birth in gutters and dirty lots. We stress calving pens that are clean and accessible. The first 48 hours are as critical as the next year when it comes to healthy calves. A good manager will dip the naval, identify the calf and get hand-milked colostrum into the calf as soon after birth as possible. This is a good time to use specific vaccines if necessary for that particular client or discuss dry cow vaccines as part of the program.

We prefer calf hutches from this point on even though it's 50 degrees below zero. But it is so important to preach management again -- proper location, proper bedding and crushed rock underneath for drainage. We try to eliminate calves between cows and overcrowded box stalls. We like to see the calves raised by groups according to size throughout their struggle to become milk cows.

VENTILATION

Improper ventilation probably kills more calves per year than pneumonia and scours. Your eyes, nose and ears will really be of value when evaluating a ventilation system. Especially if you have glasses, you have a real advantage in judging the system in the winter time. Proper ventilation is a big part of our program because we feel proper ventilation will eliminate most respiratory diseases. We look at air exchange and make sure we replace the air we remove. We also like to eliminate the bad air

from the floor by putting ducts over wall fans, especially in young calves. Our success rate has been higher with less coughing calves and healthier calves. It is necessary for air to exchange at all times, but we must have the ability to control this exchange. Humidity is also so important and can be controlled through proper ventilation.

NUTRITION

This is an area of major consequence and is an area that we can really help control. Feed salesmen are too busy bragging about how much milk they can get out of the cows to worry about the young stock. But through proper nutrition we can get the calves to fully reach their genetic potentials and reach breeding weight sooner, therefore enter the milking herd sooner.

We must start with the calves and provide them with a proper calf feed, not cow feed. We must meet their specific needs and keep them growing. We must educate the dairymen to use only the best milk replacers or whole milk. When problems occur, be sure to check over the management and be sure proper amounts are being mixed and fed, that the water is proper temperature so all the high energy fat isn't on the sides of the pail or on the bottom because it didn't dissolve.

As the heifers are weaned and should be on grain, it is important to keep challenging the growth via proper nutrition. Now is the time to analyze the roughage and balance a growing ration with proper amounts of the right roughages. Look at the heifers. Are they short and heavy from too much energy, or tall and thin from lots of protein and little energy? We want dairy

replacements, not beefers. It is so important to eye the dairyman's heifers for him because we can compare them to so many others as well as seeing them less often so that we can comment. Heifers with unbalanced nutrition will fail reproductively as well. Fat heifers breed poorer than heifers in proper condition, just as underfed and thin heifers breed poorly also.

We have found that we can really see results and get rewarded through proper nutrition. This has been an excellent place for us to get coccidiostats into the feed. This may have been one of the most significant changes we have made in our calf programs. A constant supply of coccidiostats from birth to six months of age has been very rewarding. The calves are much more thrifty and gain weight much faster.

The second most important change we have made in our calf programs is looking at the water. The availability of water to young stock is often so restricted that it actually limits growth. We have repeatedly replaced adult drinking cups with fountain type waterers and seen tremendous results. The calves look brighter and healthier. Their feed intake increases dramatically. Some trials we have done indicate calves get just enough water and survive with most water cups, but not as much water as they truly need. Water is a factor in dry matter intake. The less water that is available, the less feed consumed. Calves learn to adapt to most situations, but this is an unnecessary adaptation. When we really think about it, the calf comes off a primarily liquid diet and is weaned and put on a limited liquid diet. We feel the results speak for themselves.

GENETICS

Don't be afraid to get involved here either. Look at his animals and observe. Spell out his herd's weaknesses. This advice from an independent eye can help him to breed better. Let's not sit back and watch him milk his mistakes, but instead let's help him develop a genetically superior herd. It is amazing how much different cows can look if you see them everyday or just monthly. He pays us for our advice, so let's give him the proper advice he needs.

DISEASE CONTROL

I am sure everyone here today has an ideal young stock prevention program. This is what makes it so hard to standardize the industry. I am sure our beliefs will differ, but we feel it is absolutely necessary to cover the basics for every herd and use the specifics only in those herds that warrant their use.

At 2 to 3 months of age we vaccinate all herds for brucellosis and remove any extra teats. This is a very convenient time to deworm the calves as well. Since the calf is caught up to be vaccinated, why not take the time to deworm it as well. Now with the new products that are available we can also take care of ecto-parasites.

BVD-IBR-PI3-LEPTO 5 modified live vaccine is used on all of our young stock at 4 months of age. Current research indicates that many calves have lost material passive immunity by 60-90 days of age. Even if passive immunity is present, the shot is still beneficial. We then re-booster the 8-way shot at 10 to 12 months of age. We feel this 8-way shot covers 95% of the dairy

farms and have had tremendous success with the product. Only in 5% of our herds are hemophilus, pasturella or clostridial vaccines indicated.

In some herds we have elected to re-booster the 8-way shot one month before breeding along with deworming the animal as well. This is usually done on known problem herds.

In disease control it is also well worth the time spent to educate in proper treatment procedures as well. All herd health clients should own a thermometer and learn to use and read it. Observation is necessary and probably the key to successful treatment of animals. Early detection along with a proper game plan of treatment is helpful. Outline proper treatments so they can at least take the proper first step. Educate them to treat long enough and with adequate levels of antibiotics. Educate them as to when to call for help or reassess their therapy in case a drug change is necessary.

It is our belief that the young stock must be included in our herd health programs. The results are rewarding not only for the dairyman, but for yourself. Now more than ever the dairyman needs help to become efficient so they can survive. It is through a total herd health program that this efficiency can be obtained. The economic returns are higher for the dairyman and a good young stock program is economical for you as well.

PILIGUARD[R] PINKEYE
(M. bovis Bacterin)

T. Wolff, D.V.M.

INTRODUCTION

Laboratory and field testing support use of a new piliated bacterin, PILIGUARD[R] Pinkeye, for the control and prevention of pinkeye (IBK) caused by Moraxella bovis. The satisfactory performance of the bacterin under rigorous testing is a tribute to new pili technology, pioneered by Dr. C. Brinton and associates at the University of Pittsburgh and developed in conjunction with Schering's Elkhorn Research Center, Elkhorn, Nebraska.

THE PRODUCT

PILIGUARD Pinkeye is a highly piliated, formalin-inactivated, aluminum hydroxide adjuvanted whole cell bacterin, with gentamicin as a preservative. The product contains M.bovis strains EPP 63 and FLA 64, the two serotypes of M.bovis expressing the pili types most frequently isolated from cattle afflicted with pinkeye. Additional research indicates that EPP 63 and FLA 64 confer a broad range of cross protection against other M.bovis strains as well. Pili are long, thin, nonflagellar protein appendages which allow attachment of M.bovis to the corneal epithelium with subsequent colonization and manifestation of clinical disease. Vaccination with a highly piliated product such as PILIGUARD Pinkeye induces pili antibodies which block attachment of M.bovis, thereby preventing damage to the eye.

Label recommendations state that cattle less than 300 lbs should receive 2 ml of bacterin subcutaneously, with a booster 1-4 months later. Cattle over 300 lbs should be given 3 ml of bacterin subcutaneously, and boostered in 1-4 months.

IMMUNOGENICITY TESTS

Two trials were completed, utilizing laboratory challenge with 2 major strains of M.bovis, EPP 63 and FLA 64. In the first trial, 40 calves, 175-300 lbs, were divided into 2 groups. All calves were negative for clinical symptoms of IBK at the initiation of the study. Twenty calves received 2 subcutaneous 3 ml doses of the bacterin at 28 day intervals, and 20 calves were held as controls. Challenge was 14-17 days post second vaccination. Prior to challenge, each calf's eyes were irradiated with ultraviolet light, 20 minutes daily, for 4 consecutive days. Ten animals from each group of calves were challenged with M.bovis strain EPP (0.5 ml inoculum containing approximately 5×10^8 organisms in a $>$ 80% piliated phase) or FLA (0.5 ml inoculum containing 3×10^8 organisms in a $>$ 80% piliated phase) by placing the organisms directly into the conjunctival sac. Each calf was observed daily for 7 days postchallenge for clinical signs of IBK. Eye lesions were scored as follows:

0 = Normal

1 = Lacrimation and photophobia with slight clouding of cornea

2 = Clouding of cornea plus a small ulcer (1-2 mm)

3 = A larger ulcer ($\geq$ 3 mm)

The eye lesion scores for each group of calves were averaged to provide a Mean Clinical Score for each group. Blood samples were drawn from vaccinated calves on the day of initial vaccination, booster vaccination, and challenge, and from control calves on the day of challenge. The ELISA method was used to detect antibody levels for M. bovis EPP and FLA pili. Results of Trial 1 are shown in Tables 1-3.

TABLE 1

EPP/FLA CHALLENGE RESULTS

Group	EPP # IBK/total	EPP % IBK	FLA # IBK/total	FLA % IBK
vaccinates	2/10	20*	2/10	20**
controls	6/10	60	8/10	80

* 20% IBK in vaccinates is 66.6% less than 60% IBK in controls.

** 20% IBK in vaccinates is 75% less than 80% IBK in controls.

TABLE 2

EPP/FLA CHALLENGE: MEAN CLINICAL SCORE

Group	EPP Mean Score	FLA Mean Score
vaccinates	.6*	.8*
controls	1.7	2.3

* Eye lesions were as severe as those seen in control group, i.e. vaccination reduced incidence but not severity of disease.

TABLE 3

MEAN SERUM EPP AND FLA PILI ANTIBODY TITERS

Group	EPP Prevaccination	EPP Prebooster	EPP Day of Challenge	FLA Prevaccination	FLA Prebooster	FLA Day of Challenge
vacc.	< 2.4	13.5	73.6	5.8	17.6	107.2
controls	ND	ND	< 2	ND	ND	< 3.8

ND = not determined.

In Trial 2, 59 calves, 175-300 lbs, were divided into 2 groups. Twenty-nine calves were vaccinated with 2 subcutaneous 3 ml doses of bacterin at 28 day intervals, and 30 calves were held as controls. Both groups of calves were challenged 14-17 days postbooster (after prechallenge exposure to ultraviolet light as described in Trial 1). The challenge in this trial utilized strain EPP (0.5 ml inoculum containing approximately 3×10^8 organisms in a $>80\%$ piliated phase, placed directly in each eye). Each calf was observed daily for 7 days postchallenge for clinical signs of IBK, and blood samples were collected from both vaccinated and control calves on the day of initial vaccination, booster vaccination, and challenge. Results of Trial 2 are shown in Tables 4-6.

TABLE 4

EPP CHALLENGE RESULTS

Group	# IBK/total	% IBK
vaccinates	8/29	27.6*
controls	18/30	60

* 27.6% IBK in vaccinates is 54% less than 60% IBK in controls.

TABLE 5

EPP CHALLENGE: MEAN CLINICAL SCORE

Group	Score
vaccinates	.62*
controls	1.57

* Vaccination reduced incidence but not severity of disease.

TABLE 6

MEAN SERUM EPP PILUS ANTIBODY TITER

Group	Prevaccination	Prebooster	Day of Challenge
vaccinates	$\leq$ 58.7	131.2	468.9
controls	76.4	138.6	82.8

SAFETY

A total of 617 beef, Jersey, and Holstein calves ranging in weight from 125-500 lbs were vaccinated subcutaneously with the bacterin. NONE of the calves showed adverse reaction. The results of this study are summarized in Table 7. A total of 1,505 beef, Jersey, and Holstein calves weighing 150-400 lbs were vaccinated intramuscularly with the bacterin. Seventeen calves exhibited delayed hypersensitivity type reactions (frothing at the mouth, difficult respiration, weakness) within 2 hours post injection. Two of the seventeen calves died. All of the calves which reacted to intramuscular vaccination were of dairy breeds and all were under 250 lbs. The results of this study are summarized in Table 8. The product, PILIGUARD Pinkeye, is recommended for subcutaneous use only in all breeds.

TABLE 7

SUBCUTANEOUS VACCINATION

Breed	# vaccinated	# reactions	% reactions
Beef	172	0	0
Holstein	362	0	0
Jersey	83	0	0

TABLE 8

INTRAMUSCULAR VACCINATION

Breed	# vaccinated	# reactions	% reactions*
Beef	440	0	0
Holstein	814	11	1.3
Jersey	251	6	2.4

* Primarily delayed hypersensitivity. Two calves died.

FIELD SAFETY/EFFICACY

Florida:

One hundred sixty-five Holstein calves, 3-6 months of age, were vaccinated twice with 2-3 ml bacterin subcutaneously during November and December 1982. The time interval between vaccinations was 28 days. No adverse reactions were seen. The calves were pastured in 3 fenced areas with fence contact between pastures, and from January through March 1983, a total of 55 non-vaccinated calves purchased from out-of-state were randomly added to the 3 groups of vaccinated calves. In May of 1983, a natural field outbreak of IBK occurred in the 3 pastured groups of calves. NONE of the vaccinated calves came down with IBK while all of the 55 nonvaccinated calves exhibited clinical lesions. The reason for such a high infection rate (100%) in this particular outbreak may have been due to the over-abundance of face flies found with these herds. Continuing observation of these calves throughout the summer months showed that while endemic pinkeye flourished in the area, the 165 vaccinated calves remained free of clinical disease.

Wisconsin:

Four hundred and ten mixed-breed yearlings were involved in this field study. At the time of initial vaccination with the bacterin in August 1983, the herd was experiencing an IBK outbreak of 2-3 weeks duration, and it was estimated that 30% of the animals were affected. An initial subcutaneous 3 ml dose of bacterin was given to 210 head, which had been divided into 3 groups of 40, 80, and 90 animals for easier handling and observation. Vaccination was repeated 28 days later. The remaining 200 head were held as nonvaccinated controls and were pastured 100 yards away from the 3 groups of vaccinated cattle. Within 1 week after the initial vaccination, NO NEW CLINICAL CASES OF IBK were reported in the vaccinates, while the 200 head of nonvaccinates continued to develop new cases. Approximately 3-4 weeks after the booster injection was administered, some of the vaccinates from the group of 90 began to evidence what appeared to be clinical IBK. On closer examination, 11 animals (5% of the total 210 vaccinates) were showing symptoms ranging from severe lacrimation to ulceration and coning of the eyeball. The eyes of all affected animals and 20 normal animals were swabbed for bacterial and viral isolation. Branhamella catarrhalis, a close relative of Neisseria and usually considered to be a nonpathogen, was isolated from 15 of the animals. Viral (IBR/BVD) and Mycoplasma isolation were negative. Moraxella bovis was not isolated from any of the cattle and no new cases were observed.

SUMMARY

Laboratory testing of PILIGUARD Pinkeye utilizing severe challenge with EPP and FLA strains of M. bovis showed reduction in incidence of IBK in vaccinates and marked serum antibody titer rise following booster vaccination. Extensive safety testing showed the bacterin to be safe when administered according to label recommendations. Field testing to date supports use of the product in control and prevention of IBK caused by Moraxella bovis, and in addition suggests that use of the bacterin in the face of an outbreak may be of value.

PROBLEMS OF FIRST CALF DAIRY HEIFERS

Robert H. Keith, D.V.M.

The subject "Problems of First Calf Dairy Heifers" is very broad, and the questions I ask may not have absolute answers. However, I hope that someone, somewhere, will be challenged, and will further study the problems, come up with some solutions, and thus help the dairyman in the future.

I would like to ask a few questions about an average Wisconsin dairy operation. I am using facts and figures for Wisconsin farms, but I believe the answers are quite similar in Minnesota.

Question 1 - What is the normal yearly culling rate of the average herd?

Question 2 - What is the average age of the milking cows in the average herd?

Question 3 - What does it cost to raise a heifer to two years of age?

Question 4 - Of the first calf heifers that calved this year, how many will not be in the milking line next year?

The feeding, housing and management practices of raising dairy heifers have changed greatly during the last several years. In the past, heifers were not pushed to the limit as they are today, and they were housed mainly in small individual stanchions.

Now most heifers are housed in loose housing or free stall situations, and fed at a bunk from silos containing haylage, high moisture corn, and hardly ever get on dirt. On most farms there are an equal number or more heifers than milking cows. These heifers are brought

into the milking string just before or at calving time, and put in a stanchion. Now the problems begin:

1. The heifers do not know how to get up and down in the stanchion.

2. They bruise themselves up badly, both front and back legs.

3. They are not accustomed to being handled.

4. They are fed a completely different ration.

5. They are not accustomed to the temperature and humidity of the milking barn.

6. Many are filthy, and soon udder and teat problems appear.

7. Many are in much pain, with sore feet, stiffness and laminitis.

8. They need to be accepted by their herd mates.

9. Within two to four weeks, these heifers are expected to be milking 60 to 75 pounds of milk. The dairyman has high hopes of having a 20,000 pound heifer in a 305 day lactation period.

10. In addition, the heifer is still growing, and is expected to be bred back within 60 to 75 days or before. Many of these heifers show no heat signs, and at monthly herd check, rectal examinations show very small, inactive ovaries.

11. More energy is needed by the heifer, so it is recommended that she be fed more grain and concentrate. Remember that high producing animals are just a mouthful away from acute laminitis.

12. Hormones are injected to further enhance and improve the breeding program.

13. Because of many of these reasons, a lot of these heifers are culled; they do not meet the standards that have been set. In most cases, only 67% to 75% of these heifers will see their second

lactation.

Now let's take a look at some of the overall conditions that affect the culling rate of heifers. The main cause of culling is due to reproductive causes: 1) calving problems, 2) calving paralysis, 3) lacerated and necrotic lesions of the vulva and vagina, 4) metritis, 5) no heat periods, and 6) breeding problems.

Another major cause of culling involves the mammary system. The following conditions many times end up with the heifer getting mastitis, and her eventual slaughter: 1) blind quarters, 2) udder edema, 3) frozen teats, 4) mammillitis teat problems, and 5) mastitis.

The next most devastating problem that causes culling of the heifer involves feet and legs: 1) swollen and bruised front legs, 2) swollen, bruised and abscessed hocks, 3) stifle and hip dislocations, 4) pain, 5) sole abscesses, 6) stiffness and soreness to the point where the heifer either can't or won't stand, and 7) laminitis. Drs. Allenstein and Greenough will discuss laminitis tomorrow. My major questions concerning laminitis are: a) why are we seeing a great increase of laminitis in our herds, b) where, in the life of the heifer, is the mechanism triggered, and c) how can we prevent laminitis?

I feel that stress on heifers has a direct relationship to most of the problems I have mentioned.

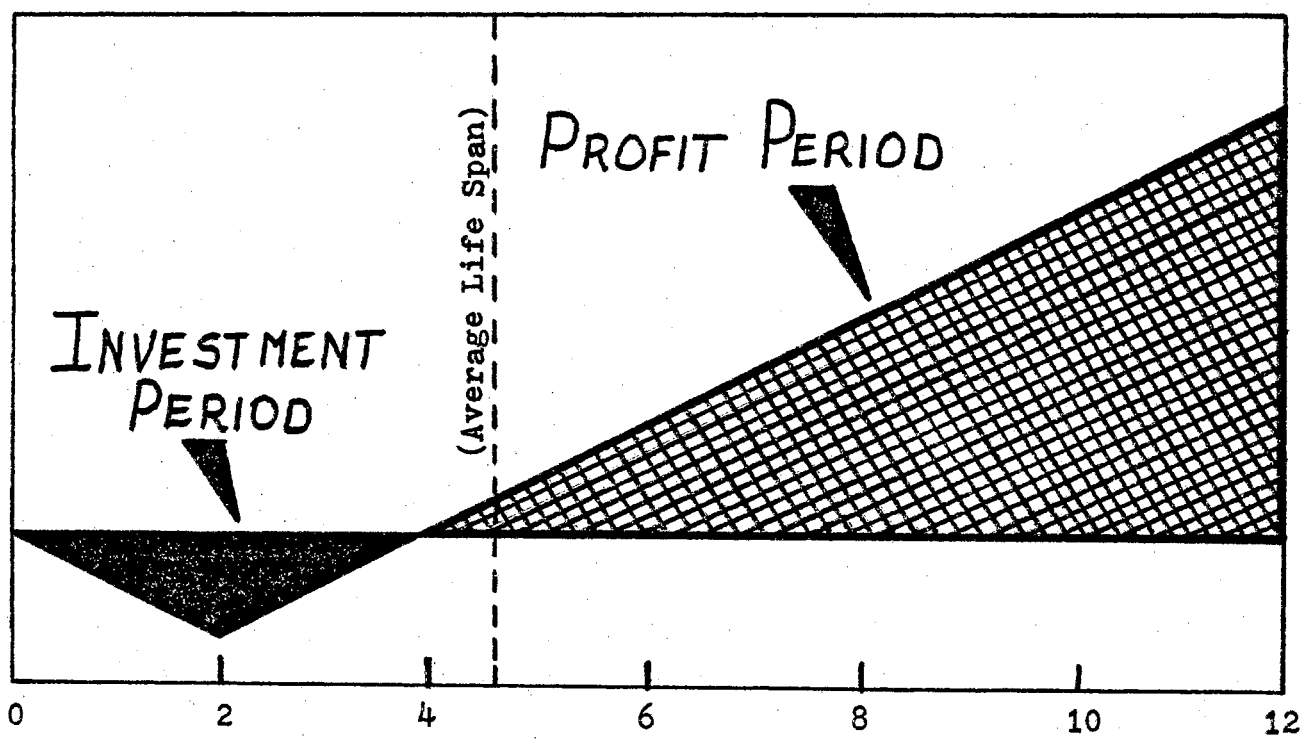
Other problems that have a direct effect on the overall health of the heifer, and that affect the culling rate indirectly are: 1) unbalanced rations, 2) incorrect types and amounts of feed, 3) acidosis in the rumen, 4) displaced abomasums, 5) cecal problems, diarrhea, including Johne's Disease, 7) external and internal

parasites, 8) respiratory problems, and 9) infectious diseases.

Most emphasis for greater profit on the dairy farm has been centered around greater production. I agree with this concept. However, I firmly believe that the most neglected link in the entire chain of the dairy operation is in the management and feeding of replacement heifers.

The following chart shows that it takes two lactations before a dairy cow starts making a profit. If the average age of a milking cow is only 4.3 years, then she is actually only beginning, finally, to make a profit. Part of this is because 25% to 33% of the first calf heifers never reach a second lactation.

PROFIT PER COW
(As Influenced by Longevity)



In conclusion, I would like to challenge universities, researchers, feed companies, veterinary schools and dairymen to do more research, field trials and studies on heifers in the following areas:

1. Ways need to be found to extend the life of the average dairy cow beyond the current 4.3 years of age.

2. More information needs to be determined concerning the proper nutrition and feeding of replacement heifers, and its effect on problems such as laminitis.

3. There needs to be more information available concerning management, housing, and ways of reducing stress in heifers.

4. More attention needs to be paid to the growing heifer.

5. We need answers to the feet and leg problems, reproduction problems and mammary problems, so that culling can be done for better reasons, namely: poor production, poor type, high strung disposition, and old age.

The answers to the questions at the beginning are as follows:
1) 30%, 2) 4.3 years, 3) University of Wisconsin - $1300 to $1400; Hoard's Dairyman - $900 to $1100, and 4) 25% to 33%.

REFERENCES

Bayley, N. D., Parker, J. B., Heidhues, T., Powman, R. D., and Swett, W. W., "Dairy Type: Its Importance in Breeding and Management." Technical Bulletin 1240, U. S. Department of Agriculture, 1960.

Crowley, J., Jorgensen, N., and Howard, T., "Raising Dairy Replacements." University of Wisconsin Extension, 1983.

"Dairy Price Review." Hoard's Dairyman (10 April, 1984): p. 494.

Gibbs, E. Paul J., "Virus Infections of the Skin of the Bovine Teat." Current Veterinary Therapy, Food Animal Practice. Philadelphia: W. B. Saunders Company, 1981, pp. 631-636.

Hutjens, Mike, "Feeding Aspect," Feet and Legs Clinic, University of Illinois, March 13-14, 1984.

Luening, R. R., Klemme, R. M., and Howard, W. T., "Wisconsin Farm Enterprise Budgets." University of Wisconsin Extension, 1983.

McDaniel, Ben T., "Management and Housing Factors Affecting Feet and Leg Soundness in Dairy Cattle." Proceedings, 15th Annual Convention, American Association of Bovine Practitioners, (April 1983): pp. 41-49.

Nocek, James E., "Laminitis: A Mysterious Cause of Lameness." Hoard's Dairyman (25 September, 1982): pp. 1185-1186.

Pinsent, P. J. N., "The Management and Husbandry Aspects of Foot Lameness in Dairy Cattle." The Bovine Practitioner, No. 16, (November 1981): pp. 61-64.

Sigmund, Von H. M., Klee, W., and Schels, H., "Udder-Thigh Dermatitis of Cattle: Epidemiological, Clinical and Bacteriological Investigation." The Bovine Practitioner, No. 18 (November 1983): pp. 18-23.

Weaver, A. D., "The Prevention of Laminitis in Dairy Cattle." The Bovine Practitioner, No. 14 (November 1979): pp. 70-72.

UPDATE ON JOHNE'S DISEASE (PARATUBERCULOSIS)

David M. Sherman, D.V.M., M.S.
Department of Large Animal Clinical Sciences
College of Veterinary Medicine
University of Minnesota

A. INTRODUCTION

1. Johne's disease is becoming a major concern of cattlemen in the U.S. Recent slaughterhouse surveys indicate a high prevalence of subclinical infections in cattle; 18% in New England and 11% in Wisconsin.

2. It is a chronic granulomatous disease of the digestive tract. Because it is so slow to develop, Johne's infection is usually widespread in a herd by the time the first clinical case is diagnosed.

3. It is characterized in cattle by the development of chronic, persistent, watery diarrhea and emaciation. Sheep and goats become emaciated, but do not show diarrhea. Recent evidence suggests that subclinical infections may be more costly than clinical disease with serious reductions in milk production and reproductive performance.

B. ETIOLOGY

1. Mycobacterium paratuberculosis (M. johnei)

2. It is antigenically related to M. bovis and M. avium

3. Strongly acid-fast with Zeihl-Neilsen stain

4. It is a fairly resistant organism, and it will survive on pasture for up to one year, and in slurry pits up to 2 years. This makes clean up difficult on infected premises.

5. It is difficult to grow in culture. It requires specialized media, mycobactin growth factor, and cultures require 8-12 weeks of incubation routinely. In Minnesota cultures cost $5.00 per sample when sampling a whole herd.

C. EPIDEMIOLOGY

1. It can affect cattle, sheep, goats, and wild ruminants. Antelope and deer may serve as carriers to domestic cattle on common pastures. Cattle, sheep and goats may cross transmit the Johne's organism when maintained together.

2. Horses and pigs have been infected experimentally but they are not thought to be natural carriers of infection.

3. Transmission

   a) The primary route is fecal - oral through contamination of feed and water supplies with feces.
   b) Calves usually pick up the infection at the time of birth from an infected dam or from a contaminated environment, particularly when calving takes place in the cow barn.
   c) Johne's can be transmitted <u>in utero</u> but this is rare.
   d) The organism has been isolated from semen and milk, however venereal transmission is unlikely.

4. Cattle are infected early in life, being the most susceptible up to 6 months of age. As they get older, they develop a strong, natural resistance to this infection. It is very difficult to produce an experimental infection in adult cattle. The rate of infection in calves depends on pathogen load and management factors.

5. All breeds of cattle appear to be equally susceptible.

6. Clinical disease is not usually seen until at least 2 years of age, and is most likely at 2-6 years of age, despite the fact that infections begin at calfhood.

7. In infected herds, usually 1-5% of infected animals will show clinical signs. You can assume that for every one animal showing clinical signs, 2-5 additional animals are nonclinically infected.

8. Cattle fall into four categories in an infected herd:
   a) Clinically affected cattle
   b) Asymptomatic shedders that are passing organisms in their feces.
   c) Infected non-shedders
   d) Non-infected, resistant animals

   Stress causes animals to go from one category up to the next toward being a clinical case. Parturition or shipping are common stressors which may trigger the onset of clinical disease.

D. PATHOGENESIS

1. Calves ingest the organism early in life, go through a nonclinical septicemic phase, and then the organism localizes in the G.I. tract and adjacent lymph nodes.

2. Once localization occurs, the animal may remain asymptomatic for several years before showing clinical signs or it may never show clinical signs at all.

3. A few animals eliminate the organism during the septicemic phase and become resistant.

E. CLINICAL SIGNS

1. Clinical cases often have a history of some type of stress. Many break with diarrhea shortly after calving.

2. Animals develop a profuse diarrhea characterized by homogenous consistency with no offending odor, no necrotic tissue, and no blood. The diarrhea has a characteristic watery pea soup appearance.

3. The diarrhea may be preceded by weight loss but this may go unobserved by the farmer. Diarrhea can be either intermittent, with temporary recoveries, or persistent.

4. Animals often maintain good appetite despite progressive emaciation and increasing diarrhea.

5. Anemia and hypoproteinemia are common.

6. Eventually animals become so thin and debilitated they are shipped by owner or collapse and die.

7. Sheep and goats commonly show progressive emaciation <u>without</u> diarrhea. For this reason, Johne's disease is often overlooked in these species.

F. DIAGNOSIS OF CLINICAL CASES

1. Agar Gel Immunodiffusion Test
   a) Extremely accurate for confirmation of clinical cases.
   b) Less reliable for identification of subclinical cases.
   c) Results available in 48 hours

2. I.V. johnin test
   a) Good accuracy in clinical cases
   b) Requires 6-8 hours of temperature taking after injection of johnin.
   c) Formerly available only by regulatory veterinarian. Now the practitioner can do it.

3. Intradermal skin test
   a) Similar to TB test
   b) Requires follow-up visit to read reaction
   c) Advanced clinical cases may show false negative results due to anergy.

4. Rectal scraping
   a) Positive results are accurate
   b) Negative results misleading
   c) Lesions may be present but not as far caudally as the rectum

5. Fecal smears - acid fast stained
   a) Rapid results
   b) Good reliability if performed accurately
   c) Must examine methodically, at least several minutes.
   d) Must differentiate larger saprophytic hay bacillus M. phlei from M. paratuberculosis

6. Fecal culture
   a) Recommended as test of choice
   b) Accuracy very good
   c) Impractical for confirmation of clinical cases. Cow dead or culled before results come back.

7. Postmortem
   a) Gross lesions
      1. Thickening of wall of ileum, cecum, colon
      2. Enlargement of ileocecal, mesenteric lymph nodes
      3. Variable extent of lesions
      4. Occasional calcification of aorta
      5. Sheep and goats may have minimal or absent gross lesions.

   b) Histopathology
      1. Granulomatous infiltration of lamina propria of intestine.
      2. Acid fast organisms in macrophages

   c) Culture ileocecal valve and lymph node at post mortem for definitive diagnosis.

G. CONTROL

1. Unless the clinically confirmed case was recently purchased, it can be assumed that additional subclinical cases are present in the herd.

2. Implications of a Johne's positive herd vary depending on state regulations (quarantine vs. no quarantine) and the objectives of the farmer (milk sales vs. sale of breeding stock).

3. Control programs can be frustrating, prolonged and expensive. Minimum time reported from onset to Johne's free status approximately 4 years. Some herds reported positive after 10 years of control efforts. De-population or living with the problem are alternatives to consider in discussions with the herd owner.

4. Three major components to control programs: management changes, identification and culling of subclinically infected cattle, and vaccination.

   a) Management changes
      1. Aimed at breaking cycle of new infections
      2. Clean maternity stalls or pasture calving
      3. Calves removed immediately at birth
      4. Calves fed colostrum by bottle from hand washed udder
      5. Calves raised in isolated completely away from cow herd. Calf hutches good for this.
      6. Maintain young stock separate from cow herd at least until breeding age.
      7. Improve sanitation and manure handling.
      8. Clean and disinfect premises with orthophenylphenate (Environ)
      9. Do not pasture cattle where manure has been spread for at least one year.
      10. Do not use ponds or stagnant water for water supply. Pipe in water to troughs or cups.
      11. Remove all thin or diarrheic animals immediately.

   b) Identification and culling
      1. Aimed at reducing the environmental load of <u>M. paratuberculosis</u> by removing the subclinical shedders.
      2. Herd-wide fecal culture currently the only reliable test for identification.
      3. Test results require an average of 8-12 weeks.
      4. False negative fecal cultures can occur.
      5. Twice a year sampling recommended.
      6. It is strongly recommended that all offspring of fecal culture positive cows be culled from the herd.

c) Vaccination
   1. Aimed at eliminating the development of future clinical cases.
   2. A killed bacterin produced by Fromm Laboratories recently licensed for restricted use in the United States.
   3. Given to calves only up to 35 days of age.
   4. Obtainable only by permit from state veterinarian for use in fecal culture confirmed positive herds which have tested negative for tuberculosis.
   5. Drawbacks
      a) Danger to vaccinator - nasty granuloma formation if accidently injected into hand
      b) Produces fibrocaseous nodule at injection site in calf.
      c) Interferes with intradermal skin testing for tuberculosis
      d) May not eliminate infections. Although clinical disease will not develop, some percentage of calfhood vaccinates do become subclinical shedders.

H. REGULATORY CONSIDERATIONS

1. No comprehensive national standard exists.

2. State regulations vary
   a) Johne's is a reportable disease subject to quarantine in Minnesota.
   b) In Wisconsin, no quarantine exists. Veterinarians must state in writing that the herd of origin is presumed free of Johne's disease when signing health papers for entry into Wisconsin.
   c) Great variation in state rules. Check with state veterinarian's office where you practice.

3. Rigorous export controls
   a) Bull studs must be Johne's free for semen export to many countries.
   b) Breeding stock often required to have negative Johne's complement fixation test for export.

Dr. Fred J. DeGraves

Residue avoidance in meat and milk products is becoming an increasing concern in food animal practice as surveillance procedures improve and consumer pressures force the issue. Ideally, simple and economical on farm tests for milk and meat residue would reduce the possibility of drug residues entering the human food chain. The Live Animal Swab Test (LAST) is such a test recently developed by USDA's Food Safety and Inspection Service.

The LAST test is an easily performed method of detecting antibiotic residue in a treated animal prior to listing for slaughter. The producer or the veterinary practitioner can readily tool up to run the test using inexpensive materials and very little time.

The LAST test is composed of a vial of Bacillus subtilis spores suspended in alcohol, a petri dish of antibiotic no. 5 media, a 5 mcg neomycin control disk, tweezers, a millimeter rule, and an incubator.

An inexpensive incubator can be constructed from a styrofoam ice cooler, an automatic aquarium heater (75 watt units are adequate), a jar and a thermometer. To construct the incubator, we simply place the aquarium heater in the jar which is filled with water (to provide humidity) which is then placed in the cooler, plugged in and calibrated for 84 F using the thermometer which is inserted through the side of the cooler at the level of the petri dish. I would discourage using the aquarium heater out of water as most of these heaters are not designed to be used in this manner.

To perform the test a cotton swab is used to spread the B. subtilis spore suspension over the media in the petri dish evenly. Tweezers are then used to place an N5 neomycin disk on the petri dish. Tweezers are used to avoid contaminating fingers with neomycin, which then might contaminate the sample being tested. Another swab is then dipped in urine, serum, milk, or saliva and excess fluid is shaken off. The shaft of the swab is broken off without touching the cotton with fingers and is then pressed onto the petri

dish without breaking the surface. The petri dish is covered and placed into the incubator which is set at 84 F. The test is read in 18-24 hours. There should be a clear zone of 16-24 mm around the neomycin control disk for the test to be valid. If the bacteria grow right up to the swab, the animal is antibiotic residue negative and is ready to market. Any clear zone around the swab indicates the animal has antibiotic residue and should be retested in 2-3 days. If the clear zone around the control disk is less then 16 mm the test should be repeated, if residue negative. If there is a clear zone around the swab the test is residue positive, regardless of the control disk.

It is recommended that the test not be used before established withdrawal times have been observed. However, with the myriad of physiological processes occurring in the convalescent bovine, I believe the LAST test will prove quite valuable in reducing withdrawal times to the economic benefit of the producer. The test will also be quite valuable when veterinarians are forced to use drugs off label.

The LAST test has been designed to be used with several body fluids, such as urine, milk, serum and saliva. At the present time urine is the fluid of choice as it has the highest correlation with antibiotic levels in the tissues. The correlation between antibiotic levels in the serum and tissues are currently being worked out and may be more useful in the future. Presently, it is recommended that samples be refrigerated immediately after collected and the test be run within 4 hours. Further work may be able to increase this time considerably, studies are being conducted on the feasibility of using air dried samples on disks of analytical paper.

A similar swab test, the Swab Test on Premises (STOP), has been used by the USDA since 1979 on carcasses at the slaughterhouse to reduce antibiotic residues from cull dairy cows. The LAST test is a significant improvement in that it can be performed on live cows while still on the farm. The LAST

test has been an economical, easy to perform test in our practice with many useful applications. The test has been a welcome addition to the services that we supply our clients and I believe that its widespread use will markedly reduce the problem of antibiotic residues entering the human food chain.

Genocol-99

Charles R. Muscoplat, D.V.M.
Molecular Genetics
Minnetonka, MN

GENECOL™ 99

Scours is the number one killer of baby calves in the United States. More than one million calves are lost each year to scours-associated deaths according to USDA statistics. It is estimated 50 percent of the herds across the country experience a chronic scours problem. Even with conservative numbers being applied, this means dairy and beef producers are absorbing a $100 million loss every year.

Calf scours is not a single disease. Rather, it is a clinical sign associated with several diseases characterized by diarrhea. Regardless of the cause, diarrhea prevents absorption of fluids from the intestines while essential body fluids pass from the scouring calf's body into the gut. Death from scours usually results from dehydration, acid buildup and loss of electrolytes (body chemicals).

CAUSES OF CALF SCOURS

Overfeeding is the primary factor in noninfections or common scours. Others include inadequate nutrition of the pregnant dam, particularly during the last third of gestation, which affects quality and quantity of colostrum; unsanitary environment, which includes crowded, damp housing or pasture; and insufficient attention to the newborn.

There are several infectious cases of calf scours with the major cause being bacteria, particularly Escherichia coli.

Research shows E. coli strains that cause diarrhea adhere to the calf's gut with the help of pili-fine, fuzz-like protrusions. There are various types of pili, but the ones that make E. coli dangerous to baby calves are K-99 pili, which act like glue and adhere the E. coli cells of the intestinal wall. The bacteria then multiply and cause cells to secrete excess body fluids, called hypersecretion. This results in loose stools. Substantial fluid loss from the animal's blood and tissues causes dehydration.

Symptoms of an affected calf are loss of appetite, weakness and depression. Within 10 to 12 hours, the animal may not be able to stand. If dehydration continues another 6 to 12 hours, the calf becomes comatose and death may occur in 24 hours. (Remember, a calf is 70 percent water at birth!)

MANAGEMENT PRACTICES TO CONTROL E. COLI SCOURS

Particular attention should be paid to nutrition, environment, sanitation and care of the young calf in order to control scours. However, the most important requirement for the newborn which lacks scours-fighting antibodies to nurse colostrum early in life. Any effort to prevent scours by vaccinating a cow is wasted unless the calf receives colostrum soon after birth as the calf loses its ability to absorb colostrum antibodies by the hour. A calf should receive at least two quarts of its mother's first milk after birth.

Prevention of the disease is preferable to any treatment. Injecting pregnant dams with scour vaccines prior to calving is one alternative. The cow then develops antibodies which are transferred to the calf when it nurses. However, timing of

vaccination is critical, and it may be difficult to pinpoint expected calving dates. Plus, handling the cow in the last weeks of gestation can be stressful. If you haven't had scour problems in the past, it's easy to skip this prevention practice.

The ideal solution is to use a calf-directed product which is easy to administer and complements good management practices. Genecol™ 99, which is given to the calf within 12 hours after birth, is an effective prevention for fatal E. coli scours. The K-99 antibody of Genecol™ 99 ties up pili on the E. coli bacteria cells and prevents them from adhering to the intestinal wall.

A SPECIFIC ANTIBODY

Genecol™ 99 is the first monoclonal antibody to be introduced in the U.S. for animal disease prevention. A monoclonal antibody is an antibody derived from a single source or clone of cells which recognizes only one kind of antigen.

The first step in the relatively new process of producing monoclonal antibodies is to inject a specific disease-causing agent, like bacterium, into a mouse. The mouse, in response, begins to develop an immunity to the disease. An antibody-producing cell is then removed from the mouse's spleen and fused with mouse cancer cells, called myelomas, which enable the hybrid cells, called hybridomas, to survive under laboratory conditions. The hybridomas are then cloned to create more identical hybrid cells. These cells mass-produce specific antibodies almost indefinitely either in a mouse into which they have been injected, or in laboratory culture. The antibodies are called "monoclonal"

because the hybrid cells producing them trace back to a single cell. Molecular Genetics, Inc. of Minnetonka, Minnesota, manufacturer of Genecol™ 99, has evaluated various ways of producing the antibody and found using mice to be most economical.

Monoclonal antibodies, like Genecol™ 99, are pure and safe, containing no live organisms that can endanger the animal or herdmates. The most important advantage to Genecol™ 99, however, is its effectiveness.

UNIVERSITY AND FIELD TRIALS

Clinical tests in Canada and the U.S. prove that Genecol™ 99 significantly reduces mortality from E. coli scours. In combined laboratory trial results from the Veterinary Infectious Disease Organization in Canada and the University of Minnesota, Genecol™ 99 had a 71 percent survival rate among calves exposed to E. coli bacteria. Only 18 percent of the unprotected calves lived.

On-farm performance has been excellent. Calves on dairy farms in Florida and Wisconsin were fed colostrum and then exposed to naturally-occuring levels of E. coli bacteria. All calves who contracted scours were treated with electrolytes or antibiotics. In spite of these efforts, 15 percent of the unprotected calves in the on-farm trials died. There was no death loss among those protected on the farms with Genecol™ 99.

WHEN TO USE GENECOL™ 99

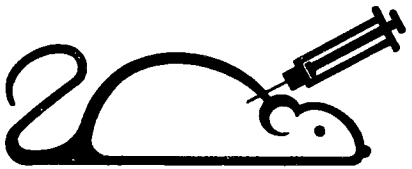
This revolutionary new product can make a difference in controlling calf scours, but it must be used correctly if its life-saving benefits are to be obtained. This means it must be

given to the calf as early after birth as possible. The first 12 hours post partum are the most critical in preventing E. coli bacteria with the K-99 antigen from adhering to the intestinal wall. Remember, too, Genecol™ 99 is not a treatment. It is designed to be given to healthy calves to keep them out of trouble. As soon as you diagnose a calf with E. coli scours, give Genecol™ 99 to future calves. Or, use it routinely on valuable purebred animals. Innoculating calves against scours may be incorporated with other post partum management practices such as navel dipping and ear tagging.

Genecol™ 99 is a liquid that comes in a 10 ml syringe and is given orally. It is sold only to licensed veterinarians and is available from them throughout the U.S. and Canada.

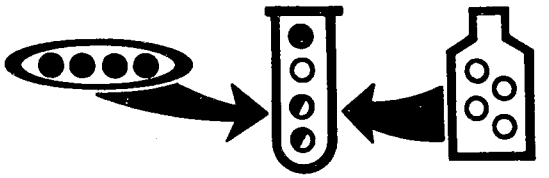
Genecol™ is a trademark of Molecular Genetics, Inc.

Monoclonal Antil dy Production

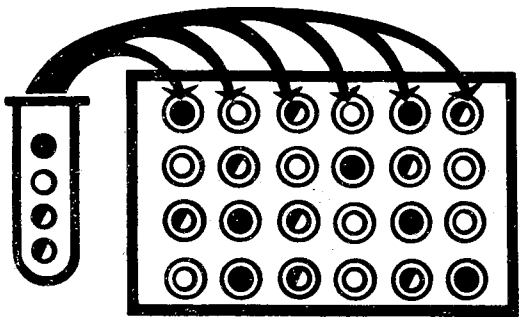


STEP 1. Hyperimmunization. Mice are immunized repeatedly with antigen of choice.

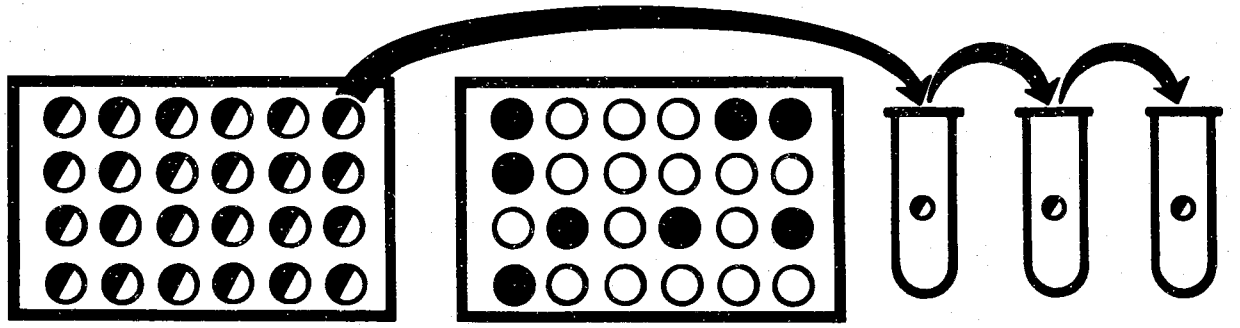
STEP 2. Collection of anitbody-producing lymphocytes. The immunized mouse is euthanized, its spleen removed and disrupted. Individual antibody-producing lymphocytes (B cells) are collected.



STEP 3. Cell fusion process. B-lymphocytes from Step 2 are incubated with mouse myeloma cells in media containing polyethylene glycol, which promotes cell fusion and hybridoma formation.

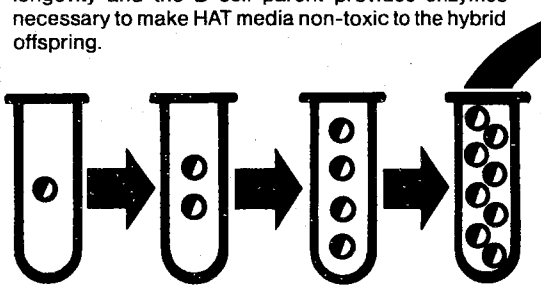


STEP 4. Selection of successful hybrid cells. The cells are transferred from the fusion media to a new specialized media known as HAT (hypoxanthine, aminopterin, thymidine). Because of enzyme deficiencies in laboratory-maintained myeloma cell strains, these media constitutents are toxic to unfused myeloma cells and myeloma-myeloma fusions. These cells die in HAT media. Unfused B-lymphocyte cells (antibody-producing cells) or B cell-B cell fusions die naturally after an extended incubation in HAT media. Hybridoma cells (B cell-myeloma fusions) are the only survivors, because the myeloma parent imparts longevity and the B cell parent provides enzymes necessary to make HAT media non-toxic to the hybrid offspring.

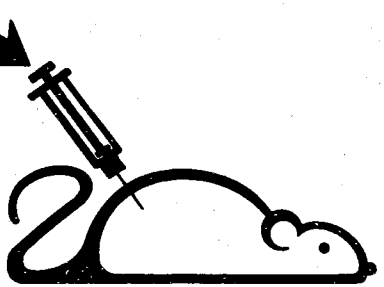


STEP 5. Identification of desired antibody-producing hybrids. Surviving hybrids are suspended in fresh media and distributed in microtiter plate wells. After incubation, supernatant fluid from the wells is screened for the presence of the desired antibody, using one of several immunologic techniques. Only a small number of B cells taking part in successful hybrid fusions are producing the antibody for which the mouse was hyperimmunized. So, screening is necessary to locate hybridomas producing the desired antibody.

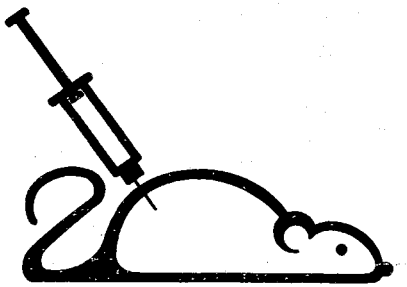
STEP 6. Isolating individual hybridoma cells. The cells in wells identified as producing the correct antibody are subjected to a repeated serial dilution process with distribution of dilute aliquots of cells to new microtiter plate wells. Wells are rescreened repeatedly for the desired antibody. This limiting dilution process is tedious but necessary to insure that a single parent hybridoma cell is isolated so that subsequent clones produce a highly pure, specific, homogenous antibody.



STEP 7. Clonal expansion. The isolated hybridoma cells are placed in fresh tissue culture media for multiplication (cloning). Clones or identical antibody-producing offspring can be frozen and stored for later use, or used immediately for large volume antibody production.



STEP 8. Antibody production. Two major options are available for antibody production. Hybridomas can be maintained in tissue culture with the antibody being harvested from the tissue culture fluid. Or, hybridoma cells may be injected intraperitoneally into mice or rats. These cells will adhere to serosal surfaces, actively reproduce, and secrete antibody into the ascities fluid of the host animal. Currently, the latter technique produces higher volumes for the desired antibody.



STEP 9. Antibody collection. To collect the antibody, the animal is euthanized, and the ascites fluid aspirated. Depending on the desired use of the harvested antibody, purification may be as simple as centrifugation of the ascites fluid, or involve more complicated immunological purification techniques.

"Crystal-balling" the Future of a Minnesota Dairy Practice

Henry Banal, D.V.M.
Sauk Centre, MN

In over 30 years of practice I am now experiencing, for the first time, a period of extensive economic change that is not in a positive direction.

For years the dairy practitioner has lived under an umbrella of protectionism-programs for the dairy farmer that has spelled out economic well being. We now see an umbrella with a few rents that no longer gives the dairy farmer, and indirectly, the dairy practitioner, some of the security they once enjoyed. This might be for the better, as adversity often has a way of bringing out the best and something better seems to result. However, this is an area that has to be addressed by all affected parties. It is an area of practice that has to be looked at in a more complete or in-depth fashion than it has been in the past. We have to address and consider recent changes that have occurred and possibly will occur in practice and face these changes in such a way that will continue to keep the dairy practitioner a viable and integral part of the dairy industry of the future.

I would like to bring out some changes or trends that have occurred in our practice through the years, some thoughts for the future, and the role of the dairy practitioner in a future perspective.

Perhaps a few words to describe our practice. We have a 5 person group practice with clinic facilities for both large and small animals. We are in a dairy area with less than a dozen beef type operations. Most of the dairy operations are of the 40 to 60 cow family size farm. Approximately 87% of our income is derived from the bovine, either in direct on-the-farm calls or in dispensing bovine related products.

It is interesting to see the impact or relationship of the herd management part of our practice to the total picture. In looking at the six year period of 1978 thru 1983 we find the following trends occurring in regard to gross business derived from:

1. Total bovine calls
2. Routine bovine calls
3. Herd Health calls

Percent of Gross Business - ALL BOVINE CALLS

1978	65%
1979	67%
1980	66%
1981	64%
1982	66%
1983	62%

Percent of Gross Business - ROUTINE BOVINE CALLS

1978	56%
1979	59%
1980	56%
1981	54%
1982	54%
1983	48%

Percent of Gross Business - HERD HEALTH CALLS

1978	9%
1979	8%
1980	10%
1981	10%
1982	12%
1983	14%

In summary, there is a gradual decrease in routine bovine call percent of income from 56% to 48% in six years, with a sharp drop in 1983 primarily occurring in the last half of 1983. A steady increase in percent of income took place in herd health from 9% to 14%. There is a possibility of some shifting of income generated on routine calls to herd health calls, but in analyzing some of the specifics I feel this to be of a very small percentage.

Comparing the six month period of October thru March of 1982 -'83 with 1983 -'84 we find some significant changes

taking place. We have to keep in mind this period of time in 1983 -'84 represents a reduction of about $2.00 per hundredweight in the price of milk and also the start of the new federal milk program.

Income	% of Total Gross Oct.'82 - Mar.'83	% of Total Gross Oct.'83 - Mar.'84
All Bovine Calls	66%	62%
Routine Bovine Calls	53%	47%
Herd Health Calls	13%	15%

The percentage change in the following areas of income occurring during these two periods is also worth looking at:

	% Change
Total Gross Income	- 4%
Total Bovine Income	-11%
Routine Bovine Income	-16%
Herd Health Income	+11%

Another source of income that is important in our practice is dispensing. Of the total dispensing dollars about 5% to 6% is dispensed directly in the country and the balance from the Clinic. In the past six years there has been a change

in dispensing income from 26% to 31% of our gross income for an increase of 5%. Interestingly enough, comparing the October thru March period of the last two years, we find an 8% decrease in total dispensing in the 1983 -'84 six month period.

The balance of our income, about 11% - 12% is derived from companion animals, swine, laboratory, and miscellaneous.

Nutrition income had been somewhat stable until the last two or three years when a concerted effort on the part of one of the partners resulted in these changes:

	Sales in Feed Additives
1978	$ 2,819
1979	2,624
1980	2,159
1981	4,530
1982	7,743
1983	19,854

This is an area that takes much dedication and effort on the part of the individual and can easily be lost in disappointment, failures, and pressures from outside feed sources. In addition to the supplement sales, about $1,200 additional income has been generated this past year in nutrition consultation.

Looking at some trends in the type and number of calls over the last five years reemphasizes what has been brought out previously in terms of income.

	Type of Calls				
Year	Program	Basic	Repeat	Milk Fever	Total
1979	885	6843	2015	575	10,318
1980	939	6828	1605	586	9,958
1981	836	6478	1403	549	9,266
1982	1064	6023	1143	699	8,929
1983	1325	5583	1303	599	8,810
5 Year Percent Change	+49%	-18%	-35%	+4%	-15%

In evaluating herd health income on a per call basis we found the following situation taking place:

Year	Herd Health Per Call Income	Routine Per Call Income
1979	$39.00	$27.00
1980	51.00	31.00
1981	61.00	35.00
1982	68.00	40.00
1983	68.00	40.00

If an average of 11 herd health calls were made per farmer per year in 1982 & 1983 it cost the dairyman (not

including nutrition) $748.00 per year for a herd health program. For the amount of increased profitability that we are providing the dairyman it seems to me that we should be looking at a different level of fees.

In view of the changes that have and are occurring in our practice how should we be looking at the dairy practitioner in a future perspective?

First of all we have to accept the fact that the dairy industry as we have known it for the last 15 to 20 years will never again exist. Irregardless of what government programs might be developed they will never again reach the degree of subsidy that we have had and as a result the dairy farmer will have to stand more on his own two feet. We are now seeing some of the very marginal operators selling out and in the next one to two years I think we can look for an increased number of them to fall by the wayside.

There is an increased amount of pressure by the financial community on the questionable operators and their list of farmers to "watch" so to speak, is getting longer.

This brings us back to the point where there will be greater competition for the farm dollar that will be available.

We have seen in the practice income figures for the last six months how drastic a reduction there has been in money spent for routine services and medication. Whether the livestock health problems are taken care of or not,

we have to realize that there is now and will be less money spent and available to spend in these areas, whatever the consequences.

We can see money being spent and the willingness to spend it in the area of herd management. This specific field, if any, is where the dairy practitioner has to grow faster than ever before. Increased competition from other people involved with the dairy farmer, offering various programs, help, and expertise will force the dairy practitioner to become more adept in herd management.

We have to make ourselves acquainted with bankers, farm financial advisors, and the dairy industry people on the local level and show them that we are and can be a part of this team in the management of the dairy herd. Visits with the bankers and other members of the financial community in the area are of utmost importance and I have always found the welcome mat out and these individuals more than willing to discuss specific or general problems. Both the financial advisors and veterinarian need an open and trusting relationship in helping the dairy farmer. <u>It can be done</u>.

In discussions I have had with recent graduates I am appalled and shocked at the lack of instruction given the student in the basic economics of herd management for the dairy farmer. We should be emphasizing this area so the graduate will at least have better guidelines to follow, rather than having to spend valuable time in practice

trying to find a way and a direction. We are lacking in preparation in this area and a specialty in consultive herd management has to be forthcoming in education, especially if we are to absorb all the graduates that we will be looking at in the near future.

It is gratifying to see the instructor-practitioner exchange program that is planned in Minnesota. There is much that I think can be accomplished by both parties in an exchange of this type. This is a very positive step forward.

This is the age of microcomputers and the veterinarian is no exception to their utilization. We need a program that will integrate the present DHIA information and individual herd health information as well as the business aspects of the practice. At the present time I cannot feel that there is anything available in Minnesota that to me would seem suitable in this regard. We have to be able to quickly and effectively evaluate where the dairyman is, and where he is going. An effective microcomputer program could do just that. At the present time I feel there are too many efforts that are not being coordinated for the good of all. Perhaps there should be more effort on the part of the Extension people and University personnel to coordinate efforts in this endeavor.

I think we have to show enthusiasm in projecting the necessary programs that we want to put forth. We also have to show and more important <u>feel</u> a sincere and meaningful

desire to help and be part of this management process. If we can portray these basic attitudes and feelings to the dairyman and all parties involved, the veterinarian will eventually be more a part of the dairy industry of the future than he ever was of the past.

Ivermectin in a Dairy Herd Health Practice

Andrew P. Johnson, DVM
Valley Veterinary Clinic, S.C.
Seymour, WI 54165

As we all know, cattle are affected by both internal and external parasites. Internal, or endoparasites, live within the host and must obtain their nutrients from that animal. The host's ability to thrive, or even to survive, is often decreased as a result of the presence of parasites. External, or ectoparasite infestations affect the health of the host animal in several ways. Cattle are so preoccupied with the itching and irritation that feeding is irregular and consequently, the animal fails to gain weight. This is called "parasite worry". They can also lead to stress and the animals are more susceptible to bacterial and viral infections.

We can break the internal or endoparasites into three categories: nematodes (worms) cestodes (tapeworms), and trematodes (flukes). Nematodes are the major cause of economic parasitism of cattle in the U.S.

As practitioners we must design parasite control programs as part of the total herd health program. We want the programs to be complete, effective and easy to use. Up to now if we asked a dozen veterinarians for the best parasite control program we could get a dozen different answers.

When dealing with parasite control we must consider the young stock and the adult cows. Most practitioners will readily agree that the young stock definitely benefits from parasite

control. The real controversy is whether the adult cows benefit from parasite control or not. Every time we pick up a magazine we get a different opinion on the pros and cons of adult parasite control. One article clearly shows success from parasite control and the next article clearly shows no difference. No wonder we have so many ideas on the perfect control program. Our practice definitely feels a parasite control program is beneficial to the adult cows. We see responses such as increased milk production, increased butterfat production, and healthier cows. It eliminates one extra unnecessary stress.

Let's concentrate on the young stock since we all agree that there is definite benefit in parasite control programs. For years we all have looked for the complete program that gets both internal and external parasites yet is simple to use. Again, we get dozens of "ideas" and effective programs. A new drug called Ivermectin or Ivomec has been introduced into our practices that is a major breakthrough we have been looking for. It is effective and easy to use. The major advantage of this product is that it gets both internal and external parasites in one simple injection.

What is Ivomec? It is an ivermectin which is a derivation of avermectins. These are natural products fermented from soil organisms, first isolated in Japan, known as Streptomyces avermitilis.

Avermectins are made up of four major components. Of these four, Avermectin B_1 was chosen my Merck because of its extraordinary anti-parasite properties. Its mode of action is

unique, not shared by other anti-parasitic agents. This chemical is a neurotransmitter called GABA (gamma aminobutync acid). In nematodes the GABA binds and blocks nerve impulses thus killing the parasites. In arthopods the GABA blocks nerve to muscle impulses thus killing parasites. Ivermectins have no effect against flukes and tapeworms presumably because they do not have GABA as nerve impulse transmitters.

Ivermectin, the active ingredient of Ivomec, is structurally related to any presently available parasiticides. There has been no cross resistence. Ivomec has been evaluated in 56 controlled efficacy trials and 181 field trials. At 200 mcg/kg or the optimal dose for Ivomec, they have found a 95% or better efficacy against nematodes. The drug was also very effective against louse and mite infestations as well as rubs. Ivomec was effective against all stages of cattle grubs. For the most effective results cattle should be treated shortly after the fly season.

One of the most significant clinical responses we have seen is on its efficacy toward Type II Ostertagiasis. Many times we have treated a group of calves with a parasiticide and we actually see a negative response such as diarrhea and rough looking calves. This is due to the old drugs inability to get the Type II Ostertagia larvae. Ivomec gets these and we usually see a marked clinical response with positive results such as brighter and slicker type calves. We feel this is a very important step and makes Ivomec superior to others.

Ivomec is safe and has low toxicity. There were no clinical signs of toxicosis at 6000 mcg/kg or 30 times the regular level.

Ivermectins were tested against teratology at double the dose. No abnormalities were detected it was concluded that the use of ivermectin at recommended levels in pregnant cows does not induce teratological changes.

The Ivermectins were tested against pregnancy as well. Cattle in their second and third trimester of pregnancy were injected with twice the regular dose.. No adverse effects were noticed indicating that there is no drug-related risk in dosing pregnant cows. There is no effect on the breeding soundness of bulls when injected with ivermectins. It was concluded that dosing cattle with ivermectin at twice use level did not result in an increased incidence of health problems compared to similar animals given a control.

Ivermectins are excreted in the feces in very low levels. They bind strongly to soil and will not enter the underground or surface water in hazardous amounts. It breaks down and does no accumulate in the soil.

Current dosage levels are 1 ml/110 lbs. in one injection site, under the skin. At this time the drug as a 35 day slaughter withhold. At this time there is no milk withdrawal time established, therefore it cannot be used in milking cows.

We feel Ivomec is safe for cattle when used under practical husbandry conditions. The unique activity of this product also permits control of external parasites of significance at times of the year when currently available products such as dips, pour-ons, and sprays cannot be used. Clearly beneficial effects with economic value will result from its use, such as decreased

morbidity from parasitism and resultant increases in growth and feed efficiency.

Ivomec -- "The Parasite Solution" -- is a milestone in the battle of parasites in cattle. One injection of Ivomec can eliminate the confusion, the time, the labor, and the expense that all of these methods represent. Ivomec is a significant leap forward from the cumbersome, often expensive, sometimes dangerous and less than effective techniques that have been used to battle parasites of cattle.

USING THE DHIA REPRODUCTION REPORT AS A FERTILITY PROGRAM RECORD SYSTEM

Wayne Ode, D.V.M.
Melrose, Minnesota

I first want to say that I have been working with the Minnesota Dairy Champ Computerized Record System for about two and a half years now. I feel that this experience has enabled me to develop this record system.

The ten years I have been in practice I have tried to operate fertility programs under a variety of conditions, and with different degrees of record keeping. I feel that poor record keeping will either cause a fertility program to fail, or else the farmer will not receive the full benefit of his fertility program. I have, also, noticed that some good farmers are able to compensate for this deficiency because they have a good memory, and/or they are using records that are not compatable with a fertility program.

This record system can fill this gap. This record system is not as good as the flip charts that most of us use. It is not intended to replace the flip charts. It should only be used on farms where the farmer is unwilling or unable to keep up the flip charts adequately. I. also. feel that other computerized record systems would fail in this situation. Computerized systems have their advantages that we should all look at. However, they don't allow for a DHIA supervisor to collect all the pertinent information and feed it into a computer.

In this report I will:

1. Tell how I have constructed this record holder
2. Explain how I use it

3. Review the advantages and disadvantages of this system

Construction of the Record Holder

The materials needed are:	Approximate Cost
1 16 1/2" x 13" x 1/8" piece of masonite	$0.50
1 16 1/2" x 18" piece of flexible clear plastic	0.50
1 roll of waterproof adhesive tape	4.00
12 14" x 11" pieces of tagboard	2.50
2 large spring steel letter clips	1.00
a small amount of glue	———
	$8.50

It will take about two hours to construct this record holder.

Directions

1. Cut a 1 inch strip from the bottom (the 14" side) of each piece of tagboard, taking care to leave a 1" x 1" tab on each piece. The tabs should be staggered so that when all twelve pieces are put together each tab is visible.

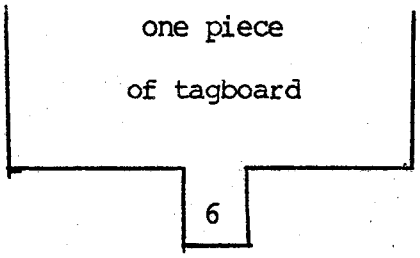


Figure 1

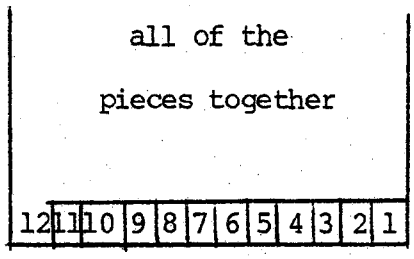


Figure 2

I usually cover each tab with a small piece of adhesive tape

for extra strength. Each tab should be numbered 1-12. For orientation please remember that this is the bottom (see figures 1 and 2).

2. Place a 2" strip of waterproof adhesive tape on the top of each piece of tagboard (see figure 3). 1" of the tape should extend beyond the edge of the tagboard.

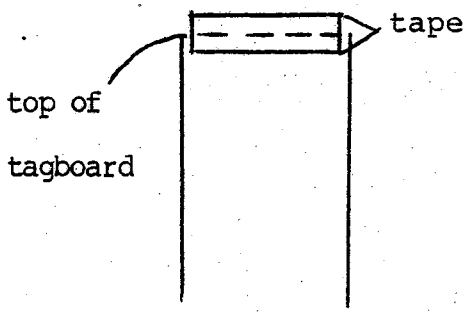


Figure 3

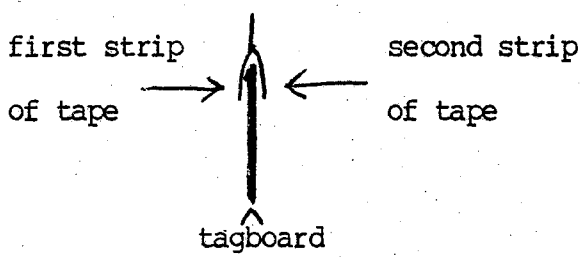


Figure 4

Next turn the tagboard with tape over, and apply another strip of tape so that it covers the first strip of tape and one inch of the tagboard (see figure 4).

3. Glue the piece of plastic to the back of the piece of masonite so that thirteen inches of plastic can lay over the front of the masonite (see figure 5).

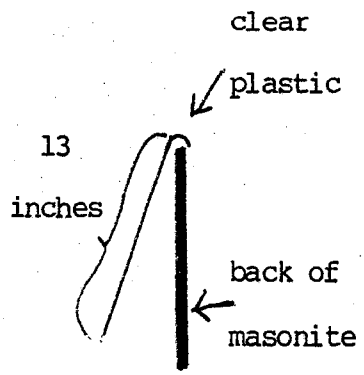


figure 5

4. The pieces of tagboard are placed on the front of the masonite with the tape edges just under the plastic. The free end of the plastic is placed over the tagboard. The letter clips are clamped over the plastic and the strips of tape to hold everything in place. (Please note that the tagboards are arranged in descending order so that, as in this example, the No. 12 tagboard has the No. 11 tagboard under it, and the No. 10 tagboard is under No. 11, etc. see figure 6.)

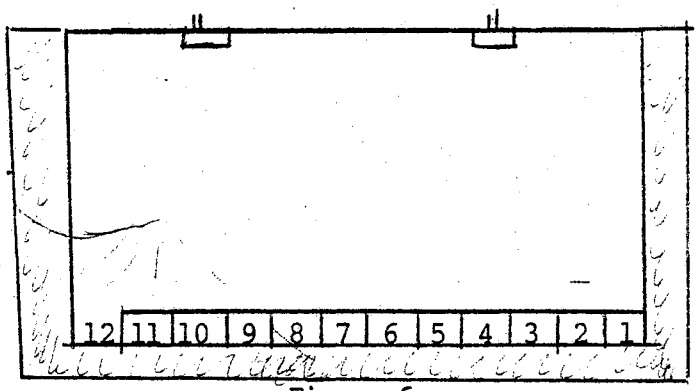


Figure 6

5. The DHIA reproduction reports are filed in this holder so that the most recent report is on top but under the plastic (for example, January). Under it is the tagboard with the number that corresponds with the previous months sample date (in this example No. 12). Under this tagboard is the previous months reproduction report (December). This allows one to file twelve months of reproduction reports, and allows for easy access to any months report. As will be explained later, this is necessary if the veterinarian wants to know the results of a previous rectal exam, and/or treatments done previously.

How I Use This Report

The minimum information I feel I need to operate a successful reproduction system is each cows last calving date, the last breeding date since calving, the number of times bred since calving, and the results of the last rectal exam. If you look at the attached example. you will find all of this information including the results of her last rectal exam, but only if she was found to be pregnant. (See last page of this report.) However, the results of all other rectal exams will not be included.

To get around this problem, I have the farmers record the date followed by the rectal results in the remarks section of the report. You can see this section is quite small, and I try to keep my entries short (Example: OK = uterus normal, edematous or tone, ovaries cycling; PG = pregnant; ME6 = metritis, infused with an antibiotic, etc.). There is a problem if more than one entry is to be made. So far, we have been able to use a neighboring cows slot and get by.

By having the eleven previous months reports filed and handy, it is usually no problem to look up the results of a previous rectal.

To this date, I have not used the management information available on the top of the report. Most of this information is not yet available, but I do plan on using it as part of my herd health programs in the future.

Advantages and Disadvantages

I started using this record system in September, 1983 on the following four farms.

No. 1 - 90 cow herd, 18.500 lbs. average, farming 10 years

No. 2 - 40 cow herd, 18,500 lbs. average, farming 15 years

No. 3 - 40 cow herd, 15,000 lbs. average, farming 35 years

No. 4 - 70 cow herd, 18,000 lbs. average, farming 20 years

Farmers number 1, 3 and 4 had used the flip charts before, and had stopped using them. Farmer No. 2 had not been on a reproductive program before this time. I visit farm No. 1 twice a months, and farms 2 and 3 once a month.

Farmer No 4 has quit using this record system. I feel that the reason this hasn't worked out is because this farmer is very aggressive in having his cows checked as soon as possible. I presently do mostly herd health work, and I am seldom the one going to his farm to treat sick animals. Consequently, whatever veterinarian goes to his farm will probably check 4-10 animals for him, and the results aren't recorded. When we call for a herd check, he often doesn't have enough cows to check to make the trip worthwhile. This problem wasn't apparent to me before because I was doing all of his veterinary work before then.

The other three farmers are presently using this system and like it very much. Time spent on record keeping is the same as before or reduced. They generally check this record about one to two times a week for their own information. They would recommend this system to other farmers.

The major disadvantages to this system are:

1. The recording or remark area is too small for more than one entry.
2. It is slightly inconvenient to look up a previous months information. There is also, a small time loss involved here but

at least the information is available.

3. The most current information is not recorded. If the farmer has a good memory this is not much of a problem. A quick look at the breeding chart is sometimes necessary.

4. Information from a previous year is not available.

The major advantages are:

1. DHIA collects the information and does the recording.

2. Except for the record holder, there is no extra cost if the farmer is already on DHIA.

3. Additional information is easily available. Example - milk production, days to first heat, days open, days in milk, etc.

4. Summary information will e available in the future.

5. It can be a first step in record keeping that could evolve into a more complete system.

6. It is an added encouragement for farmers to get on, and stay on DHIA.

JOHN DAIRYMAN
101 HAECKER HALL ST PAUL MN 55108

HERD CODE 41-00-0015 | MAIL DATE 7-07-83

MINNESOTA
DAIRY HERD IMPROVEMENT
DHI 230 11/82

REPRODUCTION

SAMPLE DATE 6-28-83 | PAGE 1

MONTHLY REPRODUCTIVE CYCLES

MONTHS	JUN	JUL	AUG	SEPT	OCT	NOV	DEC	JAN	FEB	MAR	APR	MAY	JUN
Est Num Heats	0	0	1	1	5	6	8	10	12	11	10	7	4
Reported Heats	0	0	0	0	1	6	4	4	7	9	6	5	4
Num Breedings	0	0	0	0	1	6	4	4	7	9	6	5	4
Num Conceived	0	0	0	0	1	2	2	3	4	3	5	1	1

MONTHLY CALVING PATTERN

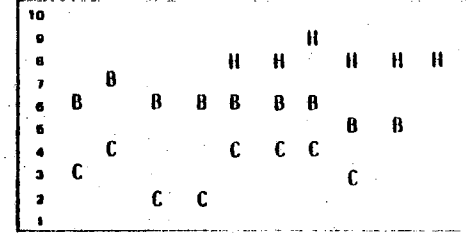
Cows Calved Last	1	0	3	1	2	1	6	2	0	0	0	0	2
Heifers Calved Last	0	0	0	1	1	2	1	0	1	3	0	0	0
Cows To Calve	0	0	2	3	2	3							
Heifers To Calve	0	0	0	1	1	2							

LIST OF PROBLEM COWS

BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN
MOLLY *195					
PLUTO *161					

REPRODUCTIVE ACTIVITY

March 15, 1982 →



COMPUTER NUMBER	COW'S SIRE ID	COW'S SIRE PD$	DATE CALVED	LACT NUM	DAYS TO 1ST HEAT	DAYS OPEN	NUM HEATS MISSED	LAST BREEDING OR HEAT TYPE	LAST BREEDING OR HEAT DATE	LAST BREEDING OR HEAT SERVICE SIRE ID	LAST BREEDING OR HEAT SERVICE SIRE PD$	BARN NAME	DATE TO DRY	DAYS BRED TO PG EXAM	DUE DATE	DAYS IN MILK	PROD INDEX	PEAK MILK	SAMPLE DAY MILK ACTUAL	SAMPLE DAY MILK EXPECT	REMARKS
0069	APACHE		9-04-82	1	68	107	0	3	12-20-82	7HI115	+173	APACHE	8-07-83		9-26-83	298	97	58	32		
0077	17H365	+180	3-06-83	1	93	93	2	1	6-07-83	21H380	+186	CB			POSS PG	115	83	58	46		
0049	29H2417	+161	1-16-83	3	52	72	0	2	3-29-83	10H6335		CRISCU	11-14-83	35	1-03-84	164	96	98	68		
0051	29H2510	+190	6-16-83	5		13						DANA				13		77	77		
0070	FORD		10-17-82	1	89	89	1	1	1-14-83	29H3820	+206	DEANNA	9-01-83		10-21-83	255	96	57	47		
0043			12-14-82	3		70		2	2-22-83	23H217	+178	EFFIE	10-10-83		11-29-83	197	99	97	64		
0072	29H2477		11-03-82	1								ELOISE	***** OU	NOT	BREED **	238	86	66	43		
0042			9-24-82	3	60	199	4	4	4-11-83	11H2238		ELSIE	11-27-83	64	1-16-84	278	100	91	34		
0071	40H2789		11-02-82	1	75	171	2	4	4-22-83	40H2763	+95	EMY	12-08-83	53	1-27-84	239	107	77	60		
0053	ART		8-25-82	2	66	66	0	1	10-30-82	11H2354		FAWN	DRY		8-06-83						
0033	29H1879		10-25-82	4	49	149	2	4	3-23-83	11H2238		GYPSY	11-08-83	41	12-28-83	247	101	91	54		
0061	KNIGHT		8-24-82	2	87	87	1	1	11-19-82	40H2763	+95	HULLY	7-07-83		8-26-83	309	99	75	29		
0062	ART		2-05-83	2	76	76	1	1	4-22-83	40H2455		IRIS	12-08-83	53	1-27-84	144	95	83	66		
0052	STARWAR		6-14-83	3		15						JANE				15		93	93		
0056	STARWAR		9-27-82	2	83	83	1	1	12-19-82	29H3820	+206	JILL	8-06-83		9-25-83	275	91	79	21		
0066	BILLY		10-30-82	2	40	80	1	2	1-18-83	40H2763	+95	JODY	9-05-83		10-25-83	242	109	90	55		
0060	KNIGHT		12-31-82	2	78	78	1	1	3-19-83	11H2238		KIM	11-04-83	45	12-24-83	180	127	113	71		
0078	17H248	+133	3-12-83	1	48	48	0	1	4-29-83	10H6335	+222	LORI			POSS PG	109	90	59	52		
0075	29H2389		2-04-83	1	70	92	1	2	5-07-83	29H4000	+31[illegible]	MAGDA	12-23-83	38	2-11-84	145	94	68	51		
0076	40H2652	+148	3-01-83	1		108	0	3	6-17-83	10H5801	+164	MAMIE			POSS PG	120	88	55	49		
0040	29H2434		12-13-82	3	50	127	3	2	4-19-83	29H3645	+146	MANDY	12-05-83	56	1-24-84	198	99	87	59		
0064	BOUQUET		12-10-82	2	53	195	2	6	6-23-83	11H2521		MOLLY			POSS PG	201	107	88	61		
0059	ERIC		11-24-82	2	59	80	0	2	2-12-83	17H387	+213	NICKY	9-30-83		11-19-83	217	119	105	65		
0068	ERIC TW		12-25-82	2	91	91	1	1	3-26-83	29H3303		OLIVE	11-11-83	38	12-31-83	186	98	88	65		
0065	KNIGHT		1-15-83	2		161	3	3	6-25-83	10H6466	+181	PLUTO			POSS PG	165	104	93	66		
0073	BUTERDA		12-27-82	1	87	106	1	2	4-12-83	10H6335	+222	RITA	11-28-83	53	1-17-84	184	94	61	49		
0058	GERRI		8-12-82	2	77	77	1	1	10-28-82	11H2339		SALLY	DRY		8-04-83						
0031	29H1928		6-28-82	6	112	166	4	2	12-11-82	11H2354		UNA	7-29-83		9-17-83	366	119	108	31		
0054	29H2434		12-05-82	2	55	55	0	1	1-29-83	11H0466	+229	VENUS	9-16-83		11-05-83	206	110	90	62		

AGRICULTURAL EXTENSION SERVICE

UNIVERSITY OF MINNESOTA

Dairy Extension
Department of Animal Science
101 Haecker Hall
1364 Eckles Ave.
St. Paul, Minnesota 55108

(612) 373-1014

March 5, 1984

NEW DHI HERD SUMMARY

TO: County Ag. Agents and DHI Supervisors

RE: Enclosed John Dairyman Copy of New DHI Herd Summary

A new DHI Herd Summary has been developed to provide additional management information and to include protein data. New items include:

1. Sample day % protein and protein pounds are listed on the left side of the page. After June 1 herds will have completed a full year of protein testing so rolling herd average % protein and pounds protein will be printed. Also the rolling herd average protein and milking cows only % protein by sample dates will be printed in the box at the lower right hand side of the page.

2. The Current SCC Evaluation by age groups shows the percent of negative cows (linear scores 0,1, & 2); suspicious cows, (linear scores 3 & 4); positive cows (linear scores 5 & 6); and very strong positive cows (linear scores 7, 8, and 9). Soon the sample day milking cows only linear SCC averages will be printed in the column provided. The sample day average shows SCC changes over time. Seasonal effects, days in milk, management and equipment changes can all cause SCC changes.

3. The Reproductive Summary box has several new additions to help dairymen to analyze and improve the reproductive status of their herds.
   a) A new line - Problem Cows - summarizes those cows which are more than 120 days post calving, either not bred or bred but not confirmed pregnant.
   b) Average days to first heat after calving shows possible problems in management, nutrition, and/or heat detection. Dairymen and DHI supervisors must work together to report all heat and breeding dates when they occur to use the Reproduction Summary to improve calving intervals.
   c) Services per conception is the average number of breedings per pregnancy.
   d) Cows with extended calving interval are those with projected calving intervals of more than 14 months (425 days).
   e) Percent of cows in heat by 60 days is the percent of cows reported in heat by 60 days post calving.
   f) Repeat breeders are those cows bred 3 or more times.

   Remember that cows to be culled may be listed as "Do Not Breed" by supervisor memo and are removed from the reproductive averages. Again, complete reporting of all reproductive information, heat and breeding dates, is a must for full use of reproductive data.

4. The Standardized ME (Mature Equivalent) averages at the upper right of the sheet compare 1st calf heifers with older cows using protein data in addition to milk and fat. Dollar value is calculated using National Sire Summary prices for milk, fat, and protein. Milk records are projected to 305 days, adjusted for age and month of calving, then standardized to 3.5% fat and 3.2% protein. Both M.E. milk and dollar values are for within herd comparisons only.

5. Peak ratio is first lactation milk peak divided by the other cows' peak. Low ratios (below 70%) suggest thin, poorly grown heifers or heifers with limited genetic potential. High ratios (over 80%) indicate heifers of high genetic potential or older cow management problems, mastitis, or underfed early lactation cows, etc.

6. Percent of milk sold (Daily Herd Totals) is milk sold divided by DHI milk. Herdowners can then subtract milk not sold--home consumption, fed to calves, etc.--for better comparisons. Too much difference may suggest milk meter problems, reading foam, etc.

The back side of each Herd Summary mailed has the same explanatory information as this "John Dairyman" Report. Encourage your dairymen to study and use it.

If you have questions, contact us.

Sincerely,

Bill Mudge

J. William Mudge
Extension Animal Scientist - Dairy Management

JWM:jf

Enclosure

UNIVERSITY OF MINNESOTA

Dairy Extension
Department of Animal Science
101 Haecker Hall
1364 Eckles Ave.
St. Paul, Minnesota 55108
(612) 373-1014

Dairy Update

THE NEW DHIA REPRODUCTION REPORT

October, 1983
Issue 68

J. K. Reneau and G. R. Steuernagel
Extension Dairy Specialists

Over the past 13 years the mean calving interval for Minnesota DHIA herds has changed very little. (Table 1). The average actual calving interval for Holstein cattle has been 12.76 months with a range of 12.7 to 12.9 months. Relatively speaking, this is reasonably good reproductive performance. Minnesota calving intervals compare favorably with other midwestern states (Table 2).

Table 1. Calving Intervals in Minnesota Holstein Herds From 1970 to 1983.

1970-1971	12.7	1977-1978	12.7
1971-1972	12.7	1978-1979	12.8
1972-1973	12.7	1979-1980	12.8
1973-1974	12.8	1980-1981	12.8
1974-1975	12.8	1981-1982	12.8
1975-1976	12.8	1982-1983	12.9
1976-1977	12.7		

Average = 12.76 Months

Table 2. Measure of Reproductive Performance in Midwestern DHIA Herds. (1982-83 data).

	CI	Ave. Day 1st Service	Average Services per Conception	Estimated Conception Rate %	Heat Detection Index %
*Arkansas	13.2	85	1.8	55	
*Illinois	13.3	88	1.8	55	
*Iowa	13.1	85	1.7	59	
*Kansas	13.3	81	1.9	53	
Minnesota	12.9	86	1.7	59	45
*Missouri	13.2	83	1.8	55	
*Nebraska	13.3	84	1.8	55	
*North Dakota	13.2	85	1.8	55	
*Oklahoma	13.3	83	1.9	53	
*South Dakota	13.1	84	1.7	59	

*Data from Mid-States DHIA Processing Center.

The ideal calving interval for maximum productivity is 12 months. If calving intervals are maintained below 13.0 months, there is probably not a severe economic loss. However, there is certainly significant loss when calving intervals extend beyond 13 months. Estimates on economic losses for calving intervals extending beyond the ideal 12 months have ranged between $3.00 - $5.50 loss per cow per day.

The average calving interval for Holstein herds during 1982-1983 in Minnesota was 12.9 months (Table 2). Over 40% of those herds had calving intervals exceeding 13 months with only a very small percentage of herds at the ideal 12 month level. Obviously, there is still considerable room for improved reproductive performance among Minnesota dairy farms.

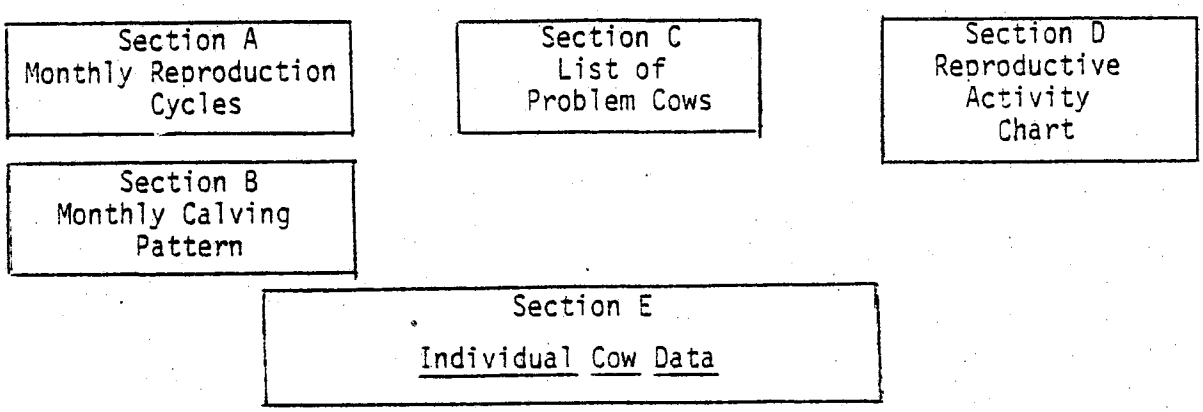
Some of the main factors influencing calving interval are heat detection, conception rate, days to first breeding, and culling for reproductive failure. These measures of reproductive performance have been displayed on the DHIA Herd Summary since 1976. When the reporting of reproduction information by the farmer and DHIA supervisor has been timely and accurate, these values have been helpful assessments of reproductive performance. The most serious criticism of these values is that they are historical in nature and, having been averaged over the past 12 months, may not always reflect recent reproductive performance in the herd. Therefore, they have not been useful monitors of reproductive performance in providing early warning of reproductive failure.

Retrospective study of reproductive performance in Minnesota DHIA herds reveals that the greatest obstacles to achievement of a 12 month calving interval are poor heat detection and too great a delay in days to first breeding. The changing of current trends and improved reproductive performance will require an increased educational focus on these factors. First, educators and dairy farmers must appreciate the inter-relationship between heat detection, conception rate, and days to first breeding and secondly, there needs to be a record system that gives both historical and current accounting of herd reproductive performance.

The Herd Summary will continue to carry the historical measures of reproductive performance. The new DHIA Reproduction Report is designed to provide more current information facilitating early identification of problems in reproductive performance, thus enabling timely correction in either management deficiences or reproductive disease. Individual problem cows as well as recent herd trends will be emphasized.

The format of the Reproduction Report is very similar to the SCC Report. Herd summaries predominate the upper portion of the report while detailed individual cow data are listed in the lower portion of the report. (Figure #6), page 11. The report will be discussed by section. (Table 4). Examples will be given to illustrate its usefulness.

Table 4.



Section A. Monthly Reproductive Cycles.

Figure 1.

JOHN DAIRYMAN — HERD CODE 41-00-0017 — MAIL DATE 7-07-83

101 HAECKER HALL — ST PAUL

MINNESOTA
DAIRY HERD IMPROVEMENT
DHI 230 11/82

REPRO

MONTHLY REPRODUCTIVE CYCLES

MONTHS	JUN	JUL	AUG	SEPT	OCT	NOV	DEC	JAN	FEB	MAR	APR	MAY	JUN
Est Num Heats	0	0	1	1	5	6	8	10	12	11	10	7	4
Reported Heats	0	0	0	0	1	6	4	4	7	3	6	5	4
Num Breedings	0	0	0	0	1	6	4	4	7	9	6	5	4
Num Conceived	0	0	0	0	1	2	2	3	4	3	5	1	1

Timely recognition of reproductive failure or infertility is crucial to avoiding serious losses due to reproductive inefficiency. The use of routine veterinary herd fertility programs will facilitate early recognition of clinical reproductive disease (cystic ovaries, metritis, etc.) so that timely treatment will lessen the number of days affected cows stand open. Herd specific vaccination programs will lessen infertility and abortions due to subclinical diseases. Early pregnancy diagnosis (prior to 42 days) will reduce days lost due to presumed pregnancy. However, even the most skilled farm managers - veterinarian teams will be unable to significantly improve reproductive performance without the use of good records. Records not only serve to monitor the success or failure of veterinary procedure, but also define reproductive management deficiencies which must be remedied if total success is to occur.

Poor heat detection is the greatest single obstacle to successful A.I. programs. Minnesota studies involving large numbers of cows show that detection of heat is more of a management problem than a cow problem. Ninety percent of all cows thought to be anestrus were cycling normally. Only 10% of supposedly anestrus cows were actually not cycling as a result of some pathological problem.

Well fed and healthy cows will normally begin to cycle by approximately 20 days post partum. Not all of these early ovulations are accompanied by strong heat signs. However, by 60 days post partum, nearly 100% of normal cows are cycling and expressing normal heat signs. Whether or not these cows are observed in heat depends on the intensity of heat detection effort. This fact is clearly verified in a summary of three studies found in Table 5.

Table 5. Percentage of normal cows detected in heat at first, second, and third ovulation when maintained under different system of observation.

Observation System	Ovulation		
	First (20 days)	Second (44 days)	Third (64 days)
(1) Continuous 24 hr. observation			
(A) King, et al.	50%	84%	100%
(B) Williamson, et al.			100%
(2) Casual (herdsman)			
(A) King, et al.	20%	44%	64%
(B) Williamson, et al.			56%
(C) Morrow, et al.	23%	46%	64%

Notice in the summary of monthly reproductive cycles (Figure #1) that an estimate of the expected number of heats for that month in the herd will be calculated. This figure will represent the number of heats theoretically possible beginning with the first reported heat date or on day 60 post partum if no heat date is reported prior to 60 days. Reported heats are those heats where the cow is observed in heat and recorded but not bred, or where the cow was both observed in heat and also bred. Reported heats divided by the estimated number of theoretical heats times 100 will give the percentage of cows detected in heat. The new DHIA reproduction report will allow monthly monitoring of heat detection efforts. For example, the John Dairyman herd (Figure #1) had 100% heat detection in November and 50% in December. This herd's yearly average heat detection index was 61%. The present average heat detection performance among DHIA herds is a dismal 45%. In general, the heat detection performance is better on the high producing herds (Table 6) although improvement could be made on these farms as well.

Table 6. 1983 Heat Detection Performance on Holstein DHIA Herds in Minnesota at Various Levels of Production.

	Rolling Herd Averages by Thousand lbs. Milk			
	11-12	14-15	17-18	720
Number of herds	316	1169	544	56
Cows per herd	44	56	53	55
Heat detection index	39%	45%	52%	52%

Good heat detection is a function of a complete awareness of the physical and behavioral signs of heat and the time spent looking for cows in heat. Since the behavioral sign of a cow standing firmly while another cow mounts (standing heat) is the most reliable sign of heat, it would seem obvious that to do a good job at heat detection, cows must be able to interact. Table 7 nicely demonstrates the relationship between the numbers of daily observation and the percent of cows detected in heat. If you are observing cows only once per day for 20-30 minutes, you are missing one half of the cows in heat. An excellent heat detection goal under Minnesota dairying conditions would be 80%. Monthly reminders of heat detection performance will help dairy farmers improve reproductive performance.

Table 7. Relative Efficiency of Heat Detection Schemes.*

Heat Detection Scheme	% Correctly Found in Heat
Continuous 24 hr. observation	98-100%
Observed three times daily	90%
Observed two times daily	80%
Observed once daily	50%

*These figures are based on the assumotion that cattle being observed for heat are allowed to freely interact.

Also listed in the Monthly Reproductive Cycle Chart (Figure #1) is a monthly compilation of the number of cows bred as well as the number of cows that became pregnant as a result of those breedings. Obviously, this enables easy calculation of conception rates on a monthly basis. This is useful information but must be interpreted with caution. The average expected conception rate under normal conditions would be 60-65%. This would reflect good reproductive performance. Conception rates calculated on a monthly basis in a small herd will most likely vary considerably. For example, the conception rate for one particular month in a herd might be as high as 80-100%. If this does occur, one should realize that this level of performance should not be routinely expected. More than likely this is a statistical phenomenon quite similar to the situation in which a farmer got 80% heifer calves in one particular calving season. Over the long run, we know that the average would be closer to 50% heifers. Likewise, the average expected conception rate under good conditions would be 60-65%. However, we should expect a somewhat higher conception rate in heifers than older cows. We also know that some bulls are more fertile than others.

What if conception rate drops below 50% in any month as was the case in the John Dairyman herd in November, March, May and June? Then we ought to try to determine why. If during that month the cows bred were older cows or were cows that had experienced post partum uterine disorders such as retained placentas or metritis, then one should not be too surprised to see lower conception rates in these individuals. Perhaps high ambient temperature and humidity had a detrimental influence on conception rate or early embryonic death in May and June. In Arizona dairies, for example, conception rate is reduced to as low as 10-20% during the hot summer months. But if monthly conception rates are low and they cannot be easily explained, then other things must be considered. The timing of A.I., A.I. technique, poor quality semen or faulty semen handling should be considered as possible explanations. Nutritional factors may also need attention.

If no management or physiological factors can be found, one can then be content that observation is a statistical phenomenon similar to the farmer who got all the bull calves this particular year and there is really nothing that can be done about it. The point is that although on occasion, concerns may arise unnecessarily with this type of reporting system, there should be sufficient warning so that appropriate and timely action could avoid reproductive disaster.

Section B. Monthly Calving Pattern.

Figure 2.

Jun'82 Jul Aug Sept Oct Nov Dec Jan Feb Mar April May Jun'83

MONTHLY CALVING PATTERN

Cows Calved Last	1	0	3	1	2	1	6	2	0	0	0	0	2
Heifers Calved Last	0	0	0	1	1	2	1	0	1	3	0	0	0
Cows To Calve	0	0	2	3	2	3							
Heifers To Calve	0	0	0	1	1	2							

Jun'83 Jul Aug Sept Oct Nov'83

This section evaluates the monthly calving pattern of cows and heifers (figure #2). It is a historical account of the numbers of cows and heifers that has occured over the past 13 months as well as the anticipated number of cows and heifers expected to calve during the next 6 months.

Calving patterns may be helpful in managing labor or in anticipating or adjusting milk flow. The planning of calving management as well as heifer breeding and labor will be facilitated by recording of monthly calving patterns.

Section C. Problem Cow List.

Figure 3.

LIST OF PROBLEM COWS

BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN
MJLLY	*195				
PLUTO	*161				

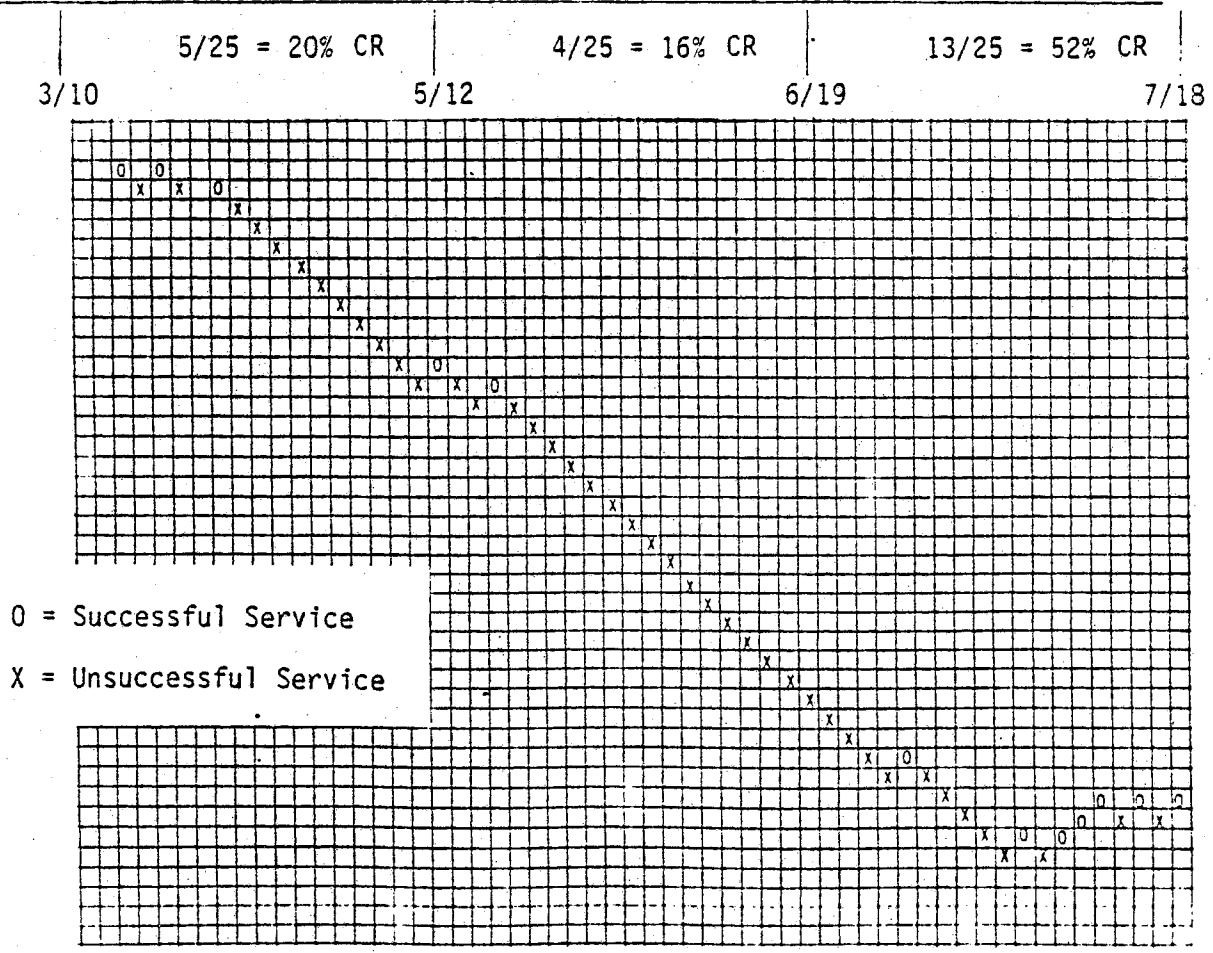
The problem cow list is an effective means of focusing attention on those individuals in the herd that are most hindering reproductive performance. Note the cows Molly and Pluto (figure #3) in the John Dairyman example herd. This list consists of heifers or cows in the herd that are greater than 120 days open and not confirmed pregnant or that began a lactation by abortion or premature calving. These cows are listed in calving order so that those of greatest concern are listed first. Those listed with an asterik next to the number are problem cows that are bred but not confirmed pregnant. Study of the problem list gives an indication of the depth of the reproductive problem. In comparing two herds each with 13.5 month calving intervals, one would be more concerned about reproductive management when the list of problem cows is numerous compared to the herd with one or two cows with excessively long calving intervals.

It is important to be able to get an assessment of herd performance at a glance. Graphic presentations of herd summary data often are the most effective means of calling attention to both strengths and weaknesses in reproductive management.

Construction of a Q Sum Graph (figure 4) is a simple method of keeping abreast of recent herd reproductive performance trends. This can be used to supplement DHIA reproductive records. The success or failure of successive breedings are charted on graph paper by beginning at an arbitrary reference point. With each diagnosed pregnancy, a circle is drawn in a square to the right and up. A pregnancy failure is indicated by an X marked to the right one square and down. Such a graph is demonstrated on the following page.

Section D. Reproductive Activity Chart.

Figure 4. Q Sum Graph

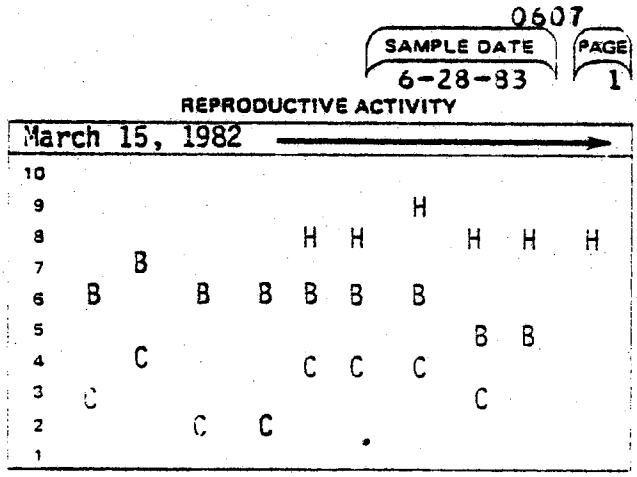


Failure trends are quickly noticed allowing correction of problems before a disaster occurs. This particular graph (Figure #4) was constructed in retrospect in an attempt to solve reproductive problems on one dairy farm. The dairyman had begun doing his own A.I. sometime in March and had serious A.I. technique problems which did not get resolved until June. Conception rates between 3/10 and 5/10 were 20% and from 5/10 to 6/19 were 16%. Conception rates after June 19th were 52%. Had performance been monitored with the Q Sum graph, the problem may have been discovered and corrected sooner.

Q Sum graphs can be adapted readily to micro-computer technology but are awkward when the printout is confined to a small space. The Reproductive Activity Chart (Figure #5) found on the upper right hand corner of the new DHIA Reproduction Report is meant to be used in a similar manner that Q Sum graphs are used. The graph consists of ten columns with 10 squares per column. Each square represents the last 100 theoretically estimated heats in the herd since the date printed to the left of the arrow found in the top of the chart. For example, in the John Dairyman example herd, each square represents the last 100 estimated heats

in that herd since March 15, 1982. Since the John Dairyman herd is relatively small (27 cows) the chart represents the reproductive activity in that herd over the past 15 months. On each column of 10 heats the letter H is placed over the number of observed and recorded heats. The letter B is placed over the number of heats that were not only observed but a breeding took place. The letter C is placed over the number of heats in which a breeding took place and the cow conceived.

Figure 5.



This enables you to spot poor reproduction performance early enough to get corrective measures in place before the entire herd is in trouble. For example, if in the first column of ten heats an H was placed over the 7th space we would know that heat detection was approximately 70%. If a B was placed in the 5th space this would mean that 5 cows were bred. If a C was placed over the 3rd space then we would know that 3 of the 5 bred cows (60%) had been confirmed pregnant. This would be good reproductive performance. Notice in the John Dairyman herd that there are no H's placed in the first 4 columns. During this period of time, heats were only recorded when cows were bred. Note that in the first two columns conception rate was acceptable (50-55%) but that conception rate declined over the next 20 heats (12 breedings) to 33%. A management change was then made and all heats recorded. Conception rate improved. It is a well known fact that when prebreeding heat dates are recorded, conception rates usually improve 10-15%, presumably because the subsequent more accurate heat detection at the time of breeding enable better timing of A.I.

As was pointed out in the example of the herd plotted on the Q Sum graph, dairy farmers beginning their own A.I. should carefully monitor their results. Rapid decline in conception rate should be a warning that A.I. technique or semen handling may not be correct. It is hoped that this chart will be helpful to both farmers and those with whom they consult on reproductive matters in assessing the herd's reproductive performance at a glance.

Section E. Individual Cow Data.

Individual cow data (Figure #6) other than breeding dates and projected calving dates has not been reported on previous Minnesota DHIA reports. The new DHIA Reproduction Report has considerably more individual reproduction information than offered before. Most of that information is self-explanatory however, it may be of value to highlight how some of this information may be used, particularly to improve reproductive performance.

Assuming all heats observed are being recorded by the farmer and also are being accurately transferred to the DHIA barn sheet by the DHIA supervisor, the study of the days to first heat would be interesting. Even more revealing would be a calculation of the average days to first heat on a herd basis. One hundred percent of normal cows will show standing heat by 60 days post partum. (Table 8).

Table 8. Standing Estrus at First Heat Postpartum.

Postpartum Days	Type of Estrus	
	Nonstanding %	Standing %
1-20	64%	36%
21-40	15%	85%
41-60	11%	89%
61+	0%	100%

Lauderdale, 1974.

Therefore, the percentage of cows seen in heat by 60 days post partum is an excellent reflection of either the herd's reproductive health or the heat detection efficiency. For example, in a high producing herd, you may find that the percentage of cows showing heat is acceptable, but the farmer is complaining that the cows are not showing heat well at the time he would like to begin breeding (60-70 days post partum). It could be that heat detection in this herd is adequate but there is need for adjustment in early lactation feeding to maximize DM intake thus minimizing a negative energy balance with its subsequent depression of heat signs.

As previously cited, 90% of all cows not seen in heat are cycling normally but are not being observed. The column labeled "Missed Heats" (Figure #6) will serve to emphasize this fact. In a few cases where there is reproductive pathology (cystic ovaries, etc.) or stress related reproductive inactivity, cows will be listed as having been missed in heat when this may not be true. For example, it is not uncommon to find first calf heifers with completely inactive ovaries due to the stresses resulting from adjustments to stall barn living or to the lactation ration, recuperation from a difficult calving, or needing nutrients to continue growth. In such cases, rectal examination of the cow or heifer by a veterinarian will determine if the cow is truly anestrus or being missed because of poor heat detection.

Repeat breeders are defined as those cows that are cycling normaly, are showing heat normally but have not become pregnant after being bred three times. Repeat breeder cows are common among Minnesota dairy herds. Normal incidence is 10-15%. The column labeled "Times" under the "Last Breeding or Heat" column can be used to identify repeat breeder cows and to calculate the percentage of repeat breeders in the herd. Several large studies have shown that repeat breeder problems tend to occur in older high producing cows. It is common for a cow to habitually have repeat breeder problems year after year. These studies also showed that there is an increase in repeat breeder cows as herd size increases.

The most common cause of repeat breeders is faulty heat detection. Hormone test of milk samples collected at the time of insemination show that nearly 20% of cows bred were not even in heat. The timing of insemination should be based on standing heat, that is, a cow standing firm with all four legs braced while mounted by another cow. For best A.I. results, cows should be inseminated in the middle or last half of standing heat. Some dairymen inseminate cows based on non-standing signs of heat such as mounting other cows, hyperactivity, mucus discharge or a swollen vulva, etc. This leads to poor timing of A.I. and many repeat breeders. These factors should be considered when the incidence of repeat breeders is greater than 10%.

The future of any herd is determined by the quality of the bulls used. The DHIA Herd Summary since 1976 has been printing the average PD$ service sires being used in the herd. This information has been excellent as a monitor of the general breeding policy in the herd. The new DHIA Reproduction Report will report PD$ of service sires on the last breeding of every cow. This will add greater depth to the DHIA genetic information.

Pregnancy diagnosis is an important part of the herd fertility program. Equally important is the determination that a cow previously bred and thought pregnant is open. The discovery of an open cow 42 days after breeding or sooner is important to minimizing days open. In a herd on a monthly veterinary reproductive health program, the range of days from breeding to the pregnancy exam should be a maximum of 35 days - 70 days. The exam of most value is that one prior to 42 days post breeding so that timely treatment and/or more intensive observation for heat can prevent undue loss of time. Examinations of cows for pregnancy beyond 2 months post breeding will not be as effective in helping maintain low calving intervals.

The last four columns, "Days in Milk", "Production Index", "Peak Milk" and "Sample Day Milk (Actual) (Expected)", are useful in culling aids.

Overall, the new DHIA Reproduction Form offers many new dimensions in better understanding and managing dairy farm reproduction. As time goes on, we are certain that DHIA reproduction information will become more vital to the management success of every Minnesota dairy farm.

JOHN DAIRYMAN
HERD CODE 41-00-0015 | MAIL DATE 7-07-83

101 HAECKER HALL ST PAUL MN 55108

MINNESOTA
DAIRY HERD IMPROVEMENT
DHI 230 11/82

REPRODUCTION

SAMPLE DATE 6-28-83 | PAGE 1

MONTHLY REPRODUCTIVE CYCLES

MONTHS	JUN	JUL	AUG	SEPT	OCT	NOV	DEC	JAN	FEB	MAR	APR	MAY	JUN
Est Num Heats	0	0	1	1	5	6	8	10	12	11	10	7	4
Reported Heats	0	0	0	0	1	6	4	4	7	9	6	5	4
Num Breedings	0	0	0	0	1	6	4	4	7	9	6	5	4
Num Conceived	0	0	0	0	1	2	2	3	4	3	5	1	1

MONTHLY CALVING PATTERN

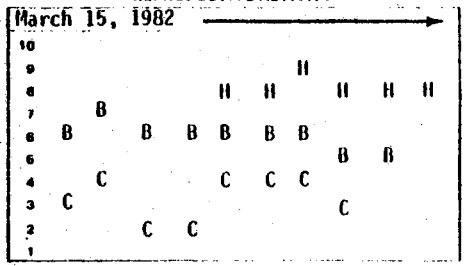
Cows Calved Last	1	0	3	1	2	1	6	2	0	0	0	0	2
Heifers Calved Last	0	0	0	1	1	2	1	0	1	3	0	0	0
Cows To Calve	0	0	2	3	2	3							
Heifers To Calve	0	0	0	1	1	2							

LIST OF PROBLEM COWS

BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN	BARN NAME	DAYS OPEN
MOLLY	*195				
PLUTO	*161				

REPRODUCTIVE ACTIVITY

March 15, 1982 →



COMPUTER NUMBER	COW'S SIRE ID	COW'S SIRE PD$	DATE CALVED	LACT NUM	DAYS TO 1ST HEAT	DAYS OPEN	NUM HEATS MISSED	LAST BREEDING OR HEAT TYPE	LAST BREEDING OR HEAT DATE	LAST BREEDING OR HEAT SERVICE SIRE ID	LAST BREEDING OR HEAT SERVICE SIRE PD$	BARN NAME	DATE TO DRY	DAYS BRED TO PG EXAM	DUE DATE	DAYS IN MILK	PROD INDEX	PEAK MILK	SAMPLE DAY MILK ACTUAL	SAMPLE DAY MILK PROJECT	REMARKS
0069	APACHE		9-04-82	1	68	107	0	3	12-20-82	7H1115	+173	APACHE	8-07-83		9-26-83	298	97	58	32		
0077	170365	+180	3-06-83	1	93	93	2	1	6-07-83	21H380	+186	CB			POSS PG	115	83	58	46		
0049	29H2417	+161	1-16-83	3	52	72	0	2	3-29-83	10H6335		CRISCO	11-14-83	35	1-03-84	164	96	98	68		
0051	29H2510	+190	6-16-83	5		13						DANA				13		77	77		
0070	FORD		10-17-82	1	89	89	1	1	1-14-83	29H3820	+206	DEANNA	9-01-83		10-21-83	255	96	57	47		
0043			12-14-82	3		70		2	2-22-83	23H217	+178	EFFIE	10-10-83		11-29-83	197	99	97	64		
0072	29H2477		11-03-82	1								ELOISE	***** DO	NOT	BREED **	238	86	66	43		
0062			9-24-82	3	60	199	4	4	4-11-83	11H2238		ELSIE	11-27-83	64	1-16-84	278	100	91	34		
0071	40H2789		11-02-82	1	75	171	2	4	4-22-83	40H2763	+95	EMY	12-08-83	53	1-27-84	239	107	77	60		
0053	ART		8-25-82	2	66	66	0	1	10-30-82	11H2354		FAWN	DRY		8-06-83						
0033	29H1879		10-25-82	4	49	149	2	4	3-23-83	11H2238		GYPSY	11-08-83	41	12-28-83	247	101	91	54		
0061	KNIGHT		8-24-82	2	87	87	1	1	11-19-82	40H2763	+95	HOLLY	7-07-83		8-26-83	309	99	75	29		
0062	ART		2-05-83	2	76	76	1	1	4-22-83	40H2455		IRIS	12-08-83	53	1-27-84	144	95	83	66		
0052	STARWAR		6-14-83	3		15						JANE				15		93	93		
0056	STARWAR		9-27-82	2	83	83	1	1	12-19-82	29H3820	+206	JILL	8-06-83		9-25-83	275	91	79	21		
0066	BILLY		10-30-82	2	40	80	1	2	1-18-83	40H2763	+95	JODY	9-05-83		10-25-83	242	109	90	55		
0060	KNIGHT		12-31-82	2	78	78	1	1	3-19-83	11H2238		KIM	11-04-83	45	12-24-83	180	127	113	71		
0078	17H248	+133	3-12-83	1	48	48	0	1	4-29-83	10H6335	+222	LORI			POSS PG	109	90	59	52		
0075	29H2389		2-04-83	1	70	92	1	2	5-07-83	29H4000	+317	MAGDA	12-23-83	38	2-11-84	145	94	68	51		
0076	40H2652	+148	3-01-83	1		108	0	3	6-17-83	10H5801	+164	MAMIE			POSS PG	120	88	55	49		
0040	29H2434		12-13-82	3	50	127	3	2	4-19-83	29H3645	+146	MANDY	12-05-83	56	1-24-84	198	99	87	59		
0064	BOQUET		12-10-82	2	53	195	2	6	6-23-83	11H2521		MOLLY			POSS PG	201	107	88	61		
0059	ERIC		11-24-82	2	59	80	0	2	2-12-83	17H387	+215	NICKY	9-30-83		11-19-83	217	119	105	65		
0068	ERIC IW		12-25-82	2	91	91	1	1	3-26-83	29H3303		OLIVE	11-11-83	38	12-31-83	186	98	88	65		
0065	KNIGHT		1-15-83	2		161	3	3	6-25-83	10H6466	+181	PLUTO			POSS PG	165	104	93	66		
0073	BUTERDA		12-27-82	1	87	106	1	2	4-12-83	10H6335	+222	RITA	11-28-83	53	1-17-84	184	94	61	49		
0058	GERRI		8-12-82	2	77	77	1	1	10-28-82	11H2339		SALLY	DRY		8-04-83						
0031	29H1928		6-28-82	6	112	166	4	2	12-11-82	11H2354		UNA	7-29-83		9-17-83	366	119	108	31		
0054	29H2434		12-05-82	2	55	55	0	1	1-29-83	11H0466	+229	VENUS	9-16-83		11-05-83	206	110	90	62		

HERD CODE	TYPE OF RECORD	ASSOC	SUPVR	LAB	CO	SC DAY
41-00-0005	OFFICIAL DHI	62	859	4	62	

TEST INTERVAL	LAB DATE	RECEIVED	MAILED
11-04	12-07	12-07	12-09

AGRICULTURAL EXTENSION SERVICE
UNIVERSITY OF MINNESOTA
WILLIAM MUDGE
EXTENSION DAIRYMAN

REPORT	
11-05	125

OPTIONS
ACTION LISTS
SCC

JOHN DAIRYMAN
101 HAECKER HALL
ST PAUL
MN 55108

DAIRY HERD IMPROVEMENT

HERD SUMMARY

SAMPLE DATE	LAB
12-07-83	7.38
NUMBER COWS: 41	COMPOSITE: 14.60
COW-DAYS	TOTAL: 21.98

DHI 102
6-76

PRODUCTION, INCOME AND FEED COST SUMMARY

DESCRIPTION	SAMPLE DAY AVG PER COW	DHI ROLLING HERD AVG (11 TESTS)
NUMBER COWS	41	42.7
% COWS IN MILK	85	88
MILK LBS	49.2	16377
% FAT	3.84	3.79
% PROTEIN	2.95	
FAT LBS	1.89	621
PROTEIN LBS	1.45	
DRY FORAGE LBS (100 PCT DM)	12	3626
HAY SILAGE LBS (100 PCT DM)	17	2371
CORN SILAGE LBS (100 PCT DM)	16	2660
OTHER FORAGE LBS (100 PCT DM)		
GRAIN LBS (100 PCT DM)	22	6670
FORAGE DM PER CWT BW	1.8	1.8
ENERGY INDEX	110	116
PROTEIN INDEX	115	125
MILK PER LB GRAIN DM	2.5	2.5
VALUE OF PRODUCT $	6.43	2112
TOTAL FEED COST $	2.34	834
INCOME OVER FEED COST $	4.09	1278
FEED COST PER CWT MILK $	4.76	5.09
MILK PRICE PER CWT $	13.07	12.90

MANAGEMENT INFORMATION

SAMPLE DAY FEED	AVG LBS CONSUMED	PCT DM	NET ENERGY	CRUDE PROTEIN	COST $/TON
HAY	12	85	50	17	69
CORN SILAGE	16	44	65	8	28
HAY SILAGE	17	40	46	16	30
GRAIN INDIV FED	22	90	78	*14	132

* 15% CRUDE PROTEIN RECOMMENDED

SUMMARY OF COWS NOW IN HERD

LACT NO	NUMBER COWS	PROJECTED 305-2X-ME MILK	FAT	INDEX	AVERAGE AGE	% IDENTIFIED SIRE	DAM
1ST	17	16743	647	98	2-03	100	100
OTHER	24	17844	666	105	4-07	100	100
ALL	41	17392	659	102	3-07	100	100

COWS MILKING ON SAMPLE DAY

LACT NO	NUMBER COWS	AVG DAYS IN MILK	AVG LBS MILK	AVG PEAK LBS MILK
1ST	15	147	47	56
OTHER	20	106	65	76
ALL	35	124	58	68

CURRENT MASTITIS EVALUATION

LACT NO	NUMBER COWS	PERCENT COWS NEGATIVE	SUSPECT	POSITIVE	V STRONG
1ST	15	40	53	0	7
OTHER	20	40	50	10	0
ALL	35	40	51	6	3

YEARLY SUMMARY

LACT NO	COWS ENTERING HERD NUMBER	%	COWS LEAVING HERD NUMBER	%
1ST	17	40	8	19
OTHER	0		11	26
ALL	17	40	19	44

REPRODUCTIVE SUMMARY

	NUMBER COWS	AVG DAYS SINCE CALVING	NUMBER COWS OPEN <60 DAYS	60-120 DAYS	>120 DAYS	NUMBER COWS BRED 1 TIME	2-3 TIMES	4+ TIMES	COWS - BREEDING INTERVAL <18 DAYS	18-24 DAYS	>24 DAYS	CALVED TO 1ST BRED DAYS	CALVED TO LAST BRED DAYS	MINIMUM CALVING INTERVAL MONTHS
PREGNANT COWS	11	301	1	8	2	8	2	1		1	2	77	110	12.9
POSSIBLY PREGNANT	14	121	2	9	3	10	4				2	85	101	12.6
OPEN COWS	16	76	7	6	1									

MAXIMUM CONCEPTION RATE = 66% HEAT DETECTION INDEX = 38%

AVERAGE AI SIRE PREDICTED DIFFERENCE

SIRE	NUMBER	MILK	DOLLAR
SERVICE	24	1803	227
1ST LACT	17	1046	132
OTHER	24	802	93

COWS DRY BEFORE CALVING

NO COWS	AVG DAYS DRY	<40 DAYS	40-70 DAYS	>70 DAYS
21	63	1	15	5

BREED OF HERD	HOL	AVERAGE BODY WT	1310

DAILY HERD TOTALS

MILK SOLD	DHI MILK	GRAIN	INCOME OVER FEED COST
	2017	902	168

SAMPLE DAY PRODUCTION

SAMPLE DATE	TOTAL COWS	% IN MILK	MILKING COWS ONLY AVG DIM	MILK	%	DHI ROLLING HERD AVG ENTIRE HERD MILK	%	FAT
12-07-83	41	85	124	58	3.8	16377	3.8	621
11-03-83	42	88	117	57	3.7	16525	3.8	626
10-07-83	40	83	130	55	4.0	16412	3.8	620
9-02-83	43	72	175	46	3.6	16228	3.8	611
7-23-83	44	73	189	43	3.7	16353	3.8	614
6-04-83	45	89	187	51	3.7	16594	3.7	621
5-05-83	45	93	190	48	3.8	16553	3.7	619
4-05-83	41	93	201	48	3.8	16546	3.7	618
3-03-83	42	90	177	55	3.8	16443	3.7	612
2-02-83	41	93	164	54	3.8	16346	3.7	606
1-05-83	42	93	143	55	3.9	16367	3.7	603
12-04-82	42	98	124	58	3.8	16298	3.7	598

NUTRITION FOR THE WELL AND SICK CALF DURING THE SUCKLING PERIOD

Thomas H. Herdt DVM, MS
Department of Large Animal Clinical Sciences
Michigan State University

The liquid feeding period

I. Colostrum period - first three days

A. Immunoglobulin transfer - The importance of colostrum for immunoglobulin transfer is well recognized and will not be reviewed here.

B. Other immune factors

1. complement - Calves are born with low serum complement concentrations, which increase following the consumption of colostrum, a rich source of complement.

2. lymphocytes - Colostrum contains lymphocytes which are absorbed through the intact intestinal epithelium. These may transfer some degree of cellular immunity.

C. Nutritional factors

1. Vitamin A - Calves are born with essentially no vitamin A stores and are dependent upon immediate supplementation. Under natural conditions they receive this supplementation from colostrum, a rich source of vitamin A.

2. Other nutrients - Colostrum is a more concentrated source of most nutrients than milk.

3. Amount - During the first day or two of life there is no reason to limit the consumption of colostrum. This is one case where the owner can be assured that some is good and more is better. There may be some loose feces associated with colostrum feeding, but this is physiological and probably serves to rid the colon of meconium.

II. The post colostral period

A. How much should be feed?

1. First, consider the energy requirement.

a. Maintenance requirement for an 80 to 100 pound calf
1800 to 2000 digestible Kcal/da
(23 Kcal/# body wt.)

b. Additiona requirements for growth

.5# per day - 681 Kcal
1.0# per day - 1362 Kcal

(1362 Kcal/# gain)

c. temperature - The critical temperature is the temperature below which animals must catabolize energy sources, in excess of their body maintenance requirements, to maintain body temperature. The exact value of the critical temperature varies markedly and is dependent on such things as hair thickness, moisture in the hair coat, air movement and insulating quality of the environmental surfaces. For three day old calves housed under reasonably normal conditions, 55F is a good approximation of the critical temperature. As the temperature drops below this point, the number of calories required to maintain body weight increases. This increase is about 100 Kcal for every degree Fahrenheit below the critical temperature. Assuming a critical temperature of 55F, calves receiving what would be a maintenance diet under thermoneutral conditions would loose one pound per day at 17F.

2. Second, consider the energy content of the feeds.

a. Milk gross energy (G_E - Kcal/ pound fluid milk) formula:

$G_E = 1.102(41.84 \text{ fat\%} + 22.29 \text{ solids-not-fat\%} - 25.58)$

Example - Caloric content (in Kcal) of one quart or one gallon of 7.5% solids-not-fat milk at various fat concentrations

%fat	qt	gal
4.0	681	2724
3.5	635	2535
3.0	589	2556

b. Milk replacer gross energy (G_E - Kcal/pound dry powder) formula:

$G_E = 28.09 \text{ fat\%} + 8.15 \text{ protein\%} + 1730.5$

Examples illustrating the effect of fat concentration on the caloric content (Kcal) of one pound of dry powder or one gallon of reconstituted milk replacer.

%fat	# dry replacer	gal (1:7)*
20	2471	2471
15	2331	2331
10	1904	1904

* 1 pound of replacer per seven pounds water = 14.2% dry matter

The formulas presented above may be used to calculate the gross energy content of milk or milk substitute. The energy requirements given previously are in terms of digestible energy. Thus, a direct comparison cannot be made between the digestible energy requirements of the calf and the gross energy content of the feeds. However, in the case of whole milk, the digestibility is greater than 95%, so a rough comparison between digestible and gross energy contents can be made. For milk substitutes this is not as true. Even the best of them are less digestible than milk and some are considerably less digestible than milk. Most are in the range of 85 to 91% digestible, so some discount should be applied if the gross energy content is used to calculate the dietary energy received by the calf from milk substitutes.

3. Feed the calf at least enough to meet its maintenance requirements and preferably enough to allow some growth. The thumb rule of 10% of the calf's birth weight as whole milk or reconstituted milk substitute per day is valid and useful. Adherence to this rule will result in calves receiving a maintenance diet under most conditions. If milk or high quality milk replacer with 15 to 20% fat is feed, application of this thumb rule should result in some calf growth, even prior to significant solid feed intake.

B. What should be fed?

1. Cost

It costs about $44.00 to raise a calf to weaning weight, if milk is worth $13.00 per hundred weight. The cost of milk substitutes varies, but with most it will take $20.00 to $30.00 worth of feed to raise the calf to the same stage. (With some milk substitutes it may cost as much as $40.00.) Thus, the money saved from feeding a milk substitute is approximately $18.00 per calf. This is a small portion of the total cost of raising a heifer calf to freshening.

2. Quality

a. Protein

1. Gastric digestion in the preruminant calf - Milk, under the influence of rennin, clots rapidly (3 to 4 min.) in the abomasum. Initially, the clot traps fat and whey. As the clot, or curd, contracts the whey is squeezed out of it and subsequently passes into the small intestine. Eighty-five percent of the whey leaves the abomasum with in 6 hr of ingestion. The clot itself is slowly digested over a 6 to 12 hour period. While milk clot formation is not essential to proper digestion in the calf, it does have several beneficial effects. First, it provides for the slow release of nutrients from the abomasum. Second, it results in the predigestion of protein and fat, before they reach the abomasum. Both of these effects serve to enhance the efficiency of small intestinal function.

2. Protein ingredients

a. skim milk - This is an excellent source of protein for milk replacer. However, even skim milk can be poorly digested by the calf if the proteins have been denatured by heat during the drying process. If the whey proteins are damage by excessive heat, clot formation in the abomasum will not occur efficiently and protein digestion will be impaired.

1. Ways of evaluating skim milk powder

fiber

American Dry Milk Institute ratings
high heat - acceptable
medium heat - unacceptable
low heat - unacceptable

In vitro rennet response
Adjust the pH of 100 ml of reconstituted milk substitute powder to 5.5 using pH paper and hydrochloric acid. Warm the mixture to 39C. Dilute standard cheese making rennet 1:1 with water and add 2 ml of this to 100 ml of reconstituted milk substitute. Complete coagulation should occur within two minutes. Use whole milk as a control.

b. Whey protein - This product is made by precipitating the protein from whey. It is not equivalent to dried whey, although whey protein is included in dried whey. It is highly digestible by the calf. It does not, in the absence of casein, form an abomasal clot and thus, when included in high proportions in milk substitute powders, results in rapid passage of fat and protein from the abomasum.

c. Dry whey - dried whey can be included in milk substitute powders as a source of protein, as well as lactose and minerals. When it comprises more than 20% of the dry matter, an increased incidence of diarrhea may result.

d. Casein - Pure casein protein can be obtained and is sometimes added to milk substitute powders. It is a highly digestible protein which participates in curd formation.

e. Soy protein - several types of soy protein are used in the formulation of milk substitutes.

   1. Soy flour (44 to 48% crude protein) - Raw soy flour contains several undesirable constituents: trypsin inhibitor, hemagglutinins, other antinutritional factors and oligosaccharides. While the first of these can be at least partially inactivated by heat treatment, the oligosaccharides are not affected by heating. These complex sugars cannot be digested by the calf and thus proceed unaltered through the small intestine to the colon where they serve as substrate for bacterial fermentation. The protein in soy bean flour is poorly digested by the calf. While the digestibility of milk protein approaches 100% in the calf, the digestibility of soy flour protein is in the range of 72 to 75%.

   2. Specially processed soy flour - Alternate treatment with acid and base appears to increase the digestibility of soy flour protein and may alter the oligosaccharides. Many milk substitutes contain specially processed soy flour; it is more desirable than raw soy flour.

3. Soy protein concentrate - The protein in soy flour can be concentrated and some of the impurities, including the oligosaccharides, can be removed. This yields a product that should be more desirable for inclusion in milk substitutes than either soy flour or specially processed soy flour.

The performance of calves fed soy protein, of any kind, is never as good as that of calves fed milk protein. This is especially true in calves under three weeks of age. Some potential reasons for this are:

1. Lack of clot formation in the abomasum
2. antinutritional factors and oligosaccharides
3. hypersensitivity like reactions.

Calves feed soy protein develop antibodies to the protein and sprue like hypersensitivity lesions have been described in their small intestines. These observations have lead some people to believe that soy protein creates a hypersensitivity response in the intestine of most calves. This has lead to the frequent recommendation that calves less than three weeks old not be fed milk substitutes containing protein from sources other than cows milk.

f. Other protein sources

Fish protein, meat hydrolysates and single cell proteins (yeast and bacteria) have been evaluated for their use as protein sources in milk substitutes. While fish protein and single cell proteins may have some promise for this use, I know of no milk replacers currently for sale in the state of Michigan that contain protein from these sources.

b. Fat

High fat versus low fat milk substitutes - Some disagreement exists as to the desirability of using milk substitutes with more than 10% fat. The use of higher fat (15% and preferably 20% of dry powder) diets for calves should be generally recommended for the following reasons:

1) Fatty acids liberated from the digestion of fat suppress bacterial growth.
2) As the proportion of fat in the diet increases, the proportion of protein and carbohydrate decreases. This reduces the possibility of overloading the intestinal capacity for protein and carbohydrate digestion.
3) More energy is provided which may protect the calves during cold weather and result in less of a growth set back at weaning.

1. Source of fat

The digestibility of fat varies depending on the source. Butterfat is more than 95% digestible while tallow is only about 87% digestible. Most commercial milk substitutes contain animal fats such as tallow and lard. These fats contain relatively long chain fatty acids and are more digestible if some coconut oil is added as a source of short chain fats. It is important that all milk substitute powders contain some antioxidant, such as BHA, to prevent rancidity.

2. Method of dispersion

Fat may be incorporated into liquid milk substitute either by homogenization or blending with an emulsifying agent, such as lecithin. When fat is homogenized, the fat globules average about 3 to 4 microns in diameter. The globules in nonhomogenized products usually average 10 to 20 microns. Calves less than 2 to 3 weeks of age digest fat from the smaller globules more efficiently than from the larger ones. The size of the fat globules can be easily measured by examining an unstained drop of milk under a cover slip with the high power objective of a clinical microscope. If you have an ocular micrometer, the size of the droplets can be measured directly; if not, the size can be roughly estimated as being larger or smaller than a canine erythrocyte, which is 5 to 7 microns in diameter. Hair loss, particularly over the areas of the body which come in contact with feces, may be associated with feeding fats of poor digestibility.

c. Fiber - Many of the factors that we would like to know about a milk replacer cannot be found on the feed tag. However, the concentration of fiber can supply us with some indirect information. If the fiber content is above 0.5% then we may assume that either the product contains more plant protein than should be incorporated into milk substitutes for calves less than three weeks old, or the skim milk powder has been heated excessively. In either case the product is undesirable for young calves. Young calves can be raised on such products, but at only at low levels of intake. Digestive problems are likely to occur if amounts large enough to support growth or maintenance during cold weather

are fed. Calves older than three weeks may be feed liquid diets from powders containing more than 0.5% fiber, but their performance will probably be less than if they were fed from powders containing a lower concentration of fiber.

III. Feeding calves with diarrhea.

A. Disadvantages of feeding the calf with enteric disease

1) osmotic catharsis - fluid and electrolyte depletion

When malabsorptive diarrheas occur, carbohydrate and protein that would normally be absorbed remains in the intestine. Fermentation of these substrates by bacteria creates a high osmotic pressure within the lumen of the intestine and water is drawn from the blood into the gut. The flow of water into the intestine interferes with electrolyte absorption. Thus, it may be detrimental to feed scouring calves during the period of rehydration and electrolyte repletion. However, experiments here at MSU suggest that calves with malabsorptive diarrhea seldom become severely dehydrated; the total fecal fluid output seldom exceeds the fluid intake. Therefore the concern over aggravating dehydration by feeding may not be of prime importance.

2) lactic acid production

Calves with diarrhea and dehydration frequently become acidotic and lactic acid is the major acid accounting for this. There are two potential sources of lactic acid. The first is from anaerobic metabolism in tissues with poor blood profusion. This is the explanation given for the lactic acidosis observed in animals in shock. The other source is from bacterial fermentation of nonabsorbed carbohydrate and protein. Feeding calves with diarrhea may then increase the lactic acid load by this second mechanism. However, in calves with good hydration and tissue profusion, lactic acid can be rapidly converted to glucose by the liver.

3) small intestinal bacterial overgrowth

Normally the numbers of coliform bacteria in the small intestine are 100 to 1000 fold less than in the colon. During diarrhea, regardless of etiology, it appears that the population of all types of colon bacteria, both potential pathogens and normal organisms, proliferates in the small intestine. These organisms may be benign when located in the colon, but they may aggravate diarrhea when present in large numbers in the small intestine. It has been suggested that with holding milk from calves with diarrhea reduces the substrate for bacterial growth with in the intestine. This may be the case in some instances of calf diarrhea, but some

types of food are bacteriostatic and may have a beneficial, suppressive effect on the bacterial population of the small intestine. Free fatty acids liberated by the digestion of dietary fat appear to suppress bacterial growth in the small intestine; calves feed whole milk have markedly lower intestinal bacteria counts that those feed skim milk. Thus, feeding diets with appropriate quantities of digestible fats, may help to control diarrhea due to small intestinal bacterial overgrowth.

B. Advantages of feeding the calf with enteric disease

1. Increased nutrient intake

It is well known that calves with diarrhea lose large amounts of water and electrolytes and that in many cases this is the ultimate cause of death. The role of nutrient loss, other than water and electrolytes, is not as well known, or understood. It is known that calves with diarrhea, even when given normal diets, develop negative energy, nitrogen, calcium, phosphorus and magnesium balances. Thus, calves with enteric disease loose weight and are under tremendous nutritional stress. Limiting their feed intake could only increase the degree of nutrient deficiency.

I know of no data examining the effects of feeding versus starvation on weight changes of calves with diarrhea, but there is some available from children. In one experiment, infants with diarrhea were divided into two groups and one group fed and the other group starved for the first 24 hours following the onset of diarrhea. At the end of the first day the fed group had lost more weight than the starved group, suggesting that feeding indeed aggravated fluid loss. By the second day, however, the fed group had regained all the weight they had lost while the starved group did not regain their original weights until the third day. Weight gains then continued to increase at a more rapid rate in the fed children than in the starved ones. These results suggest feeding during diarrhea may reduce the nutritional stress placed on the individual.

2) Inductive effect of feeding

The concept of "resting the gut" for treatment of enteric disease was once popular in both veterinary and human medicine. The theory was that by with holding food from individuals with enteric disease, the enteric epithelium was given a chance to recover from its insult and regenerate. In fact, however, disuse atrophy is the more likely outcome; if experimental animals are given total intravenous nutrition, the villi of the intestine shrink. Moreover, the intestinal epithelium hypertrophies in response to increased feed intake. This is called the inductive effect of feeding. Animals in cold climates, pregnant and lactating animals, and others with increased

feed intake have elongated intestinal villi. So it appears that there is some effect of feeding that stimulates intestinal epithelial growth.

luminal nutrition

One theory used to explain the inductive effect of feeding is luminal nutrition. This theory holds that the presence of nutrients in the intestinal lumen directly stimulates the proliferation of enterocytes. The idea being that the enterocytes utilize the nutrient substrate for growth.

endocrine effects

Gastrointestinal hormones probably play a major role in the inductive effect of feeding. It is well established that gastrin, a hormone who's release is stimulated by the presence of food in the stomach, is necessary for for the maintenance of gastric epithelium. A hormone that may play a role in the maintenance of the small intestinal epithelium is enteroglucagon. (Sometimes called gut glucagon or GLI.) This hormone is secreted, primarily by cells in the ileum; secretion is stimulated by the presence of glucose. Since glucose is normally absorbed almost completely by the upper small intestine, only small concentrations of it usually reach the ileum. Thus, under normal circumstances, there is a low level of enteroglucagon secretion. When the amount of food reaching the upper intestine is large enough, it overcomes that portion of the intestine's ability to absorb glucose and the amount of glucose reaching the ileum is increased. In response to this, enteroglucogon release is stimulated, the small intestinal epithelium proliferates and more glucose is absorbed in the upper gut. Subsequent to the increased glucose absorption in the upper gut, the glucose concentration in the ileum decreases and the secretion of enteroglucagon abates.

The potential significalce of this to diarrhea therapy lies in the role enteroglucogon may play in stimulating regeneration of damaged intestinal epithelium. If villous atrophy has occured, carbohydrate malabsorption will occur, glucose will reach the ileum and enteroglucagon secretion will be stimulated. The enteroglucagon will then stimulate the proliferation of enterocytes and cause the villi to elongate. Assuming this mechanism occurs in calves, feeding milk or oral glucose solutions during diarrhea could hasen the regeneration of intestinal epithelium.

THE ROLE OF BOVINE RESPIRATORY SYNCYTIAL VIRUS (BRSV) IN ENZOOTIC PNEUMONIA OF DAIRY CALVES

John C. Baker, DVM, MS

Bovine respiratory syncytial virus (BRSV) was first isolated in Switzerland in 1970 and in 1974 was first reported from the United States. Recently this virus has been recognized to have a more important role in the bovine respiratory disease complex than previously thought. Reports from Nebraska have revealed BRSV to be the cause of a unique and important respiratory disease in feedlot cattle. The vast majority of research on BRSV has been in beef cattle and there is a lack of knowledge on the importance of this agent in respiratory disease of dairy cattle in the United States. Studies were undertaken to determine if BRSV had an etiologic role in enzootic pneumonia of dairy calves.

Bovine respiratory syncytial virus is an RNA virus and is grouped along with PI_3 virus in the paramyxoviruses. Unlike PI_3, BRSV does not hemaglutinate red blood cells. The BRSV should not be confused with the bovine syncytial virus which is a retrovirus.

Clinical signs associated with BRSV disease outbreaks in feedlots include anorexia, depression, nasal and lacrimal discharge, increased respiratory rate and elevated body temperature (104-108°F). In later stages of the disease dyspnea becomes pronounced with mouth breathing and in some cases the formation of subcutaneous edema around the throat and neck. Two phases of BRSV disease have been reported from some outbreaks. The first phase is milder and is characterized as having signs typical of shipping fever. The second phase is more severe and characterized by severe dyspnea. It is believed by some that the second phase of the disease may represent a hypersensitivity reaction to the virus, but this remains unproven. The 2

phases of BRSV disease can blend together or can be separate and distinct. In some outbreaks it is reported that animals recover from the first phase of disease only to relapse after a period of time into the more severe second phase of the disease.

The pathologic findings associated with BRSV are distinctive. While there is almost always a secondary bacterial bronchopneumonia present the striking feature are lesions that are similar to those found in adult cattle with atypical interstitial pneumonia. Gross pathologic findings include a severe diffuse interstitial pneumonia which is characterized by the presence of interstitial edema and emphysema. Histopathologic lesions typically include syncytial cell formation in bronchiolar epithelium and lung parenchyma which often contain intracytoplasmic eosinophilic staining inclusion bodies, thickening of alveolar septal walls, edema fluid in alveolar spaces and hyaline membrane formation. Some areas of lung may show proliferation of bronchiolar epithelium while in other areas a necrotizing bronchiolitis can be present.

Studies were undertaken during the fall of 1983 and winter of 1984 to investigate if BRSV had an etiologic role in naturally occurring outbreaks of enzootic pneumonia of dairy calves. In addition, attempts were made to determine the importance of other viruses along with bacteria, mycoplasmas, ureaplasmas and chlamydial agents in these outbreaks.

In this study, BRSV was the virus most often associated with outbreaks of enzootic calf pneumonia and was demonstrated in 10 of 14 outbreaks investigated. Other viruses, which included BVD, IBR, PI_3 and adenovirus type 3 were much less commonly associated with these outbreaks. Pasteurella multocida was the bacteria most often isolated from both nasal swabs and lung cultures. The next most common bacteria isolated was Pasteurella hemolytica. Haemophilus somnus was only isolated from the nasal swab collected from one

calf from one outbreak. Mycoplasmas and ureaplasmas were frequently isolated from nasal swabs from all of the outbreaks, but the significance of this finding is questionable. Mycoplasmas were occasionally cultured from lung samples. Chlamydial agents were not demonstrated from any of the outbreaks.

Clinical signs observed in outbreaks of calf pneumonia in which BRSV was demonstrated were not specific to make a diagnosis of BRSV infection. One clinical impression noted from several of the outbreaks was the rapid progression of respiratory disease such that some calves although in the acute phase of disease showed signs of severe respiratory distress more typical of a chronic pneumonia. Two distinct stages of respiratory disease as described from BRSV disease outbreaks in feedlots were not recognized in these outbreaks of calf pneumonia.

DIAGNOSIS OF BRSV

ANTEMORTEM DIAGNOSIS

Virus Isolation

Virus isolation represents the poorest method available to establish a diagnosis of BRSV. The virus is labile and does not survive well using standard techniques of collection and transport to the laboratory. Also it is thought that antibodies against BRSV appear rapidly in the respiratory tract which neutralize the virus thus increasing the difficulty in isolation. Even if the virus is isolated it can take multiple subpassages in cell culture and serologic diagnosis can in some cases be more rapid. Increased success in isolation of BRSV has been reported by inoculating cell cultures in the field immediately after the collection of samples, but this procedure would be impractical in routine veterinary practice.

Direct Fluorescent Antibody (DFA) Exam of Nasal Smears

The technique DFA examination of nasal smears is useful in diagnosis of BRSV and will give better and faster results than virus isolation. Nasal swabs should be collected as far caudally as possible in nasal cavity and then smeared onto glass slides, fixed in acetone and then sent to the Veterinary Diagnostic Laboratory for DFA examination.

Serology for BRSV

Several serologic testing procedures have been developed for BRSV. At present, the microtiter serum neutralization test is available through the Minnesota Veterinary Diagnostic Laboratory. This test has proven useful and reliable for establishing a diagnosis of BRSV. Acute serum samples should be collected as early as possible after respiratory disease is recognized in that antibody titers to BRSV can rise rapidly after the onset of disease. Antibody titers to BRSV begin to decline shortly after peaking and convalescent serum samples should be collected at about 2 to 3 weeks. Acute and convalescent samples should be collected on multiple animals in order to establish a serologic diagnosis.

One problem with serologic diagnosis is that passive immunity in young calves to BRSV, although not protective from disease, prevents a humoral antibody response to BRSV infection. This can be overcome by sampling different age groups such that some calves are included which are likely to have lost their passive immunity to BRSV.

The microtiter serum neutralization test for BRSV tends to be an insensitive test. This means that some animals with low levels of BRSV antibodies can be falsely classified as negative by this test.

Postmortem Diagnosis

Calves dying during outbreaks of enzootic calf pneumonia represent an excellent source of samples for rapid diagnosis of BRSV infection.

Gross examination of the respiratory tract can be helpful in establishing a diagnosis. The prominent lesion found is an interstitial pneumonia with interstitial emphysema and edema. A frequent finding is failure of the lungs to collapse upon opening of the thoracic cavity. A secondary bacterial bronchopneumonia is a common feature in the cranial lung lobes.

Virus isolation can be attempted on lung tissue but as with nasal swabs this technique has been poor in establishing a diagnosis of BRSV.

Direct fluorescent antibody examination of frozen lung samples for BRSV has been an excellent and rapid technique for diagnosis of BRSV. Fresh lung should be submitted to the Diagnostic Laboratory. Samples should be collected from areas bordering the obvious bronchopneumonic lung as well as samples from the caudal lung lobes.

Histopathologic examination of lung has proven useful in diagnosis of BRSV. Lung samples should be collected and placed in formalin and then submitted to the Diagnostic Laboratory. It is important that samples be collected from not only the areas of bronchopneumonia but also from the caudal lung lobes. Often the typical lesions seen in association with BRSV are masked in the area of lung where secondary bacterial bronchopneumonia is present and for this reason samples from the caudal lung lobes should also be submitted for examination.

TREATMENT

Antibiotic therapy is indicated because of the common occurrence of secondary bacterial pneumonia. Sound judgement should be used in antibiotic therapy such that an appropriate antibiotic is selected and is used at proper dose and frequency. Antibiotic therapy should be started as soon as respiratory disease is recognized and should be continued several days after apparent recovery.

Good results have been reported from Nebraska with the use of corticosteroids and antihistamines in the treatment of BRSV disease. Because of the positive response to these drugs it has been suggested that the severe stages of this disease may be a hypersensitivity reaction to BRSV infected cells. This form of treatment can be considered in severely affected dairy calves but may not be indicated for all calves in a BRSV outbreak of calf pneumonia.

PREVENTION

Norden Laboratories have plans to market a vaccine for BRSV probably sometime this summer or fall. This is a modified live vaccine which is administered by the IM route. Booster vaccination is recommended at 4 weeks. Presently, field trials of this vaccine, both in dairy and beef cattle, are being conducted as part of our ongoing research on BRSV at the College of Veterinary Medicine.

Common Lamenesses in Dairy Cattle

L. C. Allenstein, D.V.M.
Whitewater, Wisconsin

Disturbances in locomotion of the dairy cow are a common source of requests for help by the dairyman in a bovine practice. Problems of the foot and leg are intriguing and we as veterinarians have to be aware of their importance. Feet and leg problems are revealing as they often indicate the state of nutrition, state of sanitation, the state of management, and somewhat the breeding status of the herd. A differential diagnosis is the main reason why veterinarians are called to determine whether age ,injury, an infectious process or a metabolic disorder is involved. Is it acquired or due to a hereditary factor? Diagnosis is first and then therapy and prevention must follow.

Not one veterinarian today enjoys looking at feet as a life's occupation. To me, I also can become irritable when 7 of my 10 scheduled calls deal with lamenesses. And then when I'm looking at that sick cow, the client will remark, "Oh yes, Doc, could you look at those 2 cows that are lame?" I'm sure you have all experienced that situation.

Yet, nothing is more gratifying than to pick up the foot of a cow that the dairyman has been injecting with penicillin for 1 week with no response and you find a nail in the sole. Or have you been caught guilty of a similar missed diagnosis just because you did not pick up the foot and examine it thoroughly. An accurate diagnosis is only established by this procedure as well as watching her in motion and in a standing position. Trimming the foot at this time is a necessity as well as using a hoof tester to find the problem.

Many ways have been devised to lift a foot. Many instruments are used to do the trimming and the examination.

Remember, 85 to 90% of the lamenesses are due to a problem in the foot. Also 90% are in the rear feet. In the rear foot, 80% of the trouble is in the lateral claw while in the front foot, 80% of the trouble is in the medial claw.

HOOF TRIMMING

Trimming of feet is an art that is fast disappearing. And yet it is an important factor in the prevention of lamenesses and should be done in problem cows at 6 month or yearly intervals. Confinement, housing on hard surfaces, large herds, crowding, and acidotic rations have made foot trimming a necessity.

Trimming may be done in many ways: standing on a plywood board with a mallet and chisel; putting the foot flexed on a wooden box; pulling the foot up to a beam; or casting a cow on a table or on the floor.

Many instruments have been used: hoof knives, chisels, clippers, sanders, files, etc.

In trimming the foot, one must determine the length desired as well as where the sensitive lamina begins. Both toes should be approximately the same length unless a condition exists that requires more trimming from one sole or toe. Leaving one toe or sole without trimming will allow the weight to be carried on the good toe.

The outside wall generally should be a little higher or longer than the inside wall. The sole should be slightly concave so weight bears in on the wall. The consistency of the sole often governs the amount to remove. Care should be taken not to expose sensitive tissue. Generally one will find the outside toe of the rear foot and the inside toe of the front foot slightly longer. This is normal, a result of more weight bearing. However most lamenesses are found in these claws.

Care should be taken to correct leg posture by foot trimming. Cow-hocked legs can be helped. Sickle hocks can be straightened. Cows with legs that are too straight an be corrected with proper hoof trimming.

Foot trimming is an art and would require a paper 2 hours or more in length to fully describe all aspects of it. It is just as important in the cow as in the horse in the prevention of feet and leg problems.

THE INCIDENCE OF LAMENESS IN DAIRY COWS

How often do we see lame cows? The incidence of foot and leg problems is difficult to evaluate, but they are increasing.

I'm sure Dr. Greenough will relate to his survey in Europe of a 5.5% incidence. Dr. Harold Amstutz reported a range of 4 to 14% per year in certain herds. My experience is higher, possibly 5 to 20% in herds. It ranks high in the frequency of calls to a dairy farm being surpassd only by mastitis, infertility and metabolic diseases.

Few dairymen realize the production loss experienced from a lame cow. Economics must be considered.

1. Loss of production
2. Weight loss
3. Breeding delayed - frequently because of anestrous
4. Culling because of lameness
   - Ranks fourth behind mastitis, infertility, and low production.

WHY ARE WE SEEING MORE LAME COWS?

1. High production
   Stress of pushing with high energy rations

2. Acidotic rations

The advent of acidotic rations has created more foot problems than any other factor. This has evolved when a high concentrate to roughage ratio is being used. Much more fermented feed with less long stem dry hay is being consumed.

3. Confinement

Most herds are confined to yards or barns with hard surfaces, usually cement - rough cement. I like grooved cement and smooth. More time should be spent on dirt with more exercise. In our practice we had only 3 herds on pasture this past summer.

4. Husbandry

There is more concentration of manure, filth, and moisture in our housing areas. Humidity in our barns may predispose the animals to infectious agents.

5. Large herds

With this factor there is an increasing lack of attention to preventative foot care. Frequently facilities lack stall space where animals can rest. This results in more time standing and thus greater stress on feet and legs.

6. Heredity

One must consider not only P-D in sire selection, but also the posture and structures of the feet and leg.

7. Infectious causes

There is a huge list of bacteria and viruses responsible for foot infections.

One could classify the above causes as either intrinsic or extrinsic factors responsible for lameness in our dairy cattle.

INTRINSIC FACTORS

Intrinsic has to be heredity. I feel this is definitely a factor and of considerable importance. Dr. Paul Miller with American Breeders Service has researched the literature on the inherit ability of the foot and leg. His findings state that the foot structure is only 11% inheritable, and the leg only 15%. I can't quite agree; I feel it is higher. However, Dr. John White and Dr. W. E. Vinson of Virginia Polytechnic Institute, have reported that many traits when added together increase the percentage of inheritability, especially bad traits such as straight legs=34%, crampiness=43%, etc. Combined traits can be selected for a positive improvement overall in feet and legs, stature and udder attachment. This work was reported by Dr. John White and Dr. W. E. Vinson, Virginia Polytechnic Institute. Further work has shown that foot conformation was very important.

Conformation

Crampiness is a problem in our cattle today even though we are constantly trying to eliminate this trait through selective breeding. Proper trimming of the feet, comfort stalls, box stalls, proper temperature and antispasmotic drugs such as tranquilizers, dipyrone, and banamine will all alleviate the symptoms but offer no permanent cure.

Legs

Legs that are too straight result in injuries to the stifle and the hock. Stifle injuries are numerous. To the adult cow it is very serious. Diagnosis is very important. Try to determine which ligaments are involved.

Medial Collateral Ligament

The medial collateral ligament is often stretched or torn from its insertion. An accurate diagnosis can be made by sedating the cow or placing her under anesthesia, rotating the hock laterally, and placing the fore finger at the joint, enabling one to feel the finger fall into the joint if there is a defect.

Surgery has been performed on the medial collateral ligament by stabilization of the joint with Lembert sutures. Dr. D. A. Nelson, University of Illinois, has described the surgery placing the sutures perpendicular to the joint plane.

Anterior Cruciate Ligament

More common in the cow is the rupture of the anterior cruciate ligament. It was first described by Dr. Greenough, Dirksen, Weaver and others. When this occurs, the tibia is displaced forward in relation to the femoral condyles showing a prominence of the tibial crest. Distention of the joint capsule is evident. There is a definite drawer action of at least 2 to 3 cm. with an audible dislocation when posterior pressure is exerted on the tibia.

Surgery has again been described by imbrication of both the medial and lateral retinacular tissue with Lembert sutures. Also an imbrication suture was placed from the femoral tibial ligament to the lateral or middle patellar ligament.

Our clinic has not attemtped anterior cruciate ligament repair in adult cows. However, we have had a 40% success rate in the 5 surgeries we have performed on heifers. Conservative treatment of stifle injuries such as rest and the use of anti-prostaglandin drugs has resulted in few cures. I feel surgery should be attempted early before degenerative joint changes take place if the animal is of sufficient value to warrant this expense.

Distention of the Tibial-Tarsal Joint Capsule

Distention of the tibial-tarsal joint capsule (the hock) is very common due to over extension predisposed by a straight leg. Etiology of this condition is usually trauma with the joint often filling with blood. Infectious agents also are involved such as Haemophilus somnus, Mycoplasma, and Chlamydia. If there is a sudden onset, cold applications are indicated for 48 hours followed by heat. If distention persists, drainage is indicated followed by an injection of 20 to 40 mg. of Depo Medrol with penicillin. During acute stages, Tylosin Hydrochloride or Lincocin-Spectinomycin may be given intramuscularly.

Bandage or wrapping of a distended hock after drainage and injection is of great value in restoring proper function. A figure 8 bandage of elastic tape will often stay in place for several days. Domoso under this bandage is of value.

Hygroma of the Hock Joint

Hygroma of the hock joint area is very common. Trauma is the usual cause. Therapy consists of the same as above. Often surgery is required to remove a fibro-cartilaginous mass just under the skin. Care must be taken to control hemorrhage.

Most hock injuries can be prevented by using an adequate amount of bedding and rubber mats.

EXTRINSIC FACTORS

Extrinsic factors which create problems with feet and legs include nutrition, infectios agents, environment, and trauma. Most of these interrelate to bring on problems.

Nutrition

Nutrition is the number one cause of foot problems creating or contributing to many lesions in the feet and legs. I also feel that laminitis is the foremost factor in nutrition which leads to these problems.

The subject of laminitis will be discussed in a paper that I will be presenting in the afternoon session of this seminar.

Other nutritional aspects of lamenesses involve deficiencies of esential elements including:

Vitamin D, Calcium and Phosphorus Deficiencies

Rickets may develop with inadequate levels of vitamin D, calcium, and phosphorous or an improper balance of these elements in the ration. This is not common except in calves and heifers raised in the absence of sunlight. More common is a condition called epiphysitis, where fast growing heifers actually outgrow their bone formation. This is created by high energy and protein rations with an improper balance or deficiency of calcium and phosphorous. Too much vitamin D can create a similar condition.

Selenium/Vitamin E Deficiencies

Nutritional muscular dystrophy is frequently seen when selenium or vitamin E is deficient. Serum levels are now easily attained for selenium. When serum levels go below .08 ppm, additional selenium must be added to the ration. Although .1 ppm of selenium may be added to the ration legally, certain areas may require more.

Other Nutritional Deficiencies

Other deficiencies may include copper, zinc, sulfur, and magnesium. Each has a specific place and may be involved as a secondary factor in certain foot conditions.

INFECTIOUS CAUSES

Foot Rot

Foot rot is a much over-played diagnosis among dairymen. I hope not also among veterinarians. This is not to imply that foot rot (necrophorous infection) does not exist, but rather to suggest that too frequently we too casually diagnose lameness as foot rot. It is seen as a primary infection often in the spring as an interdigital phlegmon and as a secondary infection with interdigital dermatitis, ulcers, white line separations, puncture wounds and trauma. Systemic penicillin or sulfa is excellent in the treatment of foot rot. Many times an interdigital injection of penicillin with a steroid is of value.

Most of our foot rots are bandaged with sulfa creme or a sulfa powder with copper sulfate. Prevention must include a foot bath of 2% formalin or 2 to 5% copper sulfate, or a dry powder bath or 1 pound copper sulfate to 25 pounds lime. Organic iodides are discouraged in dairy cattle.

Interdigital Dermatitis

Moist interdigital dermatitis is an infection caused by Bacteroides Nodosum. Secondary factors of stress (calving), vitamin A deficiency, zinc deficiency, copper deficiency, lack of protein and other nutritional elements definitely predisposes this condition. This develops into cracked heels and is a common condition seen during winter and early spring. Trimming out the cracks and shortening the toe often alleviates the condition. If there are deep cracks an astringent dressing of phenol-formalin, kopertox, copper sulfate plus sulfa powder with a bandage may be necessary. A foot bath is of value in the prevention of interdigital dermatitis, however, if there are nutritional deficiencies these must also be corrected.

Other infectious agents such as mycoplasma, haemophilus, and chlamydia should also be considered in lameness of the dairy cow.

ENVIRONMENT

Confinement, housing, cement, overcrowding are all factors that contribute to feet and leg problems of dairy cattle. Cement floors and confinement definitely predispose to laminitis. I once preferred cement floors but my preference has changed. I now want a dirt yard or pasture. However, only 3 herds in our practice were on pasture last year. The good Lord made the cow to be on pasture. The excessive wearing of the sole causes laminitis and then ulcers. But more important yet is the overgrowth it creates. This is the reason for the rubber mats in the stalls.

Sanitation--Yards and Facilities

Filth predisposes foot rot. I also see a mycobacterium infection of the lymph channels causing a granuloma.

TRAUMATIC INJURIES

Trauma creates many leg problems, especially at calving. Calving paralysis with involvement of the obturator nerve and more important a branch of the sciatic is often seen.

Sciatic or Nerve Paralysis

Sciatic or nerve paralysis is frequently seen after the cow has been in a recumbent position for sometime. The nerve usually is injured where it crosses the trochanter major or near the tendon of achilles. This can be confused with partial rupture of the gastrocnemius muscle. Sciatic or nerve injury results in paralysis of the extensor muscles of the hock and the flexor muscles of the digit.

Peroneal Nerve Paralysis

Peroneal nerve paralysis is also common. It is injured where it crosses the fibula head right next to the lateral condyle of the femur. Here there is a paralysis of the flexors of the hock and the extensors of the digit, with a knuckling of the fetlock. The use of a cast may be necessary in these conditions.

Radial Nerve Paralysis

Radial nerve paralysis of the forelimb is seen infrequently.

OTHER CAUSES OF LAMENESS

I would like to mention a few conditions seen frequently in our practice.

Screw Toes

Screw toes are seen when the lateral toe of the hind foot and medial toe of the front foot curls around and the wall becomes the bearing surface. Heredity may be involved but I feel laminitis with a wall and sole is seen following a severe laminitis or infectious disease. Here again trimming and bandaging may be required.

Sand Cracks

Sand cracks with a vertical fissure into the coronary band can be extremely painful. Dry weather and sandy soils predisposes to their occurrence. Moist bandages of an oil with an antiseptic is often required.

Interdigital Fibromas

Interdigital fibromas are common. If lameness results, then deep surgical removal into the interdigital cushion is required. A wire between the toes with a supporting bandage is preferred following removal.

Spastic paresis

Spastic paresis is not common but must be recognized. It is characterized by spastic contracture of the gastrocnemius muscle and other associated muscles causing severe extension of the hock and stifle joints. It is usually unilateral. This extreme extension causes the limb to swing as a pendulum. A complete tibial nerve neurectomy will correct the condition to market the animal. Never use these animals for breeding purposes because of hereditary tendencies.

Intervertebral Disc Involvement

Intervertebral disc involvements with prolapse and calcification of the disc are seen. Degrees of paralysis are the symptoms exhibited.

INFECTIONS/INJURY TO DIGITS

Amputation of the digit is not as common as it once was in our practice. Toe boards and a plaster of paris supporting cast have many times alleviated the use of amputation in septic joint involvement. I use the open method (no skin flap) employing a wire O-B saw to remove the toe. It is done standing with the leg restrained. Anesthesia is 10 to 20 cc of lidocaine in the digital vein after a tourniquet is applied just below the knee or hock.

This is a brief summary of common foot and leg problems in the bovine. It is not complete but represents conditions I see in a Wisconsin practice.

REFERENCES

1. Troutt, H. F. Pathology Associated with Rations. Proceedings - AABP, 1973, 68-73.

2. Weaver, A. D. The Prevention of Laminitis in Dairy Cattle. Bovine Practitioner, AABP, Nov. 1979, 70-72.

3. Greenough, P. R. The Practical Overview of the Bovine Foot. Seminar, University of Minnesota, Nov. 1977.

4. Amstutz, H. E., et al. Lameness in Cattle - Survey. Bovine Practitioner. Nov. 1975, 39-49.

5. White, John M. and Vinson, W. E. Type in the Holstein Breeding Program. 91st Holstein Convention, Philadelphia, 1976.

6. Weaver, A. D. Disease of the Bovine Stifle Joint. Bovine Practitioner, Nov. 1972, 41-45.

7. Greenough, P. R.; MacCallum, F. J.; Weaver, A. D. Book, Lameness in Cattle. Lippincott, 1972.

THE CONFORMATION OF CATTLE

Introduction

Veterinarians have had little access to scientific information concerning the conformation of cattle. Interest in the subject has mostly been left to the breeder and the show judge whose criteria for evaluating conformation are different from those of a veterinarian whose prime concern would be those aspects of conformation that relate to the prevention of disease or a better understanding of the etiology of disease. An increased awareness of the importance of preventive medicine has led the veterinarian to seek to widen the scope of his knowledge. The purpose of this paper, therefore, is to review informed opinion and such scientific data as does exist in order that the reader may be better able to consider relationships that may exist between faulty conformation and the incidence of related diseases.

By definition, conformation is the proportionate shape or contour of an animal. Berg and Butterfield[3] tend to play down the effects of shape as a commercially important entity and believe that selection should be based on productive criteria likely to lead to the development of functionally efficient animals. They quote Harrington: "...." that good shape per se was largely achieved by excess fat deposition, which overshadowed small advantages in meat to bone ratio," and they point out that the influence of the show ring, along with unrealistic feeding regimens, has done a great deal of harm in recent years in increasing the incidence of arthritis and dystocia among British breeds of beef cattle.

The judgment of conformation still is a subjective matter based on deep-rooted aesthetic principles. Accurate and practical methods of measuring the conformation of living animals have not been developed. Pragmatic data or standards are not available and, therefore, the livestock industry has no alternative for the traditional systems.

Logically, animal selection should be based on performance and functional efficiency.

Functional efficiency includes reproductive efficiency and longevity. Reproductive efficiency involves potential problems at parturition which can be related to the conformation of the pelvis and indirectly with the joint angulations of the upper limb. Bonsma[4] believes that hormonal disturbances associated with subfertility in both bulls and cows have a marked effect on conformation.

Longevity is a characteristic which may be low on the list of priorities, but some breed societies take total lifetime production as a measure of excellence. An animal's ability to survive the increasing stresses of intensive farming is no mean accomplishment. Three musculoskeletal diseases - arthritis, chronic laminitis and hoof deformation - are insidious in onset and their importance as indirect causes of culling is underestimated. They are either precipitated by poor conformation of are manifested by changes in conformation.

Conformation is hereditary. If animals with certain types of conformation can be proved to have a higher incidence of certain diseases, hereditary predisposition to those diseases can be presumed. Some conformational, particularly hoof, characteristics appear as the animal matures as a result of mechancial stress, an example being dairy cows which are forced to abduct their hind limbs during progression in order to circumvent the udder. Whether the resultant stresses affect the lateral hind digit particularly is a matter for speculation, but the higher incidence of lateral compared with medial hoof disease or abnormality is a fact. This phenomenon has never been satisfactorily explained, but Toussaint-Raven[22] was able to measure the weight borne by the medial and lateral hind hooves respectively and found a considerably greater weight was often placed on the lateral hooves.

Endocrine Influence on Musculoskeletal Growth

Male sex hormones have a direct influence on muscle growth[4] and account for the greater muscle mass in the bull relative to steers or females. The ossificaton of the growth plate is dependent upon the secretion of estrogen in the case of the bull. This ossification is delayed in subfertile animals[4] which therefore tend to become taller (Fig. 1). Bonsma's statements regarding sexuality and conformation provide a convincing argument, on the basis of functional efficiency, to condemn show ring standards. It is his considered opinion that the beef breed standards have been based on the conformation of a 2-year-old ox for both male and female. This disenchantment with the whims of the show ring are echoed by Butterfield and Berg[3]. In judging conformation on veterinary grounds, cattle should be assessed on functional efficiency with respect to factors leading to a potential loss of productivity.

The Coxofemoral Articulation

The coxofemoral and femorotibial articulations are those most frequently affected by degenerative arthritis, predisposition to which is hereditary. Undue stress on the joint will accelerate the progress of this disease. The

efficiency of the distal limb to absorb concussion plays an important part in increasing or decreasing the shock transmitted through joint surfaces of the hip. The more closely the femur approximates to the vertical, the greater will be the tendency for the angle of a line drawn between tubera coxae and ischii with the horizontal to be reduced (Fig. 2). Such an angulation would probably increase trauma to the coxofemoral articulation.

Another consideration is the predisposition to dystocia that exists in the animal with the flattened rump. The shallower the coxoischiactic line, the smaller will be the pelvic passage, a characteristic associated with subfertility. One breed society requires that the great trochanters shall be high and wide apart. The width between the great trochanters is determined to a large extent by the position of the acetabula and the length of the femoral neck. To invite great width between the great trochanters without reference to the distance between the two tubera coxae would seem to predispose to mechanical stress. Hindson[12] found a correlation between the distance between the two tubera ischii and the width of the pelvic inlet (Figs. 3 & 4).

In practical terms, a veterinarian might be unwise to endorse the selection of animals showing evidence of flatness of the pelvis and could draw the attention of breeders to hereditary, it would be wise to consider the advisability of selecting breeding stock with this in mind.

Tibiotarsal-Metatarsal Articulation

A hock angle of 170 degrees should be classified as straight and bulls with hocks at such an angle are at a severe mechanical disadvantage during service[21]. Others refer to angulation of 155 degrees + 3 as being steep and from a series of 76 such animals, twelve showed clinical lameness. These workers were also able to detect a genetic pattern. At the other extreme, Pusch states[21] that a hock angle of 130 degrees should be classified as a sickle-hocked while Duerst[6] would regard this as normal. If the angle were less that 130 degrees, an awkward gait might be expected with an abnormally high stress on the tuber calcanei. In Nesbitt's study[18], new bone formation was discovered in the plantar ligament of the tarsus although no observation was made regarding conformation. No specific evidence exists to link "sickle hock" with disease although breed societies find this type of conformation aesthetically objectionable.

The position of the tuber calcanei relative to the body is variable; however, this is a matter of posture rather than of conformation. The two concepts are undoubtedly closely linked with Duerst[6] shows that joint angulation can be changed in response to environmental need. Posture may change as the center of gravity changes as in pregnancy. Coxitis or gonitis, elongated hooves or a large udder have all established an abnormal posture which is reversible with proper hoof care.

The tuber calcanei should ideally be located directly beneath the tuber ischii if viewed from both the lateral and caudal perspective (Fig. 5). A more craniad position is termed "camped under" (Fig. 6) while the reverse is "camped back" (Fig. 7). A medial deviation of the tuber calcanei gives a "cow hocked" posture. Bilateral deviations from the ideal are unlikely to involve a primary hock etiology.

The angle of the metatarsus with the horizontal was determined to be 78-79 degrees in Ayrshire cattle[10]. This angulation was proved to be hereditary.

From the clinician's point of view, a straight hock should be considered an unacceptable characteristic for breeding stock. A conscious effort must be made to avoid confusing conformation with abnormal posture brought about by various factors, the most important being abnormalities of the hoof.

Carpo-Metacarpal Joint

The forelimb carries 53-55% of the body weight.[24] Direct concussion of the proximal articular surface of the metacarpus can result in primary arthritic deformities which Dammich[5] demonstrated to be due to the continued influence of stress and nutritional damage.

Metatarso (Carpo) Phalangeal Articulation

There appears to be no difinitive statement regarding the ideal angle of the pastern. In contrast to the upper limb, where mechanical stresses are absorbed by dynamic muscle masses, the fetlock absorbs these stresses through the elasticity of the suspensory system. This efficiency is demonstrated by the slow rate of degenerative change in these joints. The inclination of the pastern must take the status of the hoof bulbs and solear apex into account. The angle of the pastern also alters if the tuber calcanei lies cranially or caudally to its ideal position. The angle of the pastern can be altered by coxitis or laminitis. As a means of evaluating conformation as opposed to posture, the angulation of the pastern seems unreliable although Bonsma[4] makes the unqualified statement that weak pasterns are hereditary.

Hooves

When evaluating the conformation of the locomotory system, one must consider the hooves of cattle to be of infinitely greater importance than the rest of the proximal limb.

Conformation of the upper limb has a profound effect on the distribution of stress on the horny capsule. However, it is much more important to realize that many hoof conditions profoundly disturb the functional efficiency of the hoof and this change causes postural alterations that may be confused with defective conformation. Abnormal hoof conformation is almost invariably acquired as the result of disease to which the animal may, or may not, be genetically susceptible. Young animals should, therefore, have hooves that are structurally perfect, without concavity of the dorsal border and only slight concavity of the axial wall, with horn which is shiny and without ridges or grooves. The bulb should be rounded and not flattened. Each hoof should be approximately equal in size and have a solear surface area that is adequate for the size of the animal. This recommendation is prompted by the large number of animals passing through the Saskatoon clinic that are suffering from solear hemorrhages and that unquestionably have disproportionately small hooves for the size of the animal.

Adult cattle often acquire an abnormal hoof conformation sometimes from circumstances unrelated to the animal's genetic or conformational background

and an example would be hoof overgrowth that has resulted from lack of wear. Sometimes abnormalities occur as a result of an interplay between environment stresses and hereditary weaknesses. Chronic laminitis is fundamentally a nutritional disorder, but observation would suggest a possible hereditary factor.

The quality of the horn of the hoof is of considerable significance. Two properties of good horn are resilience and hardness. Resilience (i.e. compressive strength and elasticity) can be measured mechanically. Hardness, a quality for which there are no absolute values, is defined as "resistance of a body to penetration by another body of superior rigidity" and is measured by grinding tests.

Pigmentation has often been associated with the mechanical properties of horn. Leach[16] believes that there is little evidence that variations in resilience can be associated with pigmentation but pigmented horn may be 30% harder than unpigmented horn.[8] Increased resistance to wear is thought to be due to the presence of melanin.

Hydrated horn is considerably softer than dehydrated horn. External physical factors markedly affect the water content of horn.[3,17,20] For example, the loss of the stratum of externum of the hoof as occurs due to aging or on sandy soils will permit loss of moisture from the hoof which becomes brittle and may split.

Differences in the hardness of horn occur between breeds but varying factors such as pigmentation, age, sex, environmental conditions, body weight and nutrition would suggest that definitive statements should be treated with caution at the present time.

In the writer's opinion, it is essential that a clinician wishing to become complete in the diagnosis of lameness should study the morphology of the hoof. Hooves that are well-formed, smooth and shiny are likely to be resistant to disease. This is important when one considers the eighty percent of lameness is associated with disease of the digital region of the hind feet. Deviations from normality that occur in animals of less than 2 years of age should render them unsuitable for breeding. In older animals wear and the results of stress may change the shape of the hoof and these changes may be a good indication of the type of lesion that may be encountered. Gross changes in shape can sometimes be altered by proper hoof care while other abnormalities may be recognized that will inevitably lead to a loss of locomotory efficiency and in turn productivity. Examples of hoof conditions that cause alterations in posture are shown in Figures 8-18.

Discussion

At the present time knowledge concerning conformation of the limb and hoof is inadequate. Nevertheless we can perceive that it is an important topic. We can offer some advice regarding the breeding soundness of an animal and we can develop limited skills in relating poor conformation to functional inefficiency and disease.

References

1. Bargai U. Radiologic Diagnosis of 152 Cases of Chronic, Non-responsive Lameness in Cattle. Refuah. Vet. 32:137, 1975.

2. Bartels J.E. Femorotibial Osteoarthrosis in the Bull I. Clinical Survey and Radiologic Interpretation. J. Am. Vet. Rad. Soc. 16:151, 1975.

3. Berg R.T. and Butterfield R.M. New Concepts of Cattle Growth. Haisted Press Book, New York, John Wiley & Sons, 1976.

4. Bonsma, J.C. Factors Affecting Calf Crop. Edited by Cunha T.J., Warwick A.C. and Koger M. University of Florida Press, 1973, pp. 197-231.

5. Dammich K., Seibel S., Huth F.W. and Andreae U. Adaption Diseases of the Locomotory System of Fattening 12, 15 and 18 Months Old Fed Various Diets I: Morphology, Localizaton and Occurrence of Arthropia Deformans (G.e.). Berl. Much. Tierarztl Wschr 89:84-88, 1976.

6. Duerst J.U. Grundlagen der Tierzucht. Berlin, Springer, 1931.

7. Feher G. and Haraszti J. Sheep Posture of Hind Legs in Bulls, Its Harmful Consequences (H.e.). Magy Allatory lap 23:227, 1968.

8. Feder H. Untersuchugen zum Mineralstoffgehalt Hannover Inaug. Dissertation, 1969.

9. Fritsch R. The Etiology and Surgical Treatment of Diseases of the Hoof of Cattle. Bayer. Vet. Med. Rev. 2:96, 1966.

10. Habel R.E. On the Inheritance of Metatarsal Inclination in Ayrshire Cattle. Amer. J. Vet. Res. 9:131, 1948.

11. Harrington G. The Shape of Beef Cattle and Their Carcasses in Relation to Carcass Merit. In: Beef in the Seventies. Dublin, An Foras Taluntais, 1971.

12. Hindson J.C. Quartification of Obstetric Traction. Vet. Rec. 102:327, 1978.

13. Knezevic P and Wruhs O. Possibilities of Arthroscopy in Horses and Cattle (G.e.). Wien Tierarztl Wschr. 62:301, 1975.

14. Knezevic P and Wruhs O. Arthroscopy in the Horse, Ox, Pig and Dog. Bayer, Vet. Med. Rev. 1:53, 1977.

15. Kovacs A.B. Principles and New Equipment for Cattle Foot Conditions in Large Farms in Hungary (G.e.r.). Mh. Vet. Med. 32:463, 1977.

16. Leach D.H. Personal Communications, 1978.

17. MacLean C.W. The Long Term Effects of Laminitis in Dairy Cows. Vet. Rec. 89:34, 1971.

18. Nesbitt G.H., Amstutz H.E. and Lewis R.E. Lameness in Cattle: Survey of 102 Cases Including History, Radiographic Findings, etc. Bovine Practitioner 10:39, 1975.

19. Prentice D.E. Growth and Wear Rates of Hoof Horn in Ayrshire Cattle. Res. Vet. Sci. 14:285, 1973.

20. Pietzsch W. and Schauer W. The Water Content of Bovine Digital Horn Under Different Conditions of Husbandry and the Effect of Copper Sulfate and Formalin on the Hardness of Horn (G). Diplomarbeit, Sektion Tierproduktion und Veterinarmedizin, Humboldt Univ., Berlin, 1970.

21. Pusch G. Beurteilengslehre des Rindes. Berlin, Parey, 1896.

22. Toussaint-Raven E. Lameness in Cattle and Foot Care. Netherl. J. Vet. Sci. 5:2, 1973.

23. Van Pelt R.W. Idiopathic Tarsitis in Postparturient Dairy Cows: Clinico-pathologic Findings and Treatment. JAVMA 162:284, 1973.

24. Weaver A.D. Genetical, Physiological and Anatomical Factors Contributing to Foot and Limb Disorders of Dairy Cattle. Presented to Meeting of Concrete and Cement Association, 1978.

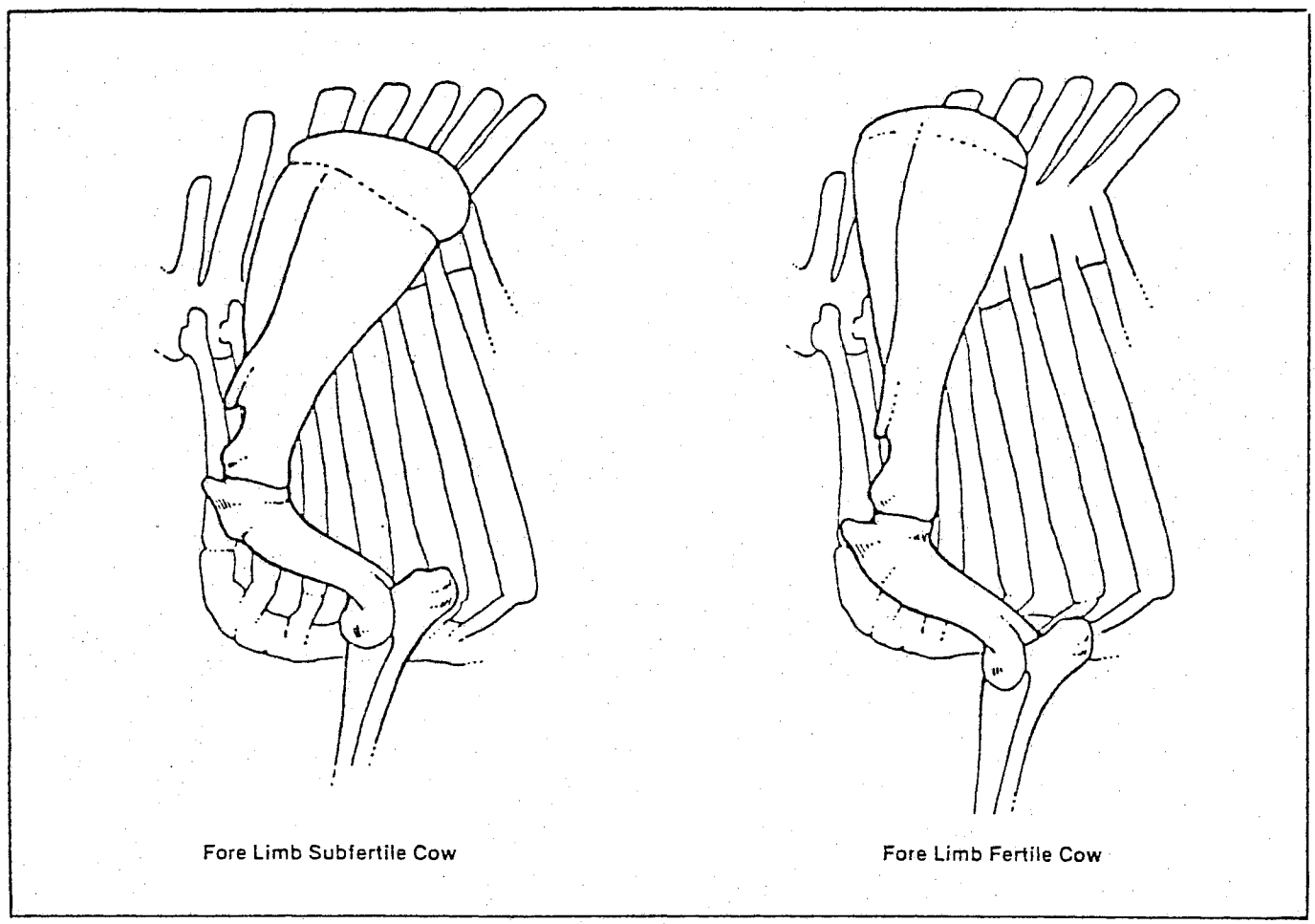


Fig. 1 — In the presence of reduced estrogen levels, growth plates fail to ossify. The dorsal spines of the thoracic vertebra continue to elongate, the sternum becomes more pronounced as does the scapular cartilage. (Redrawn from Bonsma 1973)

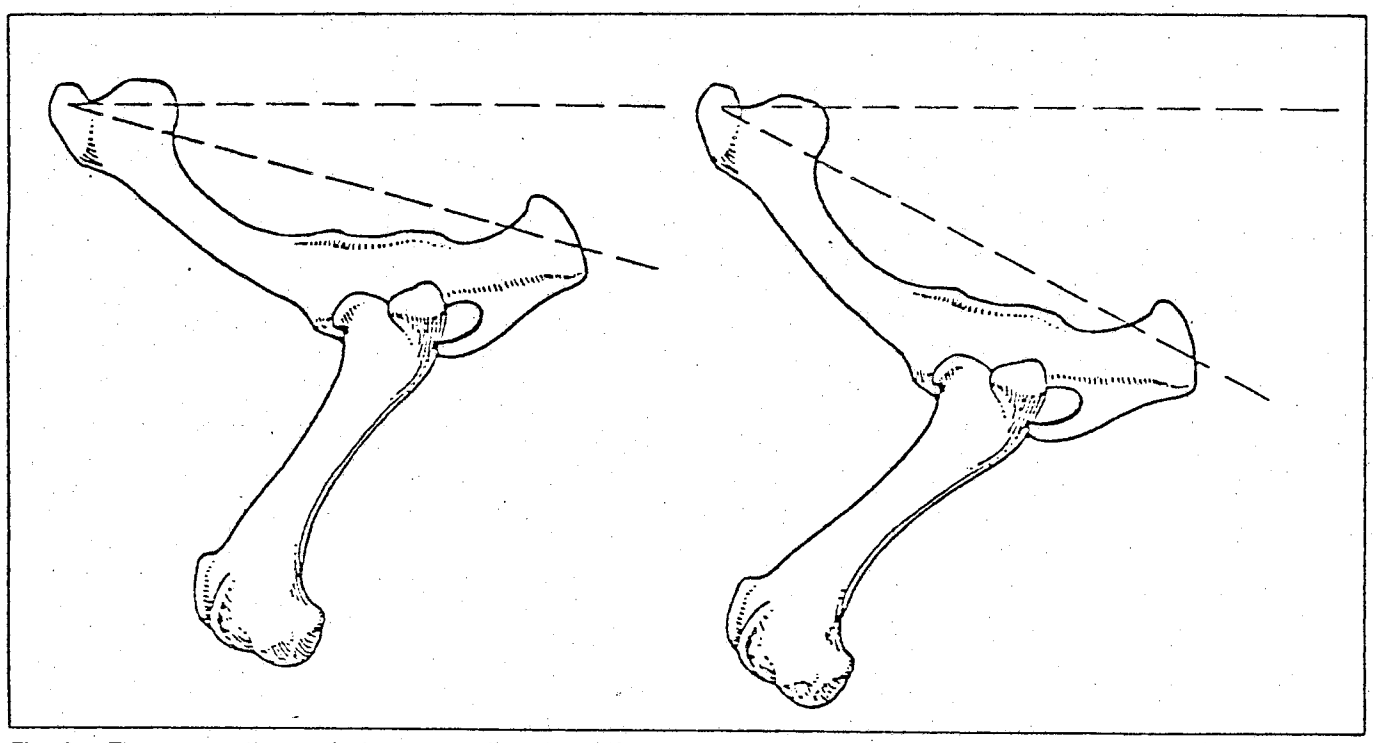


Fig. 2 — The greater the angle between a line drawn from tubera coxae and ischii and the horizontal, the greater will be the vertical diameter of the pelvis (Measurement V of Figure 3).

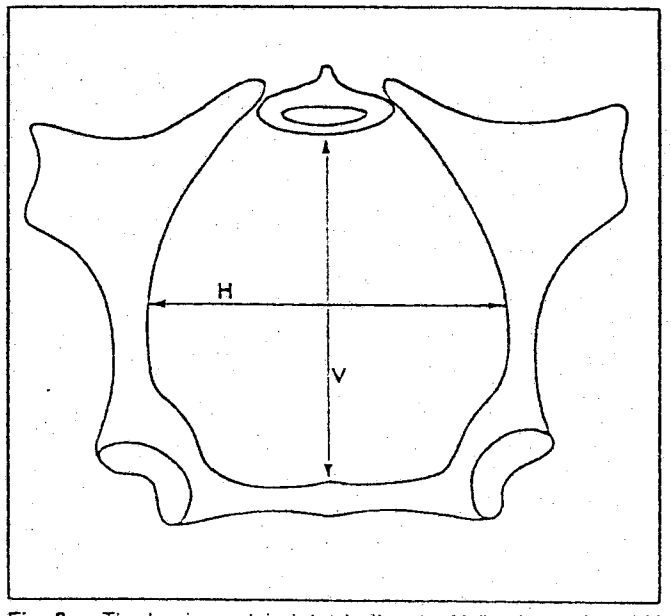


Fig. 3 — The bovine pelvic inlet indicates H (horizontal and V (vertical) diameters. (Redrawn from Hindson 1978)

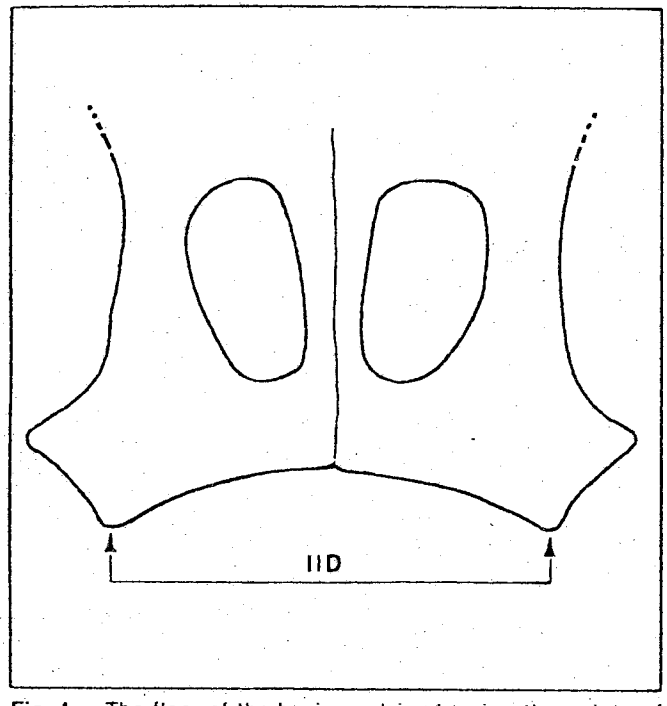


Fig. 4 — The floor of the bovine pelvis showing the points of measurement for the inter-ischial distance (IID). A correlation exists between H & IID. (Redrawn from Hindson 1978)

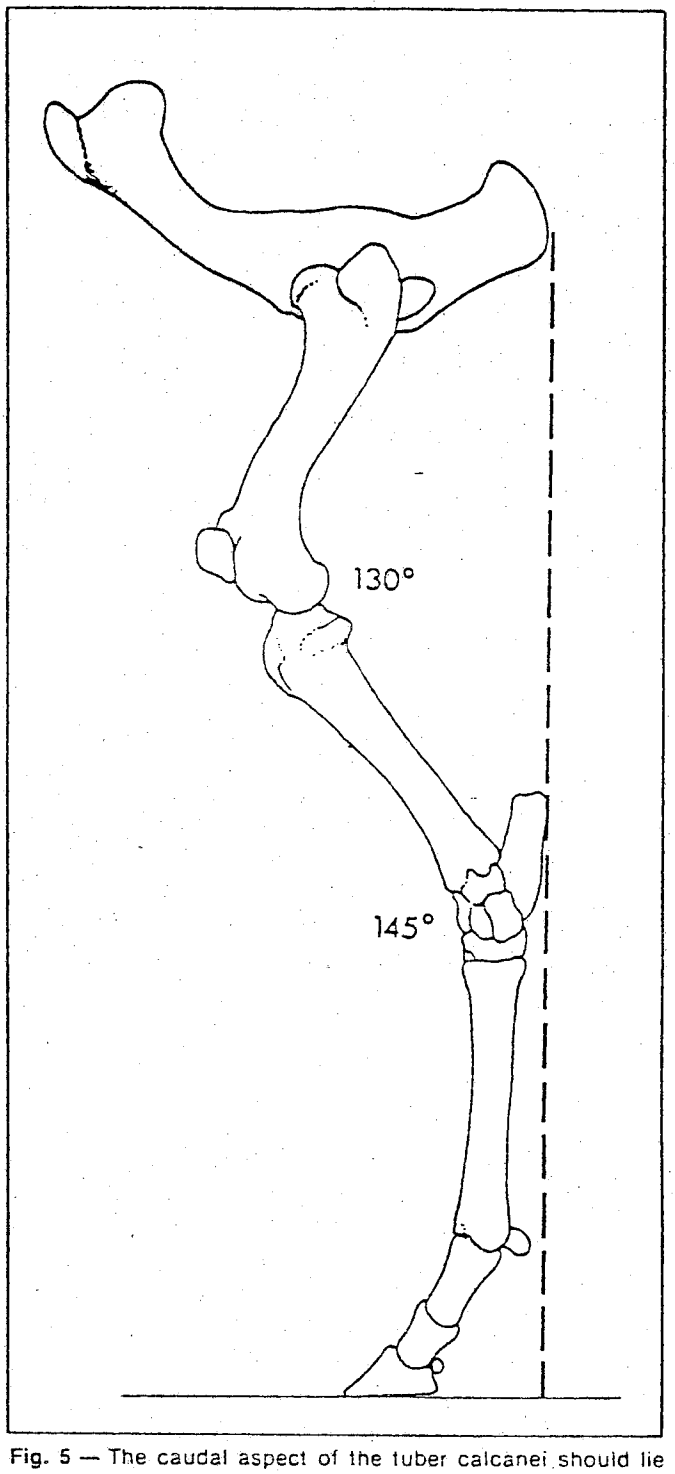


Fig. 5 — The caudal aspect of the tuber calcanei should lie directly beneath the caudal aspect of the tuber ischii.

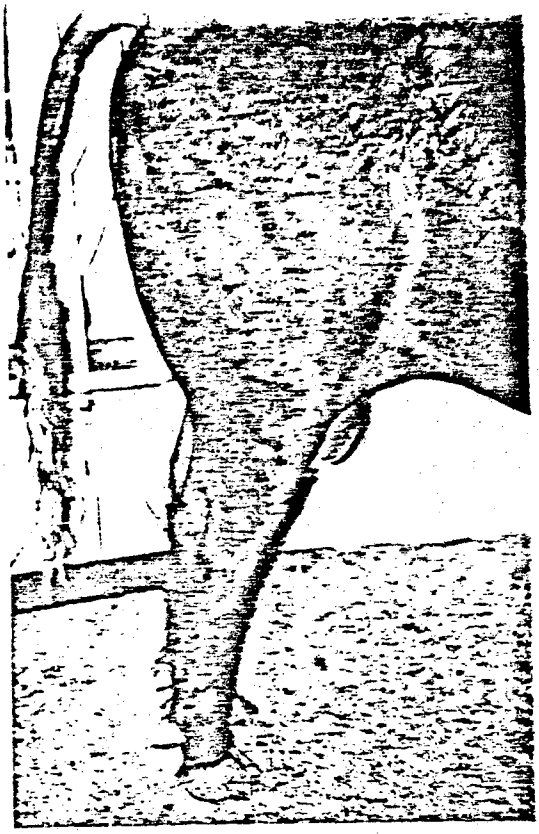


Fig. 6 — A red poll bull with straight hocks that has a camped-back posture.

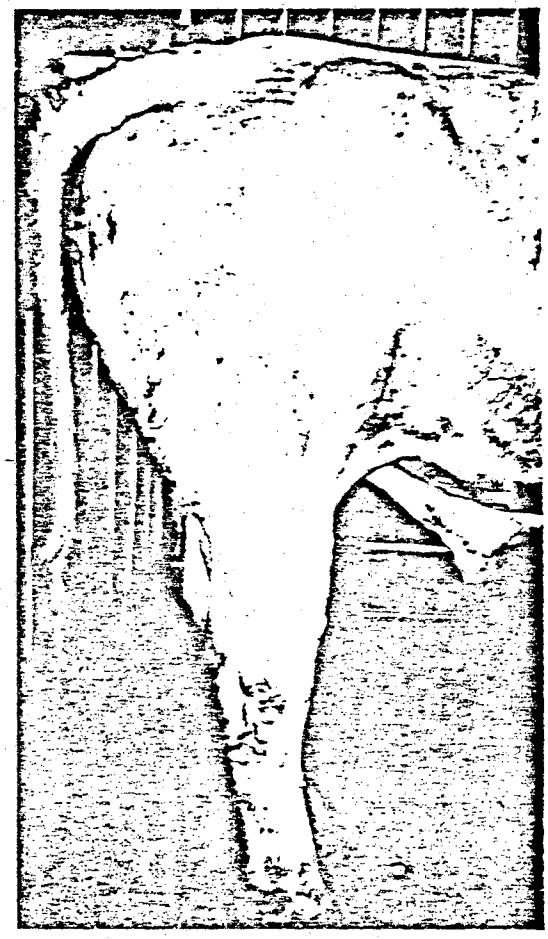


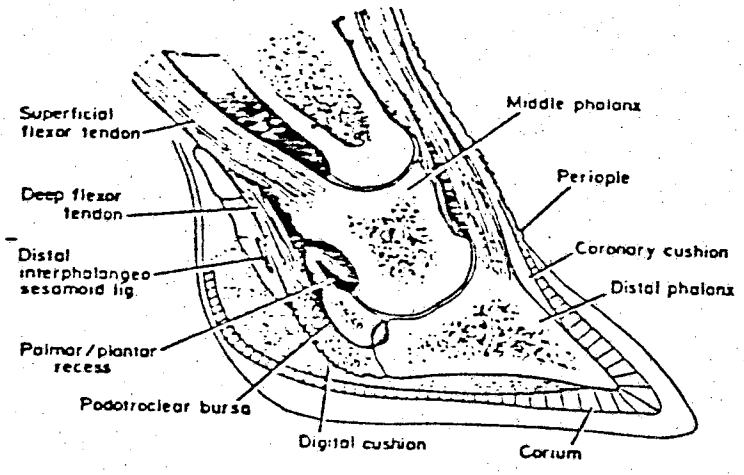
Fig. 7 — A post-legged bull that is camped under giving a posture clinically indistinguishable from spastic pareisis.

THE RELATIONSHIP BETWEEN THE ANATOMY OF THE BOVINE DISTAL EXTREMITY AND PATTERNS OF PATHOGENESIS

Under conditions of domestication the hooves of bovidae are subjected to abnormal levels of concussion and wear or, on the contrary, conditions (well-bedded corrals) that prevent wear. Inadequate exercise limits the blood supply to and from the corium and excessive body weight increases the stress on the corium and other structures. Therefore, quite apart from the ravages of unrealistic feeding, the hooves are subjected to mechanical stress. Experience suggests that this mechanical stress is concentrated in certain areas and that these points can be identified as zones most commonly associated with particular disease patterns. These disease patterns in turn depend upon the anatomical arrangement of the structures lying intimately beneath the traumatized hoof. Therefore, rather than discuss anatomy per se, disease manifestations will be examined in association with anatomical information.

SEPTIC PEDAL ARTHRITIS

The clinical appearance of septic pedal arthritis is of inflammatory and swelling of the region immediately above the coronet. The swelling is more pronounced on the dorsal surface than it is around the bulbs. Radiographic examination of the distal interphalangeal joint can be extremely useful. Widening of the joint space may be observed particularly in the early stages and the presence of gas in soft tissues may be indicative of extra articular pathology. Special radiographic techniques are required to effectively examine the joint and it would be necessary to appreciate normal bony changes that would be found in the digital region of a mature animal.



The distal interphalangeal joint forms a cup with the distal sesamoid (navicular) into which fits the articular surface of the intermediate phalanx. The joint continues dorsally as the extensor process which protects this aspect of the joint. Direct access to the joint from the solear surface rarely occurs. This joint is usually invaded from its axial and abaxial borders which are protected by a complex system of ligaments. The axial and abaxial approaches to the joint are also protected in the main part of the hoof wall. The dorsal pouch of the joint is significantly smaller that the palmar/plantar pouch.

Septic pedal arthritis may occur as a sequel to a septic sandcrack, a necrotic lesion between the claws, by an abaxial/axial approach from a lesion in the sole or by a more complex route to the palmar/plantar pouch. Septic pedal arthritis is in a sense the ultimate pedal disease which when established is difficult to resolve. Each of the routes of infection can produce a clinical manifestation that can be confused with an early septic pedal arthritis. Understanding the pathogenesis of each route can enable the clinician to recognize an early lesion, the treatment of which could prevent the ultimate complication.

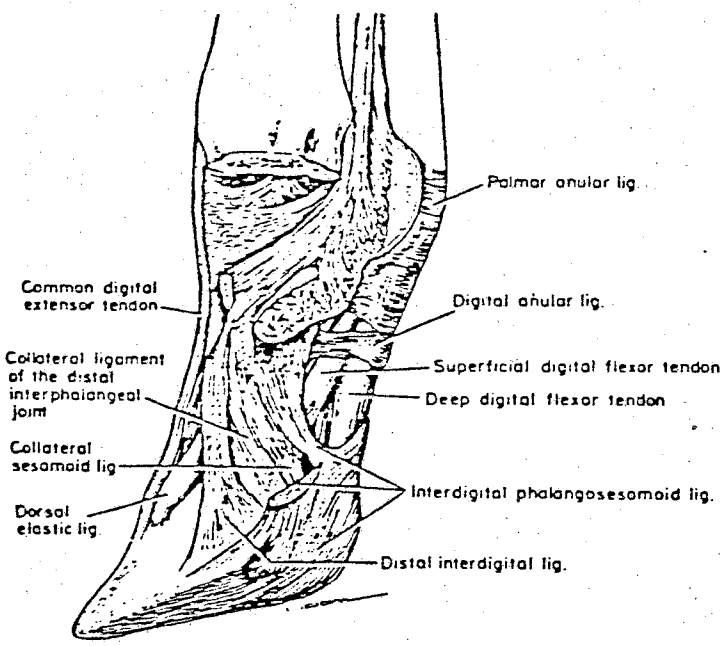
Route 1

The Septic Vertical Fissure

The most external layer of the hoof wall, the stratum externum (This is sometimes referred to as the periople, but this is not the case. The periople is a specialized area of the coronary band at the skin/coronet junction, the stratum germinativum which produces the stratum externum.) The stratum externum functions as a vapour barrier and, therefore, maintains the flexibility of horn. Loss of stratum externum occurs as the animal ages, or in animals maintained on a diet that predisposes to laminitic change or that

are exposed to conditions that may mechanically abrade the layer such as sand and wind. When there is loss of stratum germinativum, the hoof becomes brittle and may split starting at the coronet and extend distally. The major precipitating factor is the angle made by the pastern with the dorsal wall of the hoof. If the conformation of the pastern is naturally upright and any elongation of the toe is present, a mechanical stress will be placed on the coronary band.

Fissures usually occur on the dorsal/abaxial coronet. The fissures can become infected and an abscess may become established. The coronary band of bovidae is broader than equidae. Beneath the stratum germinativum of the coronary band is an extensive region of connective tissue interwoven by a venous plexus. This structure, the coronary cushion, acts as a pump to return



blood from the distal extremity to the general body circulation. Vertical fissures are first seen at the junction of the skin and coronary hoof and extend distally. Small fissures sometimes become infected and an abscess develops beneath the coronary band displacing the vascular cushion and causing extensive separation of the corium from the hoof locally. The abscess causes localized inflammation and edema. The location of the fissure abscess is critical. If located dorsally, the joint is well protected by the extensor process. If abaxially (a very rare occurrence), the joint is located well below the coronary band. However, if the abscess is located in the dorsoabaxial quadrant, the joint capsule will be found to be quite superficial at this point, and there is serious risk that the joint will be invaded.

Route II

Sequel to Interdigital Infection

The axial aspect of the distal interphalangeal joint is less well protected by horn wall than is the case with the abaxial aspect. However, the axial aspect of the joint is protected to a great extent by a more extensive arrangement of ligaments than is found on the abaxial digital surface. Nevertheless, protection is not complete and the joint capsule is vulnerable in the region of the dorsal commissures of the interdigital space. At this point complications following trauma or a debilitating necrotising infection can predispose to serious infection of the joint.

Route III

Infection from a Puncture Wound of the Sole

As was stated earlier, direct penetration of the joint by a foreign body is virtually impossible. Rarely, infection may extend from an abscess under the sole around the distal phalanx and into the joint. This route is rare because the condition is so painful that the owner of an animal intensively husbanded would seek professional assistance. However, under range conditions (or presumably wild) the condition will progress with massive tissue destruction and joint involvement.

Route IV

The Palmar/Plantar Route

This is the most complex route that usually involves an intermediate phase, namely the establishment of an abscess or at least a focus of infection in the region immediately behind the joint. This region is bounded distally by the digital cushion and the deep flexor tendon and abaxially and axially by the distal interphalangeosesamoidean (cruciate) ligament. Proximally the space narrows to accommodate the insertions of the superficial flexor tendon into the intermediate phalanx and the deep flexor tendon as they are contained by the annular ligament of the digit.

The "retroarticular" area can be invaded in three ways.

a) Axially

The region at which axial wall, bulbs, and sole join is known as the axial groove and the horn here is relatively thin. Possibly because of the somewhat obscure nature of the site the axial groove is prone to become eroded by Bacteroides nodosus infection. Perforation of the horny shell and infection may penetrate beneath the layers of the distal phalangeosesamoidean ligament to the retroarticular area.

b) Proximally

Ulceration of the sole of the lateral hind claws is extremely common in dairy cattle kept under intensive conditions. Poor hoof care, excessive solear trauma, and wear or even over-zealous hoof trimming can cause an ulcer to appear. The typical region has poor vascularisation because of the restricted space between the palmar/plantar border of the distal phalanx and the sole. The space is further restricted by the presence of elastic tissue from the forward extension of the digital cushion. If the sole is worn or incorrectly trimmed, mechanical pressure will cause an ischaemic necrosis. A similar mechanical effect will occur when bulbs are eroded by B. nodosus and the function of the bulb becomes minimal thus throwing concussion to the sole further forward. The ulcer can be resolved quite readily but occasionally the deep flexor tendon will become necrotic and avulse from the distal phalanx. The retroarticular region loses its tendonous protection and a focus of infection (but not an abscess) becomes established. It should be noted that when avulsion of the deep flexor tendon occurs, the tip of the affected hoof will fail to be flexed during progression and will eventually ankylose in a more or less dorsiflexed position.

c) Abaxially

The abaxial route initiates at the white line in the palmar/plantar quadrant. Excessive concussion causes a breakdown of the adhesion of the white line and infection becomes trapped. The infection passes proximally possibly between two lamellae and quite frequently establishes an abscess of the coronary band. This abscess is clinically similar to that which characterizes an infected fissure with the difference that this abscess is located just in front of the bulbs.

By chance a small percentage of abaxial tracks will pass close to and invade the abaxial aspect of the navicular bursa. Infection of this structure carries sepsis to the retroarticular region. Because of the tortuous route, the depth of the infection and the fibrous nature of the structures surrounding the sequestered infection natural drainage is inadequate. The bulb swells, becomes inflammed and tender. Because of the pressure within, the plantar/palmar pouch may be invaded. Necrosis of the distal sesamoid (navicular bone) or deep flexor tendon may occur; osteomyelitis and exostosis formations may be seen in the more advanced cases.

THE RETROARTICULAR ABSCESS IN CATTLE

The retroarticular abscess is described (Greenough 1980) as an accumulation of purulent material in the virtual space between the intermediate phalanx and the deep flexor tendon. Most commonly infection arrives at this location by a circuitous route, via the white line, passing up

the interlamellar spaces to infect the navicular bursa and thence enter the retroarticular space (Greenough 1963). The next most common cause of the condition is the extension of infection from a very advanced case of suppurative arthritis of the distal interphalangeal joint. It should also be noted that a case of primary retroarticular abscess that is neglected will eventually cause the joint to become involved.

The retroarticular abscess is frequently misdiagnosed or the true nature of the pathology is not understood. In the early stages there is very typical swelling of the heel bulb which will be extremely painful (Figure 1). Unless the coronary band is swollen the condition should not be confused with suppurative pedal arthritis. In cases of "phlegmon" (foot rot) both digits and the interdigital space will be inflammed and swollen. Confirmation of the diagnosis can sometimes be established radiographically by the presence of gas in the heel. Because the disease very frequently starts in the abaxial white line beneath the bulbs, an ascending track may be located. Also in some cases the horn of the coronary band may be detached and the seepage of pus and serum may be seen.

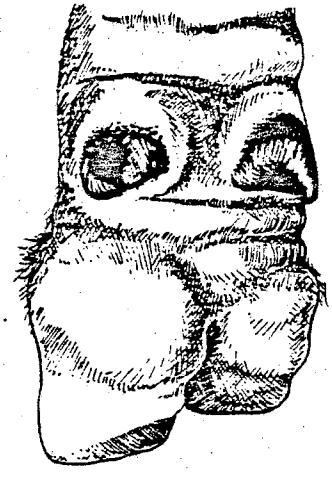


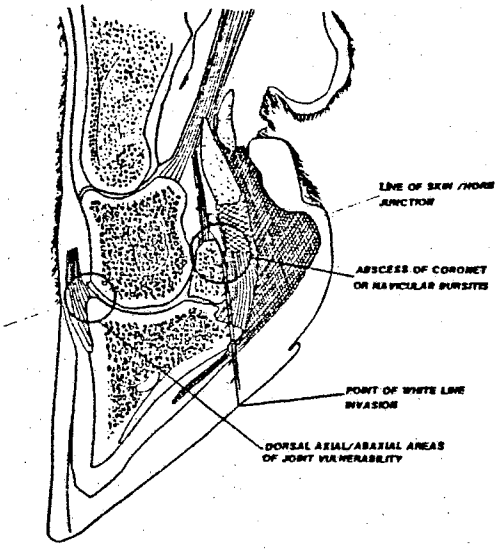
FIGURE 1

PODODERMATITIS CIRCUMSCRIPTA (ulceration of the sole)

This condition occurs on the lateral claws of the hind limb. The "ulcer" will be located close to the abaxial border of the sole, approximately one-third the distance from the heel bulb to the toe. The ulcer measure 2 cm. in diameter and appears as a knob of granulation tissue. At the time of examination the ulcer is usually covered with a pad of soft horn. Horn necrosis is often associated with the condition. The animal will walk with a crampy stilted gait, and may arch its back or adopt an abnormal posture with the hind legs "camped back". It may also stand with its toes on the edge of the gutter.

Many theories have been advanced regarding the pathogenesis of this disease. Pressure necrosis provides the most logical explanation. The distal surface of the distal phalanx is slightly concave and the posterior border of this concavity is the point at which the bone most closely approximates the sole and it is the point at which "so called" ulceration occurs. This restricted area between horn and bone also contains a significant forward protrusion of the digital cushion. The area is also anatomically remarkable for being the region of the sole in which the thickness of the horn is at its greatest.

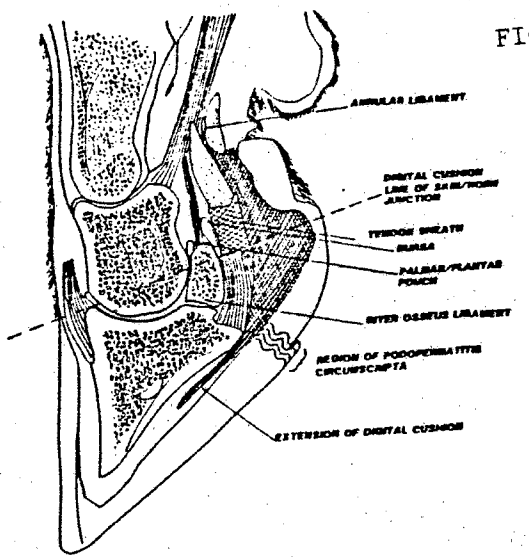
FIGURE 2



Under managemental conditions that cause excessive wear of the sole (i.e. confinement on concrete), concavity of the bearing surface is lost and abnormal pressures are brought to bear on the suspectible area. This pressure causes a localized trauma of the blood vessels and this in turn interferes with horn production. The condition is also observed after incorrect hoof trimming where the operator has either removed too much horn from the sole or lowered the heels too much in the region under the bulbs.

The clinician should bear in mind that if the condition is observed in the lateral digit of one limb that the disease process will almost certainly be in some stage of development in the lateral claw of the other limb. Examination of both extremities is therefore appropriate. It should also be noted that there is a very high incidence of this condition in herds that are managed under loose stall systems. There is an increasing body of opinion that pododermatitis circumscripta is indirectly associated with excessive horn wear that is related directly to the softening of the horn as a result of subclinical laminitis occurring in susceptible animals.

FIGURE 3


ANNULAR LIGAMENT
DIGITAL CUSHION
TENDON SHEATH
BURSA
PALMAR/PLANTAR POUCH
DISTAL SESAMOID LIGAMENT
REGION OF PODOPHLEGMATITIS CIRCUMSCRIPTA
EXTENSION OF DIGITAL CUSHION

TREATMENT OF LESIONS OF THE BOVINE DISTAL EXTREMITY

Introduction

An infection of the distal extremity, however innocuous it may seem, presents a significant risk of intractable complications developing that would markedly reduce the longevity of the animal. Simple infected lesions are frequently given a cursory examination because restraint adequate for a thorough examination is either hazardous or excessively time consuming. A similar philosophy operates in respect to treatment with penicillin being used as a panacea and its failure being interpreted as an indication that serious therapy is indicated. Precise diagnosis and accurate prognosis will make an evaluation of the respective risks realistic.

Interdigital Infection

1. Fusobacterium necrophorum -

probably working together with Bacteroides melanagenius (1) are the pathogens implicated in foot rot (interdigital phlegmon). Necrobacillosis of the liver, buccal cavity and other organs has been observed in cattle (3) and the devastating effect of the disease in wild ruminants has been described (2). It is not the purpose of this paper to unduly stress the epidemiological aspects of foot rot. However, topical treatment is of particular importance. Firstly, a necrotising interdigital lesion will discharge an abundance of organisms that will contaminate the environment. Secondly, the loss of interdigital epidermis will increase the risk of infection entering the distal interphalangeal joint. Therefore, in addition to the parenteral administraton of medication, consideration should be given to use of a

topical dressing and the application of a bandage. A useful dressing is a combination of equal parts anhydrous copper sulphate and sulphamethazine. Interdigital lesions, therefore, should be cleansed, topical dressing applied without gauze pads, cotton batten, or bandages being placed between the digits. The digits should be bound tightly together, then the whole distal extremity should be inserted in a plastic bag and the whole enclosed by the application of an adhesive elastic bandage. Simple as this procedure may seem, it has, in the writer's hands, proved to be a reliable adjunct to both parenteral treatment and surgical interference.

2. Bacteroides nodosus

This organism has been proven (4) to be the causal agent of interdigital dermatitis (or scald). This disease per se is of little economic importance and has been regarded as of little significance, although its incidence is high under unhgyienic conditions. Dutch workers (5) believe that the importance that can be attributed to B. nodosus is its ability to invade and erode horn. Erosion of the bulb is indeed a very common condition and results in a disturbance in shock absorption mechanisms and a shift in weight bearing. These changes are associated with traumatic asceptic coreitis and pododermatitis circumscripta.

Individual cases of interdigital dermatitis that are confined to the interdigital space respond rapidly to the application of anhydrous copper sulphate application. When several animals are affected or when the bulb is involved efforts should be made to introduce the use of a foot bath. Temporary baths may be constructed of a 2" x 6" frame over which a tarpaulin may be fixed. This shallow bath may be filled with a 5% solution of formalin. Copper sulphate is sometimes used, but it deteriorates rapidly and stains hair or wool.

The Vertical Fissure (Sandcrack)

Vertical fissures located on the dorso abaxial quadrant of the coronet present a particular hazard to the joint. The coronary abscess must be dealt with promptly by removing a small segment of the conorary horn. In some instances, it may be advisable to apply magnesium sulphate/glycerine mixture to ensure total cleansing. This application should not be in place for more than 12 hours in this location. If the operator is confident that the lesion is clean, copper sulphate/sulphamethazine powder may be packed into the cavity and a gauze swab held in place by an elastic adhesive bandage around the coronet. Exuberant granulation can be a problem sequel to the treatment of vertical fissures if a) too much horn is removed, and b) if an astringent is not applied to the wound.

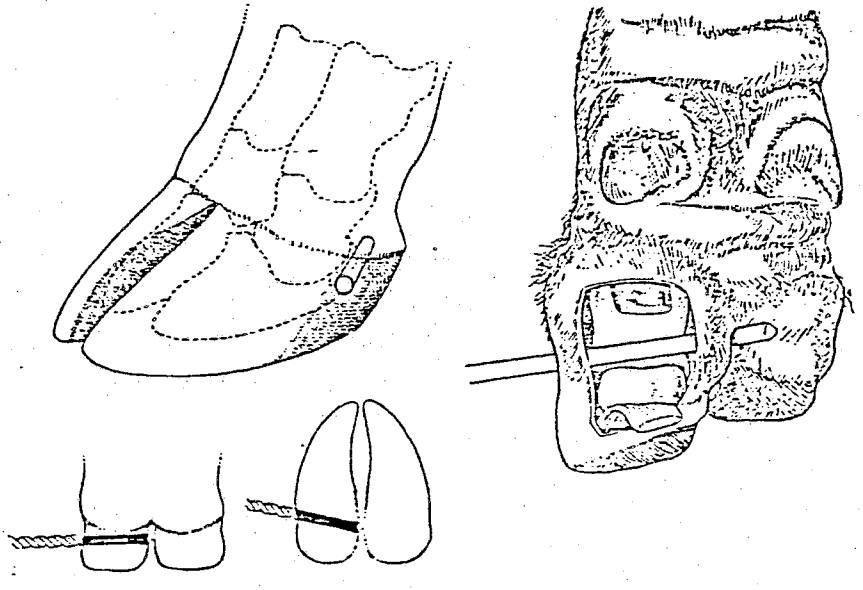
The Retroarticular Abscess

The most effective treatment for the retroarticular abscess is to establish drainage and to restrict movement of the digit.

In order to establish drainage, the writer has found it most practical to approach the abscess from the abaxial surface of the digit. In almost all of the cases treated by the writer a sinus has been present and preliminary removal of the abaxial wall has been necessary. The sinus opening should be enlarged sufficiently to admit the operator's finger. A probe should them be passed into the abscess. The probe may be pressed into the axial wall with sufficient force to permit palpation of the tip through the skin between the bulbs. An incision is then made down into the probe and the opening enlarged sufficiently to allow for the admission of a 15 mm drainage tube. The drainage tube should be sufficiently firm to prevent collapse of its lumen when subjected to the considerable pressures of the region. It is useful to seal the distal end of the tube and to develop openings in that part of the tube that will lie within the abscess. The proximal end of the tube can then be strapped to the proximal limb and thereby provide a convenient route by which the abscess may be irrigated. Irrigation with sterile saline is useful but no benefit has been observed from the addition of antibiotics or enzymes to the irrigant.

In less than a week the abscess will collapse and the tube may be conveniently removed.

Immobilization of the digit is important. A common sequel to the R.A. is necrosis and evulsion of the insertion of the deep flexor tendon. A good method of immobilization is to wire the toes together, apply a block to the sound digit and a plastic bridge between it and the claw under treatment.



Septic Pedal Arthritis (Arthrodesis of the distal interphalangeal joint)

Arthrodesis may be a practical alternative to amputation in selected cases of deep sepsis involving the distal interphalangeal joint. Either intra-articular or retroarticular infection with lameness of no more than seven days duration is likely to respond well, since sepsis will probably not have spread proximally. Radiographic evaluation of the destructive change is invaluable. The three main procedures are drainage, curettage, and immobilizaton.

Drainage

The site for entry to the joint is at a level approximately two-thirds of the distance fromt he coronet to the bearing edge of the wall and about two-thirds of the distance from the "toe" to the plantar aspects of the bulb. Correct location of the drainage tube is vital. A one-half inch (12 mm) drill is used to enter the abaxial wall at this point, directed dorsally across the distal extremity of the distal phalanx to emerge between the hooves close to the skin line at the dorsal aspect of the interdigital space. The destruction of horn producing tissue wall in minimal.

The most suitable drainage tube is of plastic tubing stiff enough to resist compression but flexible enough to conform to the irregularities of the artificially produced drainage track. Ideally, the drainage tube should be occluded at its distal extremity and have openings at points within the hoof to permit the escape of irrigation fluid.

Irrigation should take place for a period of five to seven days. Continuous drip irrigation can be done using a five-gallon reservoir with sterile saline and soluble antibiotics such as crystalline penicillin at a concentration of about 10 grams per 20 litre reservoir. If intermittent irrigation three to four times daily is perferred, the lesion may be first flushed with saline and then infused with a concentrated antibiotic solution.

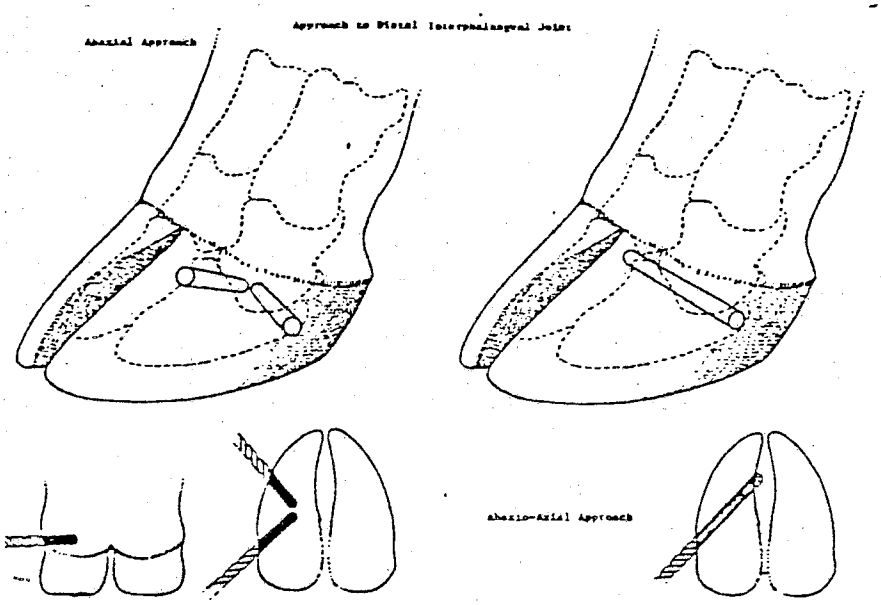
Curettage

Depending on the stage of tissue destruction, necrosis of the deep flexor tendon, distal sesamoiditis, and osteoporosis of the distal phalanx may occur. Curettage of the distal phalanx or the joint is usually required while removal of necrotic tendon or portions of distal sesamoid bone is essential if sequestration is to be avoided.

Immobilization

After surgery, mechanical immobilization of the distal interphalangeal joint improves the speed of ankylosis, reduces the amount of bony deposition necessary to eliminate movement, and reduces the extent to which infection will spread. The two hooves must be immobilized either in a symmetrical manner or with the affected digit flexed in relation to the contralateral hoof. This latter alternative relieves tension on the deep flexor tendon and the associated anticoncussive devices of the bulb.

The two "toes" should first be wired together. Two holes (4 mm) are drilled 3 cm apart immediately medial to the white zone (line) in the abaxial region of the "toe". A loop of baling wire is passed through the holes from the solear surface to be turned over the abaxial wall to the holes on the abaxial wall of the contralateral claw. After passing through these holes, the wire is twisted tight and spare ends removed.



Next a bridge of "Technovit" is formed around the "toes" to incorporate the wire. This bridge should be substantial and exended all around both "toes". The operator may advantageously put a wood block onto the sound hoof.

No further immobilization should be attempted until irrigation has been completed, but at this stage, when the lesion shows signs of settling, a cast may be applied to enclose the distal extremity as far as the fetlock. A suitable window(s) should be left in the cast in order that the progress of the condition may be observed. A plaster cast deteriorates rapidly unless it is protected from moisture, and it helps to apply a plastic bag over the entire cast and to cover it with adhesive plaster. The cast should be kept in place for six weeks and be replaced if it deteriorates.

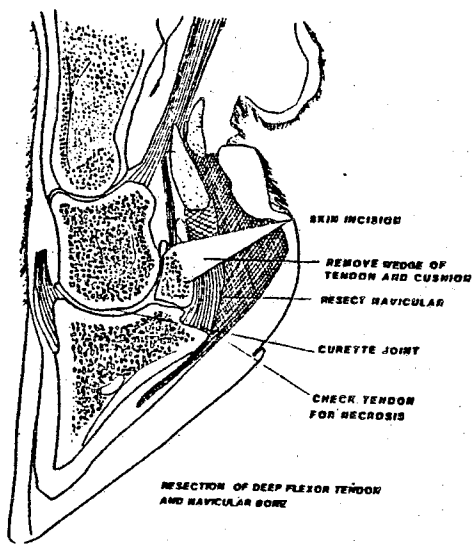
The most rapid resolution has followed the insertion of drainage tubes directly through the focus of infection. Where natural resolution has already started, surgery is likely to cause an acute reaction and is, therefore, contraindicated.

Resection of the Deep Flexor Tendon and Distal Sesamoid with Arthrodesis of the D.I.P. Joint

This procedure sounds more complicated than it is in practice. Various approaches have been described by Assmus (1964) Westhues and Breur (1964) Marolt (1966) and Clements (1965).

The digital region must be thoroughly cleansed and prepared for aseptic surgery. The best approach (Ferguson 1981) is made by making an incision about 12 to 15 cm in length around the bulbs approximately .5 cm above the skin/horn junction. A wedge of the digital cushion should then by excised, the base of the wedge facing the deep flexor tendon. Approximately 1 to 2 cms of the tendon is then removed. At this point it should be possible to forcibly reflex the entire bulb distally in order to expose the navicular bone. This structure is difficult to remove and the procedure involves incising the axial and abaxial ligaments and then cutting through the interosseus ligament of the phalangeosesamoidean joint. With the navicular removed the joint is exposed and may be curetted. The curettage should continue until it is possible to form an irrigation opening on the dorsoabaxial region of the coronary band.

The insertion of the deep flexor tendon should be examined and the remains of the tendon removed if any necrosis is present. An irrigation tube should be inserted and the wound closed with both tension and interrupted skin sutures. The irrigation should proceed as would be the case for arthrodesis of the D.I.P. joint and thereafter the entire digital region should be immobilized. First intention healing is unlikely to occur but the wound does eventually heal and although the prognosis must be guarded the animal can be returned to functional efficiency without deformitis occurring in the hooves.



Treatment of Pododermatitis Circumscripta (Sole Ulcer)

If pododermatitis circumscripta is encountered in the early stages, that is before the defect in horn growth has penetrated the superficial layers of horn, corrective hoof trimming may be successful. The bearing surface of the wall must be left in tact and any accumulated horn removed from above the level of the granulation tissue. The removal of the typical knob of granulation tissue is not necessary. In advanced cases fragmented rotten horn will be found covering a well-developed granulating button. The excessive and rotten horn should be removed. If the hoof trimming can be judged to relieve pressure from the "ulcerating" area recovery may be spontaneous. In most cases, however, the clinician would be wise to fix a wooden block to the sound claw with acrylic. Suitable blocks are provided in Technovit "kits". It may be necessary to cut these blocks down to the shape of the soles of some animals. Bandaging of the lesion may be counterproductive and the application of a dressing is probably of no great value.

HOOF CARE

The majority of hooves requiring attention will have resulted from overgrowth due to lack of wear. This lack of wear will occur when the hooves become excessively hard, when the surfaces of a pen are too soft, or when the animals have a restricted opportunity for movement. Unrealistic nutrition can play a part in altering the patterns of horn growth and specific congenital abnormalities of conformation may be identified. In this paper only the management of the irregularities of the normal hoof will be considered.

The Normal Hoof

The normal hoof is comprised of four distinct parts:

a. The Wall. The axial wall has a relatively small surface area and is reflected on a dorsal border to the thick abaxial wall which in its distal extremity is the major weight-bearing area. The wall is composed of a central "stratum medium" consisting of tubular horn, a stratum externum (periople) which is the water retaining coating of the hoof, and an inner stratum internum or lamellatum which interdigitates with the lamellae of the corium.

   The dorsal surface of the hoof will normally be straight, the axial surface may be slightly concave, and the abaxial wall slightly convex.

b) The Coronet and Bulb. These two structures are contiguous and composed of a softer textured horn with fewer tubules than are found in the wall.

   Axial and abaxial grooves are located at the junction of heel and wall. The axial groove represents an area of extremely thin horn and the bearing surface beneath the abaxial groove marks an area of maximum concussion and wear in the hind digits.

c) The Sole is anatomically a crescent-shaped area at the apex of the solear surface of the hoof. There is no physical demarkation between sole and bulb. However, the bulb (or Torus) contains the fibrous elastic tissue of the digital cushion which extends as an apex of elastic tissue beneath the solear surface.

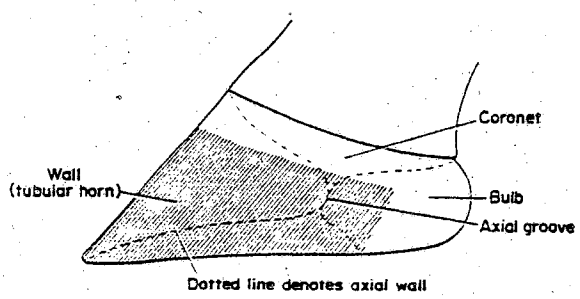
d) The White Zone. This is a homogenously keratinized area that forms a junction between the wall and the sole.

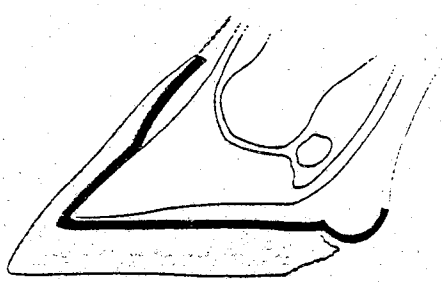
The corium of the coronet is a highly vascular layer that is particularly specialized in the region of the coronary band. Here there is a venous plexus (the coronary cushion) which functions during locomotion to pump blood from the extremity back into the circulatory system. Papillary pegs extend into the substance of the horn of the corium and are continued as the tubules of the stratum medium. At the skin coronet junction a small groove will be noted that is referred to as the limbus and the stratum germinativum of this zone produces the periople.

The Irregular Hoof

The objective in trimming the hoof is to restore balance of the digits both in the saggital and transverse planes.

The regions of the hoof.



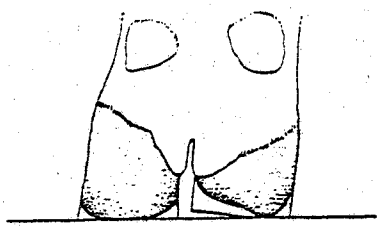


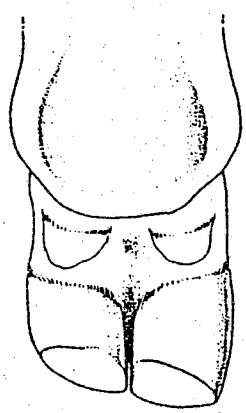
Erosion of the heel (a sequel to interdigital dermatitis).

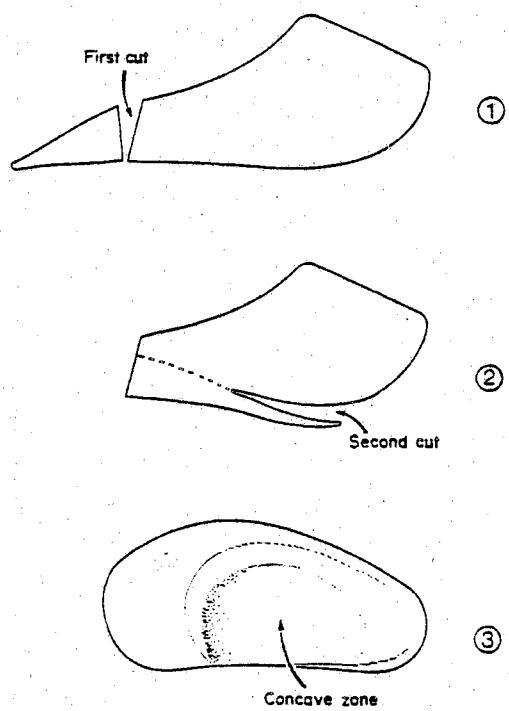
a. Elongation of the Hoof. When the rate of wear is reduced, the wall elongates and in the extreme manifestation will form a tube. The pressures of weight bearing frequently bend the apex of the hoof to establish a concavity in the dorsal wall. If the hoof is left untrimmed for any length of time, the distal phalanx will mould to the new hoof form and the abnormality will become permanent. The excessively elongated hoof also throws abnormal strain on the suspensory/flexor system and lameness or chronic changes may be observed.

The elongated apex of the hoof may be removed provided that the bearing surface of the wall beneath the apex is approximately reduced. The most effective method is to start cutting the wall at the wall/bulb junction and proceed towards the apex removing an increasing amount of horn. It should be remembered that the bearing surface of the bulb is composed of the thickest horn in the solear surface and that at the apex of the sole is relatively thinner. The common practice of removing equal amounts of horn from all over the bearing surface is, in general, inadvisable.

Having reduced the wall, it is important that the sole should be pared into a concave shape towards the axial groove. As has been indicated before, the majority of weight bearing must take place through the abaxial wall. Additional weight and, in particular, concussion is absorbed by the bulb. Direct weight bearing of the sole must be limited to a zone of about 2 cm within the white line. The remainder of the solear surface should be rendered concave.







b) Overburdening. The term overburdening is used to indicate imbalance of horn between the two claws and is usually manifested by overburdening in the bulbar region. The presence of excessive horn in the region of one bulb (almost invariably that of the lateral hind claw) causes abduction and separation of the limb or turning in of the hocks (cowhock). Normally, overburdened has only a transient effect that can be corrected by trimming; however, prolonged neglect can lead to irreversible bony moulding that results in a disadvantageous conformational change.

In practice it is preferable to select the more normal of the two bulbs (usually the medial in the hind foot) and to trim it first. This should be used as a guide for the removal of horn from the contralateral bulb.

Conditions Commonly Encountered During Hoof-Trimming

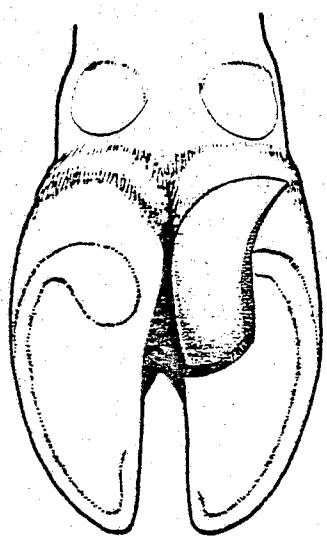
a. Haemorrhage. Discoloration of solear horn can occur due to the presence of pigment (melanin) and can be confused in appearance with the presence of blood. If there is a generalized extravasation of blood beneath the horn of the solear surface, it usually indicates excessive wear of the solear surface. Such a state is readily detected by digital pressure which reveals an extremely thin sole. Any form of hoof trimming is contraindicated.

The second type of haemorrhage commonly encountered is a flecking of the horn. In the opinion of some, this can be associated with subclinical laminitis; others feel that trauma is involved. Probably under domesticated conditions both etiological factors play a part. During the process of hoof trimming haemorrhages will be encountered, and, provided that the sole does not "give" under digital pressure, the blood-stained horn may be pared away.

In milk cattle confined on concrete a very special phenomenon is observed in the lateral hind claw. This is the sole ulcer (pododermatitis circumscripta) which appears as a circumscribed haemorrhage or area of granulation tissue in the central region of the bearing surface of the bulb. This is an extremely common phenomenon in Holstein cattle but does not appear to have been reported in wild bovidae.

b) Under-running of the Sole - False Sole. The presence of fluid (pus, blood, serum) in the corium or the region of the stratum germinativum apparently causes of disruption of solear horn production. The fluid spreads extensively as a film between the stratum germinativum and the overlying sole. However, the production of new horn starts immediately with the result that there are in fact two soles. The new sole is very delicate when first exposed but rapidly hardens within

about four days. If possible, the old sole should be removed in two stages, half at the first examination and the remainder two weeks later. This is not always possible because of the restrictions imposed by difficulties associated with examination. Protection of a newly-exposed soft sole is contraindicated in most cases because exposure to air and mechanical stimulation of stratum germinativum are necessary. Restriction of movement and confinement on very soft dry bedding is highly desirable. In all cases, only the soles or bulbar horn should be removed, the abaxial wall should be left intact.



A false sole may be stripped from the bulb but the abaxial wall should be left in tact.

c) Erosion of the Heels. Recent work suggests that Bacteroides nodosus is the cause of interdigital dermatitis in bovidae. It is suggested that the same organism can cause a progressive loss of bulbar horn. Whatever the true etiology, erosion of the bulbs does occur and is usually related to unsanitary conditions.

When bulb erosion is extensive, considerable care must be taken when trimming the rotting horn. The horn should be shaped to provide a concave surface with weight bearing transferred to the abaxial wall. Poorly executed hoof trimming will result in pressure being transferred to the center of the solear surface with a resultant aseptic coreitis or a circumscribed ischaemic necrosis of the corium.

Specific Hoof Deformities

The foregoing has discussed briefly the identification and management of hoof irregularities that result from confinement. These irregularities should not be confused with true deformities which are presumably rare in wild ungulates and will only be noted in passing as follows:

a) Curved Claws

i) Concavity of the axial wall of the medial foreclaw.
ii) Spiraliform deformity of the lateral hind claw.
iii) Normal curved claws - usually lateral hind claws.

N.B. These are generally considered to be hereditable forms or that the predisposition to deformity is hereditable.

b) Wrinkled Claws. Deformities in this group probably can be associated with a high plane of concentrate feed/overweight/lack of exercise/hard surfaces. They are characterized by ridging of the hoof wall, flattening of the hoof, and softening of the horn.

Equipment and Technique

Hoof trimming is unpopular with veterinarians partly because of the attendant problems of restraint but also because of the so-called inadequacy of equipment.

The best quality knives in the writer's opinion are those manufactured in Sweden. In any hoof care situation a pair of "sharp" left-handed and a pair of "sharp" right-handed knives should be available. A small triangular file should be used to put an edge on the blade prior to attending to the animal. Double action, well oiled and properly adjusted hoof cutters with sharp replaceable blades are the author's personal choice. Heavy duty industrial rotary sanders with a heavy grit pad is a useful instrument. In recent years, Dutch and Swiss workers have developed metal blades that can replace the sanding pad of the rotary sander. The metal faceplate (one is flat, the other is slightly conical) is perforated with holes to which a slightly raised carbon steel cutting edge has been welded. Reports on these tools suggest that this equipment should be evaluated in North America.

THE TREATMENT OF DIGITAL PROBLEMS WITH ACRYLICS

It is almost twenty-five years since Becker (1956) first discussed the use of plastic materials in the treatment of digital diseases in animals. These materials have been used to repair birds' beaks (Becker, 1974) and for the external fixation of bone fractures (Becker, 1957; Dietz and Kuntze, 1959; Sonnischen, 1964). These materials have been frequently used in the repair of equine hooves (Evans et al. 1966; Becker, 1961; Jenny et al., 1965; Hutchins, 1969) and to reduce concussion to horses' feet (Marks et al., 1971). Plastics have been used in the treatment of distal phalangeal fractures in cattle (Vaughan & Osman, 1967 and Hertsch, 1972) in surgical shoeing of cattle (DeMoore and Bouchaert, 1962; Brewer, 1963;, Koch, 1965), and in the treatment of various diseases of the bovine extremity (Thomann, 1963; Wiersma, 1965; Jurzweg, 1965; Funk, 1970).

Property of Acrylics (Polymethyl Methacrylate)

The acrylics are a family of materials giving a sparkling glass-like transparency (Perspex) and resistance to deterioration in outdoor environment, together with stiffness. Modification of the classical structure can produce polymers that are less rigid. The usable products is a mixture of polymer powder and a liquid monomer with a suitable polymerization initiator and activator. On mixing the two, polymerization occurs with liberation of heat (Bloch & Hastings, 1972).

The liquid monomer is a powerful lipid solvent and is, therefore, toxic to tissues. Residual monomer is invariably present after curing and this must be taken into consideration when applying the material directly to living tissues. Monomers may contain a bactericide but pathogens have been proven to survive in the material; however, for the application described in this paper, this observation has little application. It would be wise, nevertheless, to ensure that the product does not get contaminated during handling or storage.

Preparation and Use of Polymethyl Methacrylate

Polymethyl methacrylate (Technovit 6091) is the preparation most commonly used in veterinary practice. It has good adhesion to horn, is abrasion resistant and resistant to acids and alkalies. The tensile strength of the curing material has proved to be highly compatible with that of the horny hoof.

The surface to which the acrylic is to be applied must be clean, dry, and free from grease. Initial preparation should be cleansing with a detergent and water and then dried, preferably with heat. Chloroform may be used to ensure complete removal of grease. Flaking horn, if present, should be removed and the surface lightly scored with a hoof knife to increase the contact surface area.

The mixture (one part powder to 0.7 part liquid) should be prepared in a disposable beaker shortly before use. The total curing time is approximately five minutes but during the first two minutes the mixture is too liquid to permit controlled handling. Immediately prior to the application of the acrylic the horn surface may be brushed with a light coating of the monomer to improve adhesion. The material may be applied directly to the hoof with a disposable spatula. In many applications greater control may be exercised if a 2 mm plastic bag is used as a glove/applicator. The operater's hand is inserted into the bag and the polymerizing material is then poured into the plastic-protected palm of the hand. The changing temperature and consistency can be accurately judged, and as it warms and becomes plastic, it may be moulded into the desired position. With experience an even, smooth application can be made and as the acrylic hardens, the plastic film may be easily peeled away. When the acrylic is applied directly to sensitive tissues heat generation (and rate of curing) should be retarded by running cold water from a hose over the material as it solidifies. Thicknesses of less than 10 mm cannot be relied upon to bear the weight of a 500 kg animal without fracturing. Ideally, two or more layers of acrylic (7 to 10 mm thick) should be laid down one over the other. Reinforcement with solid or polyfilament wire should be considered if bridges are to be created; for example, between the two digits.

Windows or examination ports may be desirable for the purpose of examination, sample taking, or irrigation. These ports are most easily created by taking a plastic disposable syringe of suitable diameter and cutting through the barrel approximately 12-15 mm from the proximal finger grip flange. The acrylic can then be formed around the inspection port after it has been placed in situ. The distal end of the syringe plunger may be cut from the plunger shaft and used to effect temporary closure of the port. The hardened acrylic may be removed with hoof shears; it can be milled with a sander or cut with a hot spatula (caution-flammable).

Applications

Shoes. The shoes usually referred to are the wooden blocks (or lifts) that are supplied with a "Technovit 6091" kit. A thin layer approximately 3 mm) of the product is applied to the block and to the sole of the hoof. The two surfaces are applied and technovit will be squeezed out around the periphery. This material is moulded around the walls of the hoof and over the

juxtaposed surfaces of the block.

Blocks are usally fixed to "sound" claws in order to provide functional relief for an affected claw. This technique has become an important standard treatment for pododermatitis circumscripta (Sole ulcer, Rusterholz ulcer). Vaughan & Osman (1967) and Fund (1970) describe the value of the wooden lift in the treatment of fractures of the distal phalanx in cattle. A useful modification of this technique is to totally immobilize the affected digit in respect to the sound blocked digit. Fractures of the distal phalanx usually take place through the proximal half of the joint surface. The proximal fragment of bone is relatively small and attached to the deep flexor tendon. This tendon is one derivative of a proximal bifurcation of the deep flexor; therefore, as the tendon moves to flex the unaffected digit, inevitably tension must be placed on the fracture fragment causing movement. Therefore, blocking the unaffected digit while providing relief from pressure will not inhibit movement. Forced flexion of the affected digit does appear to accelerate healing.

The method used for forced flexion fixation is as follows: With the block affixed to the sound claw, two 2 mm holes are drilled in the bearing edge of the dorsal wall of the affected digit. Corresponding holes are drilled through the middle of the block. A wire is bent into a U and each branch is threaded from the bearing surface through the openings on the dorsal walls. The two branches are then bent over the dorsal wall and passed through the holes in the block. The digit is forced into a flexed position, the wires pulled tight and crimped. A technovit bridge can be created to reinforce the function of the stabilizing wire and the tip of the sole of the affected claw trimmed as necessary to level it with the bearing surface of the block.

Avulsion of Horn. Horn can be lost due to excessive wear or because of some violent mechanical trauma which may break away part of the hoof or cause the loss of the entire horny shell. No artificial substitute can equal the effectiveness of a natural hoof and so long as extraneous matter has not been forced between the sensitive laminae and the horn that is in the process of avulsion, surgical intervention to remove loose horn is not recommended. In some cases it may be desirable to apply a plaster or fibre glass cast to prevent the process of avulsion from progressing.

In all cases acrylics may be applied directly to sensitive laminae without any risk of tissue damage.

At first examination thorough cleansing is necessary, and if foreign matter is trapped under the horn and it cannot be removed, then the horn should be excised to permit proper cleansing. Exposure of the distal phalanx or traumatic loss of part of the bone does not preclude the possibility of recovery. The regeneration abilities of the digital corium appear to be considerable. After cleansing, the affected area should be dressed with a non-greasy, non-irritant bacteriostatic agent and protected by gauze, cotton batting, bandage, a plastic sheet, and finally an adhesive elastic binding. Depending on the temperament of the animal and the conditions, the wound should be left for up to four days.

At the end of this period the dressing would be removed and hopefully the process of granulation would have commenced. Viable stratum germination will

in an extremely short period produce a thin opalescent layer of horn which will ultimately contract around and over areas where this vital layer has been destroyed.

Where only small areas of horn have been lost or where the most distal region of the toe has been fractured and evulsed, there is little or no problem in effecting a repair with acrylic. Total avulsion of a hoof with loss of corium and damage of the distal phalanx is the most extreme phenomenon of avulsion that may be presented and indeed treated with some (guarded) expectancy of success.

In the case of a small horn breech no problem has been encountered in placing "technovit" directly into viable corium. Probably the majority of such cases will involve the loss of apex of the hoof and possibly the exposure of the distal phalanx. In these cases an acrylic prosthesis may be moulded over the dorsal wall and reflected under the sole. Provided that an initial period is permitted for reestablishment of the vascular corium and provided that a light dressing of a product such as furazolidone powder is applied, the chance of uneventful recovery is likely.

The loss of horn from the sole can be equally unproblematical. It is unwise, to simply plug a hole or cover a denuded area because there is a risk that the acrylic plug would become a pressure point and damage to the corium would result. Whenever the sole is involved, a plastic shoulder must be formed over the abaxial wall and extended towards the region of the bulb. Weight bearing is then carried away from the vulnerable areas of the sole.

The formation of a prosthetic hoof in cases of total avulsion are more problematical and must depend to some extent on the ingenuity of the individual attempting to salvage a seriously damaged digit. It should be remembered that the capability of the corium to regenerate horn is quite remarkable and that the objective of treatment must be to protect these tissues completely from further damage. Cleansing, a two- or three-day recovery period, and a bacteriostatic dressing are all implicit in the treatment. The basis for the prosthesis must be the sound claw which should be completely enveloped in acrylic. From this base a bridge would be created beneath the damaged claw. This bridge may be reinforced with wire or stainless steel pins. At this point the exposed corium must be dressed with a product that is compressible. A pad composed of several layers of gauze between which petroleum jelly or furazolidone paste would serve. A thick plastic bag would be placed over the digit, and then covered with a layer of foam rubber or plastic. The objective in this case is to provide a nonabrasive yet compressible core over which the protective acrylic may be moulded. When the acrylic is in place an evaluation of the degree of lateral movement present should be made. If it is considered desirable, the pastern may be bandaged and the acrylic may be moulded over the top.

Discussion

The innovative use of acrylics and other plastics has not been adequately explored as an adjunct to treating digital conditions. There is no doubt that these materials are extremely adaptable to many situations involving damage to horn. The suggestions outlined in this paper are capable of considerable refinement. The digital tissues are capable of remarkable regenerative activity, and the sensible use of acrylic prosthetics can facilitate the healing process to a considerable extent.

Bibliography

Becker E. (1956). A mechanical device for the trimming of hooves in horses and cattle. Berl. Munch Tierarztl. Wischr.; 69:353.

Becker E. (1961). Trials with resins on equine feet. Berl. Munch Tierarztl. Wsch., 74:123.

Becker E. (1974). Broken beak in the African Hornbilled Raven. Der. Prakt. Tierarztl., 55:942.

Block B. and Hastings G.W. (1972). Plastic Materials in Surgery, 2nd Edition, C.C. Thomas, Illinois.

Brewer P. (1963). Technovit and the shoeing of cattle. Tierartzl. Umsch., 18:543.

DeMoor A. and Bouchaert J.H. (1962). Technovit, a means of fixing cattle shoes. Vlaamis Dierg Tijd. 31:283.

Dietz O. and Knutze A. (1959). Observations on operative procedures for bone repair (percutaneous bone repair with extracutaneous plastics and intramedullary screws). Berl. Munch Tierarztl. Wschr. 72:395.

Evans L.H., Jenny J. and Raker C.W. (1966). The repair of hoof cracks with acrylic. JAVMA 148:355.

Funk K.A. (1970). Common conditions of the cow's foot. Mod. Vet. Pract., 46.

Hertsch P. (1972). The diagnosis and treatment of fractures of the pedal bone. Dtsch. Tierarztl. Wschr., 79, 517.

Hutchins D.R. (1969). Acrylics in hoof repair in the horse, Aust. Vet. Jnl. 45:159.

Jenny J., Evans L.H. and Raker C.W. (1965). Hoof Repairs with Plastics. JAVMA, 147:1340.

Koch K. (1965). Forms of and indications for cattle shoes. Mh. Vet. Med. 20:1.

Kurzweg W. (1965). Piacryl-Sh in the treatment of bovine digital disease. Mh. Vet. Med., 20:81.

Sonnischen H.V. (1964). Metacarpal fracture in cattle: Surgical treatment by percutaneous transfixation. Nord. Vet. Med., 16, Suppl. 1, p. 314.

Thomann H. (1963). Treatment of bovine foot disease in practice. Schw. Arch. Tierarztl. 5:246.

Vaughan L.C. and Osman M.A.R. (1967). Fracture of the third phalanx in cattle. Vet. Rec., 80:537.

Wiersma J.M. (1965). Treatment of lameness in the cow by using "Vetoplast" and wood prostheses. Tijdschr. Diergeneesk, 90:276.

LAMINITIS IN REVIEW

The term laminitis has by common usage come to designate an aseptic PODERMATIS or inflammation of the corium of the laminar region of the extremity of ungulates. However, although the laminar region may be most susceptible to insult other regions, particularly related to the sole, can and frequently are involved. It must also be appreciated that while the majority of cases of classical laminitis may be considered associated with nutrition, it might be more useful to employ the term toxic pododermatitis because different elements including those derived from food sources bring about pathological changes indistinguishable one from the other. Trauma either

alone or as an exacerbating factor in toxic podermatitis may produce lesions or clinical signs that can be difficult for the clinician to interpret. Localized infection can cause inflammatory reaction in the corium but these events are usually identified by the clinician even if strictly speaking they were to be classified as septic pododermatitis. In this paper we shall only be considering toxic and traumatic podermatitis.

Etiology and Pathogenesis

There is so much conflicting evidence regarding the pathogenesis and treatment of laminitis that one is forced to conclude that significantly more study is needed. It is sometimes difficult to differentiate opinion and traditional practice from scientific fact. Even some of the scientific data can be questioned. To attempt to reationalize such confusing information is clearly dangerous particularly when one is forced to make certain assumptions. The first of the assumptions made here is that the pathogenesis and histopathology of laminitis is comparable in both cattle and horses. This being the case, information can be derived from a wide range of opinion and research. However, it must be born in mind that there are structural differences in the laminae between the two species and that there are differences in systems of management and also in temperament.

Garner (1980) suggests a chain of events to explain the pathogenesis of alimentary laminitis which is based on a high carbohydrate intake which permits lactic acid producing bacteria to florish creating an intestinal environment that lyses the cell walls of resident gram negative organisms releasing an endotoxin which has a vasoactive component. While this may prove to be the most

common pattern of pathogenesis, toxins may also be released by other mechanisms such as would be the case in postparturient infection. "Grass founder" which is common among fat and unfit ponies that overeat on lush grass in spring and fall may have a toxic etiology although some workers claim that estrogens in the grass have a triggering effect.

In many cases of laminitis there will be a traumatic component to the etiology. Standing for long periods during transportation, overweight combined with hard walking surfaces are factors observed to precipitate laminitis in susceptible animals.

Nilsson (1963) has demonstrated in cattle certain histological changes that occur in the dermal laminae, such as edema, congestion, hyaline thrombosis and a partial or complete disappearance of onychogenic substance in the stratum germinativum and stratum spinosum.

Laminitis occurs most commonly in horses (Bell et al 1979, Coffman et al 1980, Garner 1975, Obel 1948, Colles and Jeffcott 1977) and less commonly in cattle (Dewes 1979, Weaver 1979, Maclean 1971 and Dougherty et al 1975). The disease has been observed in pigs (Maclean 1968) and sheep (Morrow et al 1973).

Clinical Findings

The disease can occur at four levels of severity: acute, subacute, subclinical and chronic.

a) Acute Laminitis. Usually of sudden onset, the clinical signs are mostly associated with severe pain. The animal may show signs of anxiety, it may sweat and the respiratory rate may increase to beyond 80 per minute. It will be anorexic, probably with an elevated temperature and with injection of visible mucous membranes. In the majority of cases, there will be a marked

digital pulse with engorgement of the superficial veins being observed. The temperature of the hoof wall will feel warmer than normal and uniform tenderness can be detected when pressure is applied to the feet. Edema and tenderness of the coronary region is often present. The animal may resist standing or moving. When the animal does stand the hind limbs will be drawn under the body and the forefeet thrust forward. The condition can affect all four feet or only the forefeet, the hind feet alone are less frequently involved. Risk of rotation of the distal phalanx is high.

This level of severity occurs more frequently in horses than cattle and must be regarded as an emergency because if treatment is not initiated within twelve hours it is extremely likely that irreversable changes will occur.

b) Subacute Laminitis. At this level of severity the clinical signs are similar to but less marked than the acute form. The nature of the lameness may be so indefinite that it may be necessary to apply a nerve block to confirm that lameness is in the foot.

Colles and Jefcott (1977) state that subacute laminitis in the horse is particularly prone to reoccurrence. Evidence suggests that both the acute and subacute forms of the disease can progress to the chronic form.

Subacute laminitis occurs with more or less equal frequency in both cattle and horses.

c) Subclinical Laminitis. In recent years this level of severity has been increasingly recognized in cattle and although not described in horses some of the changes seen in the hooves of cattle have been observed by Adams (1977).

Changes in posture or locomotion are not usually observed. The significant indication of the disease is a softening of the horn of the sole, a marginal change in colour of the horn from white to a yellowish tinge and the presence of

small diffuse areas of hemorrhage. In some instances it will be possible to observe erythema and edema of the skin above the coronet of the heel bulbs. This form of laminitis may be influenced by prolonged rather than a sudden diet of a high level of concentrate feeding.

Until recent years these changes in the appearance of the hoof have been regarded as of traumatic origin and while trauma may be an important component of the pathogenesis of this level of severity, the condition has been observed consistently under good environmental conditions. Dutch workers (Raven 1980) consider that subclinical laminitis is a highly important entity in cattle because the softness of the horn permits bacterial erosion. This unusual loss of horn causes an abnormal distribution of the pressures of weight bearing and is associated with localized aseptic inflammatory reaction in diffuse areas of the dermis.

d) Chronic Laminitis. Chronic laminitis is characterized by a slow change in the morphology of the hoof probably precipitated by vascular changes in the hoof (Acerman et al 1975). The phenomena is quite comparable in both horses and cattle. The dorsal convexity of the hoof is lost as it flattens and becomes broader. The toe elongates and the dorsal aspect frequently becomes parallel to the ground. The rougae (ridges) that run parallel to the coronet become very pronounced. The sole looses its concavity and in some instances will be seen to be convex. Chronic laminitis is regarded as being the sequel to one of the more severe levels of laminitis.

Treatment of Acute and Subacute Laminitis

The rationale for treating laminitis is variable as is the success and at this time it is impossible to recommend any one line of therapy that can assure recovery. Nevertheless, contemporary studies of the pathogenesis can help to

identify the most profitable line of approach.

1. Control the cause. If the laminitis is of alimentary origin the administration of mineral oil over six hours is indicated both to remove toxic substances from the gut and to block their absorption by the intestinal wall. If acidosis is marked the possible use of electrolytes containing 40 gms of sodium bicarbonate might be considered (Preuglehof 1960). Salt purgatives would be contraindicated because of the associated fluid loss. Adams (1977) suggests that a peristaltic stimulant may be of value in accelerating the beneficial action of the mineral oil.

If the condition is associated with bacterial infection as would be the case in postparturient complications, appropriate treatment should be given.

2. Control vascular disorders.

a) Hypertension. Nilssen (1963) provides evidence that the blood pressure is normal in cattle affected with laminitis but by contrast Robinson (1976) was able to confirm an elevation of blood pressure even in the anesthetized horse. To some extent this discrepancy may be explained by the difference in the temperament in the two species. Also, the level of severity may be greater and consequently more painful in the horse.

It is suggested, therefore, that when an affected animal has an elevated pulse and respiratory rate or any other indication of pain it should be assumed that the animal will be hypertensive and appropriate control measures implemented.

The most immediate of these measures would be to control pain in the feet by administering an analgesic such as phenylbutazone. Alternatively, the animal may be given a tranquilizing agent together with temporary digital anesthesia. The use of diuretics may be useful in the early vasodilation phase but contraindicated

beyond 48 hours when the risk of thrombosis of the laminar vessels would be increasing at which time hemoconcentration would be undesirable. In the longer term the removal of salt sources and the addition of 30 gms of potassium to the diet daily may be beneficial.

3. Control of laminar change. The observations of different workers regarding the histopathological changes occurring in the laminae may appear contradictory. The probability exists that each histological finding may be valid but be observed at different phases of the disease with the time of its appearance depending on the severity and type of insult.

Most tissues will respond to trauma or a toxic insult by an immediate vasoconstriction and vasoconstriction is recorded as being associated with laminitis. Vasodilation has also been observed and probably occurs after the initial insult and persists for some time. The pressure of the vasodilation phase probably accounts for the clinical signs of heat in the hoof and pain and swelling of the coronary band. Most clinicians will encounter laminitis in the vasodilation phase in which cold hydrotherapy would be indicated.

During vasodilation the extravasation of serum may occur which would account for the yellowish tinge that is sometimes observed in the horn. Diapedetic hemorrhages are a common finding. Extravasated serum pools between the stratum germanitivum and the innermost layers of the horn. This phenomenon is readily observed in cattle in which the avulsion of horn occurs quite frequently and presumably would account for the detachment of the laminae from the lamellae commonly seen in the horse.

Another observation is the presence of hyaline thrombi in the capillaries of the dermis which has been demonstrated to be concurrent with the loss of onychogenic

substance from the stratum germanitivum and stratum spinosum. This would account for the long term softening of the horn (unless vasodilation is controlled at an early stage).

Once the thrombi have become extensively established it can be reasonably assumed that areas of ischaemia will be present and that eventually arteriovenous shunting will take place. We must presume that the latter changes are irreversible and that once they have taken place a return to normal laminar function will not occur.

The foregoing sequence of events is a theoretical one based on the logical use of the data available. Even if the theory is correct it is not possible at this time to clinically evaluate the stage to which the disease process may have progressed nor do we have a useful guide to the rate at which the pathological changes will occur.

In the unlikely event that the clinician is attending an animal so early in the disease that there is no perceptible heat in the hooves it should be assumed that vasoconstriction is present and hot water therapy at hourly intervals would be logical. Dilation of the vessels at this stage would help eliminate the toxic elements from the laminae. Conversely, if heat is present in the hoof wall and particularly if there is edema of the coronary region cold hydrotherapy at three hourly intervals should be instituted.

The use of antihistaminics has met with variable success and may be of little use beyond the first 48 hours of the onset of the disease. The use of corticosteroids during the first 36 hours of the vasodilation phase is to be recommended but prolonged therapy is definitely contraindicated because it has been observed that in the long term corticosteroid therapy actually can cause the onset of the disease.

Also in the long term 10 gm of methionine should be provided in the diet for one week followed by 5 gms daily for the subsequent three weeks. Methionine provides the necessary disulfide bond substrate which is regarded as being useful in the long term repair of the hoof/bone structural stability.

4. Control of rotation of the distal phalanx. Some authors regard forced exercise as being beneficial presumably to stimulate the circulation of the blood in the distal extremity. On the other hand it is also believed that exercise may accelerate the rotation of the distal phalanx which has been observed to occur as early as 72 hours after the onset of clinical signs.

Although it is not known with any degree of certainty, it is reasonable to assume that forced exercise in the first 48 hours will be beneficial but that during the next two or three days a "critical point" will be reached at which separation of the sensitive from the insensitive structures may occur. During this high risk period it may be preferable to resort to less vigorous forms of foot action. For example, it might be preferable during this phase to use a mud bath in which the material used would have a high degree of suction. This would have the effect of actively moving the hoof in both directions in respect to the corium. However, the success of this method will depend on the temperament of the animal and if active movement does not take place limited gentle exercise over soft surfaces should continue.

Rotation of the distal phalanx does occur in cattle but it is neither as serious or as common as it is in horses. The following observations are therefore of major importance in acute equine laminitis.

Adams (1977) among other authors recommends that the toe should be shortened and the heels lowered in order to bring pressure on the frog with the idea of

providing support to the distal surface of the distal phalanx. This is the traditional method for attempting to reverse the rotation that may be present in chronic laminitis. However, there may be cause for caution in the application of such a technique during the early phases of the disease unless the limbs are in the typical extended posture. In such a posture the tension on the deep flexor tendon and thus on the distal phalanx will be reduced. However, the successful use of analgesia and/or regional anesthesia may restore the animal to a normal upright posture in which case elevation of the heels would be required in order to relieve the tension on the deep flexor tendon. The provision of shoes with removable heel caulks would provide flexibility for treatment which then could be directly related to the current posture of the animal.

The value of the animal may in some cases warrant radiographic monitoring of the position of the distal phalanx in relation to the dorsal hoof wall. This would be carried out daily from the fourth to the tenth day. Rotation can be said to have commenced when the dorsal surface of the distal phalanx is no longer parallel to the inner surface of the hoof wall. Once rotation has commenced exercise should be discontinued immediately and depending on the posture of the animal efforts should be made to relieve tension from the deep flexor tendon. Wide webbed seated shoes with provision for the application of removable protective padding should be fitted. The soles would be examined daily for evidence that the distal phalanx is penetrating the horn distal to the tip of the frog. Hemorrhage of the white line at the toe is a particularly unfavorable sign. When rotation of the distal phalanx has been detected antibiotic coverage is indicated.

In acute laminitis should there be no sign of the abatement of clinical signs by the seventh day of the disease, the hope of a satisfactory resolution will be minimal.

The treatment of subacute laminitis should follow the same pattern as the acute level of severity with similar emphasis on rigorous treatment during the first twelve hours.

Subclinical laminitis calls for corrective hoof trimming to stimulate the growth of healthy horn together with adjustment of diet which is usually the causative agent. Soaking the hooves in five percent Formalin solution is beneficial in hardening the hooves. Many animals will accept standing in a rubberized feeding bucket.

Treatment for chronic laminitis is at best palliative. Corrective shoeing for horses is practiced with the toes being shortened and the heels lowered. One of a variety of shoes may be selected provided they give support to the frog. Jenny (1962) suggests a method which he claims will assist the correct relocation of the distal phalanx and perhaps permit the normal reunion of the stratum internum with the stratum medium of the wall. With this technique, the stratum externum and the underlying debris is completely removed by rasping. The presence of the dermis will show pink through the thin layer of stratum internum. Movement of the two remaining portions of the hoof wall should be restricted by appropriate shoeing and further reinforced by the use of an acrylic bridge formed across the intervening space. This technique has appeared to have received little attention in subsequent literature but could prove useful also if practiced in acute laminitis when rotation of the distal phalanx has occurred but after the clinical signs have completely subsided.

Chronic laminitis in cattle leads to a hoof deformity commonly referred to as "slipper foot". When this deformity is present, the useful life of the animal is limited because secondary complications are quite common. The quality of the

horn can be improved to some extent by the regular trimming of the hooves at three month intervals.

A significant body of workers consider that a genetic factor may be involved in accounting for the susceptibility of certain animals to laminitis while others appear to have greater resistance.

Bibliography

1. Ackerman N., Garner H.E., Coffman J.R., Clement J.W. (1975). Angiographic appearance of the normal equine foot and alterations in chronic laminitis JAVMA 166 58.

2. Adams O.R. (1977). Lameness in horses. Lea and Febiger, Philadelphia.

3. Colles, C.M. and Jeffcott L.B. Laminitis in the horse. Vet Rec 100 262-264. (1977).

4. Dewes H.F. (1979). Transit related lameness in a group of Jersey heifers. N.Z. Vet J. 27 45.

5. Dougherty R.W., Coburn K.S., Cook H.M. et al (1975). Preliminary study of appearance of endotoxin in circulatory system of sheep and cattle after induced grain engorgement. Am J Vet Res 36 831-832.

6. Garner H.E. (1975). Pathophysiology of equine laminitis. Proc Am Assoc Equine Pract 21 385-387.

7. Garner H.E., Coffman J.R., Hahn A.W., Ackerman N. and Johnson J.H. (1975). Equine laminitis and associated hypertension: A review. JAVMA 166 56.

8. Garner H.E. (1980). Update on equine laminitis. Vet Clin Nth Am 2 25-32.

9. Greenough P.R. (1962). Observations on some diseases of the bovine foot. Vet Rec 74 1 and 53.

10. Greenough P.R., McCallum F.J. and Weaver A.D. (1981). Lameness in cattle. Wright Bristol.

11. Maclean C.W. (1968). Acute laminitis in sows. Vet Rec 83 71-75.

12. Maclean C.W. (1971). The long term effects of laminitis in dairy cows. Vet Rec 89 34-37.

13. Morrow L.L., Tumbleson M.E., Kintner L.B. et al (1973). Laminitis in lambs injected with Lactic Acid. Am J Vet Res 34 1305-1307.

14. Obel N. (1948). Studies on the histopathology of acute laminitis. Almqvist and Wiksells, Boktryckeri AB Uppsala.

15. Robinson N.E., Scott J.B., Dabney J.M., Jones G.A. (1976). Digital vascular responses, permability in equine alimentary laminitis. Am J Vet Res 37 1171.

16. Weaver A.D. (1979). The prevention of bovine laminitis. Bovine Practitioner 15 70.

17. Bell T., Smyka P., Estry S. et al (1979). Alternation in platelet function in equine laminitis. Fed Proc 38 1411.

18. Jenny J. (1962). Mechanical treatment of laminitis. Proc 8th Ann AAEP. 212.

19. Coffman J.R., Hammond L.S., Garner H.E., Thawley D.G., Selby L.A. (1980). Haematology as an aid to prognosis of chronic laminitis. Eq Vet Jnl 12 30.

20. Prugelhof F. (1960). Uber die hufrehe des Pferdes. Wien Tieraertzl Mschr 49 589.

Laminitis

L.C. Allenstein, D.V.M.
306 Pleasant Street
Whitewater, WI 53190
414-473-2094

Laminitis is a diffuse aseptic inflammation in the corium of the foot. Some investigators believe that laminitis also involves a disruption in the normal production of the keratin in the foot horn.

CLINICAL SIGNS OF LAMINITIS

Acute Laminitis

The acute form is well recognized by the signs of painful and sensitive feet, heat in the hoof wall, increased digital pulse, a sensitive coronary band, arched back stiffness, refusal to move, evidence of hemorrhage in the corium, and malpositioning of the feet in weight bearing.

Often even acute signs go unnoticed at parturition because the cow is preoccupied with the local discomfort of the swollen udder.

Sub-acute and Chronic Forms of Laminitis

The sub-acute and chronic signs are recurrent periods of pain or changes in the feet, lameness caused by ulcerations of the sole, white-line separations with abscessation, and bruising of the sole.

CAUSES

Acute laminitis has long been recognized as an upset in metabolism, the cause of which is still uncertain. Overfeeding of a high protein or carbohydrate ration creating a lactic

acidosis often causes laminitis. Lack of exercise and concurrent diseases such as mastitis, metritis, or acetonemia can be causal factors. Exposing the feet to hard surfaces (cement) too suddenly or for too long a time may also influence the development of laminitis. Calves confined to pens with no exercise may be predisposed to laminitis in the future. The severe overgrowth of such calves' hooves has an effect on what they will be like at maturity.

When too much grain is fed, histamine is released from the lactic acid that is produced in the rumen. The histamine has been postulated to be the inciting factor in laminitis. Work by Suber et al. has shown that the increase in ruminal acid with a lowering of blood and rumen pH; the increase in ruminal histamine with dehydration; and an increase in ruminal and blood acetylhistamine accompany engorgement and results in symptoms of founder. However, there is a lack of statistical difference between blood histamine values of forage-fed cattle and those of concentrate-fed cattle, indicating that yet another factor may also be involved. Often cattle with acute laminitis have low histamine values, whereas cattle with chronic laminitis have higher levels. A recent study was done in which lactic acid was given to lambs to produce an acute laminitis syndrome within 24 hours. This study confirms that rumen acidosis arising from a sudden intake of excess concentrates with no roughage or with roughage fed much later is important in the pathogenesis of the acute laminitis syndrome. An acute or subacute catarrhal inflamation of the abomasum and intestines may also be involved

in the etiology; these conditions have been found on the post mortem of foundered individuals.

PATHOGENESIS

Acute Forms of Laminitis

Histamine is clearly involved in the pathogenesis of laminitis. Certain epidermal changes in the foot occur along with thrombosis of the blood vessels. The horny part of the foot is composed of keratin which is basically protein, a small amount of fat, and minerals with a variable amount of water. The protein fraction is made up of amino acids, and it seems that the proportions of the sulfur-containing amino acids are changed in laminitis. Researchers have shown that reduction in the proportion of cystine and methionine and a variation in the structure of the hoof lead to a physically softer and waxy horn of the foot, which in turn is associated with laminitis. Hemorrhage occurs along with edema, venous stasis, and congestion of the capillaries. There may be a disturbance in the calcium: phosphorous makeup in the horny claw. These changes occur in the acute cases.

Sub-acute and Chronic Forms of Laminitis

In sub-acute and chronic cases, deformation of the horny claw is evident. Hemorrhage and scar-like inflammation, often circular, occur in the sole where ulcers develop and in the white line of the sole and wall, leading to separations. Marked sclerosis of the vessels is seen especially in the sensitive corium of the foot. Occasionally in cattle, a deviation of the third phalanx occurs, as in the horse. Each of these conditions

is part of the pathogenesis leading to the lesions seen in the sole and in the white line of the bovine foot.

CONDITIONS SEEN IN THE FIELD

The primary cause of sole ulcers and white line separations is laminitis. These are the majority of the foot problems seen in dairy cattle today. Possibly a deficiency in vitamin A, Beta-carotene, sulfur, copper, zinc, or certain amino acids with the sulfur ion may be involved in these secondary lesions. Some investigators have shown an increased incidence of lameness in herds which are copper deficient due to a reduction in the activity of certain copper containing enzymes. This leads to an instability and decreased strength of the collagen because of a lack of cross-linkage of the polypeptide chains.

When hemorrhage occurs in the sensitive lamina due to this instability at the white-line or in the sole where the bulb of the heel joins the sole, a track is formed in the horny tissue to the outside. This often extends anteriorly in the sole as it makes its way out, forming an avenue for dirt and manure to pass to the sensitive corium. An infection results with an abscess and then an ensuing ulcer. An ulcer may also occur following hemorrhage without an abscess.

Some heifers become extremely stiff and sore when they enter the milking herd. I feel this is related to laminitis. Fighting to get up in the stanchions is common, with much trauma to their feet, legs, hock, and knees. There are some veterinarians that believe this stiffness is due to an infectious organism such as chlamydia, mycoplasma, or hemophillus in the joint capsules. Much more research is needed.

It is my experience that most of the problem is due to laminitis. The hemorrhage one will find in the soles and the white-lines will confirm this diagnosis. Also the over-growth in the ensuing months will further support one's diagnosis.

Often in a cow, the claw or toe involved with a separation will show an over-growth creating a screw toe appearance. Many times you will find no infection, only a track of dried blood from the sensitive corium to the curled under sole. All four feet will often have these tracks to some degree. They are apparent in the lateral toe of the hind feet and the medial toe of the front feet. Trimming will reveal these signs of hemorrhage. Previously I was of the opinion that all screw toes were a hereditary condition. No doubt many are when some of the phalanx bones are deformed. But I feel more are due to laminitis.

THERAPY

Acute Laminitis

Acute founder or laminitis is usually treated with antihistamines with variable results. Anti-prostaglandin drugs such as aspirin, phenylbutazone, and flunixin meglumine (Banamine) are often used to relieve the pain. Corticosteroids are also used for pain relief. Treatment of an underlying cause is imperative, e.g. the use of an antacid for the acid indigestion (mineral oil will stop absorption) and antibiotics for a metritis or mastitis.

Methonine supplementation has been used recently to aid the healing process of the degenerated collagen connective fiber of the keratin in the horny claw.

Chronic Laminitis

Chronic cases of laminitis require constant trimming. Treatment of an abscess or ulcer involves proper trimming and exposing the infected area. Be sure to get them as flat as possible with no funnel left into which dirt may pack. The same is true with white-line separations.

Medication

As to the medication to use, iodine mixed with glycerin, 1/3 : 2/3, on a cotton or gauze pack, applied to the infected area and wrapped with gauze or tape will speed the healing. The use of a sulfa-copper sulphate powder is indicated if granulation tissue is forming. Redressing the foot may be necessary at 5 day intervals if the involvement is severe. Many other medications are used, such as koppertox, pheno-formalin, furacin and sulfa ointments.

Hoof Trimming

When trimming a severely infected foot, care should be taken to keep the weight of the cow on the sound toe. In severe cases in which involvement of the third phalanx or the lower joint is likely, a plaster paris cast or toe board is often used successfully so that the weight is placed on the good claw. The involved tow is raised and immobilized by the cast. Healing is enhanced because of the lack of motion and the nonbearing of any weight. I will demonstrate this later.

Antibiotics are used successfully if there are signs of infection, usually a secondary necrophorous involvement.

PREVENTION

Several laminitis prevention pointers pertaining particularly to heifers have been suggested by Weaver and others.

1. Feed a balanced ration.
2. Control certain viral infections by vaccination, IBR; BVD.
3. Get heifers accustomed to hard surfaces several weeks before parurition.
4. Give heifers a place to rest.
5. Provide plenty of exercise for all cows and heifers before and after parturition. Do not put heifers in the stanchion and leave them there for days.
6. Use rubber mats in stalls and plenty of bedding.
7. Make no sudden changes in diet three to four weeks before and after parturition.
8. Watch acidotic rations closely. Keep the concentrate/roughage ratio in the 40:60 or 50:50 range. Do not overfeed a fermented roughage as the only feed. Use some dry hay.
9. Take up to six weeks after parturition to bring the heifer to full feed.
10. Ensure that there is an immediate source of roughage after grain is fed or feed some with the grain. Too much grain alone creates acidosis of the rumen.
11. Free access to salt often stimulates the flow of saliva which creates more sodium bicarbonate and improves the pH buffering capacity of the rumen.

12. Feed two or more feeds (roughage and a concentrate) together. Better yet, use a total mixed ration of 17 to 19% fiber.
13. Watch the overfeeding of silages and haylages. The amount of moisture, the coarseness of the cut, and the age at harvest all have an effect. Lignin is present at a lower percentage in early rough roughages.
14. Consider feeding at least one pound of long stem hay for each 100 lb of body weight.
15. If enough roughage is not available, consider using a buffering agent such as sodium bicarbonate, magnesium oxide, or socium bentonite. A combination is advantageous.
16. Use a fecal pH meter if carbohydrates are appearing in the manure due to intestinal acidosis. Consider using limestone to raise intestinal pH to near neutral.
17. Control all postpartum infections like mastitis and metritis.
18. Consider using methionine to insure the proper protein makeup of the foot.
19. Promote proper foot care with periodic trimming, use of a foot bath, and maintaining good surfaces in the yards and housing. Consider the use of lime as a bedding.
20. Allow more time to be spent on dirt or pasture.

REFERENCES

1. Eddy, R.G., Scott, C.P. Some observations on the incidence of lameness in dairy cattle in Somerset. Vet Rec, 106:140-144, 1981.

2. Nesbitt, Amstutz, Lewis. Lameness in cattle. Bovine Practitioner, 10:39-48, 1975.

3. Allenstein, L.C. Lameness in cattle. Can Vet J, 22(3):65-67, 1981.

4. Storz, J. Chlamydial infections in cattle. AVMA Convention, 1981, St. Louis.

5. Weaver, A.D. The prevention of laminitis. Bovine Practitioner, 14:70-72, 1979.

6. Troutt. Personal communication. Virginia Polytech.

7. Nilsson, S.A. Bovine laminitis. Skara, Sweden. 1978.

8. Suber, et al. Profiles in foundered cattle. Am Vet Res J, 40(7) 1005-1008, 1979.

9. Berg. Current Veterinary Therapy. p. 1103-1104.

10. Weaver, A.D. Influence of feeding on the bovine foot. Skara, Sweden, 1972.

11. Sanders, D.E. Copper deficiency in food animals. Comp Con Ed, s404-s410, Aug. 1983.