

Pathology Associated with Rations

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Introduction

After the excellent series of presentations we have just heard, it is possible that this discussion, "Pathology Associated with Rations," is mistitled and perhaps misdirected. The previous speakers have very competently directed their attention to what might be described as the "anatomy of the ration." Perhaps it would be more appropriate for us to get involved with what might be described as the "pathology of the ration." This play on words, "pathology of the ration," versus "pathology associated with the ration" may sound redundant. However, when we consider that by definition "pathology is the study of the molecular, tissue, or organismal response of the living body when exposed to injurious agents or deprivations" (20) and attempt to connect this definition with a ration, we have established a unique premise:

That a "normal" feed, "usually" fed, supposedly furnishing nutrients in proportions and amounts as to properly nourish a cow for a given 24-hour period (15), can produce a harmful effect in the cow. It may be more pertinent to look for the lesion in the ration rather than in the cow; to some extent, we'll try to do this.

When discussing this association, the ration as an agent of pathologic change in the dairy cow, there are several rather basic factors that must be reiterated:

1. The ruminant emerged approximately 60 million years ago and evolutionally developed tissue systems that were necessary to successfully colonize high-fiber, low-nitrogen environments (14)—environments high in cellulose. Hence, under a comparatively uncomplicated dietary regimen, ruminants became sophisticated biological fermentation-recycling factories; they prospered, dependent on microbiological populations for cellulose digestion and enhanced protein metabolism.

Despite this relatively long phylogenetic history, the ruminant has been domesticated for only 7000 to 8000 years, and with regard to the dairy cow, highly productive only within very recent history. In the United States, dairy cow numbers progressively increased between the years 1850 and 1940,

reaching a peak of over 24 million animals. Following this period, cow numbers have progressively declined. Despite this trend, there occurred nearly a 2,000-pound increase in average milk production between the years 1960 and 1966; this increase was approximately equal to the total improvement in production that occurred between 1906 and 1930 (13). Certainly much of this production increase can be attributed to improved breeding and feeding practices. However, during this same period of improved production, certain gastro-intestinal and metabolic disorders have increased in incidence and, with respect to feeding practices, a popular idea has evolved—that we are excessively "pushing" our dairy cows for production.

2. The diverse microbiological populations of the rumen must be adapted to chemical, physical, and quantitative alterations in the ration (substrate).

3. Rumen fermentation, ideally, should be continuous and, optimally, uniform (13). Implementation of this consideration is accomplished, in part, through frequent but uniform ration intake (substrate renewal). It has been demonstrated (14), in sheep, that when the same ration, at the same total quantity, was fed in four feedings, as opposed to one or two feedings per day, there resulted:

- A. a reduction in the range of intra-ruminal pH.
- B. a progressive reduction in the number of rumen bacteria.
- C. a progressive increase in the number of rumen protozoa.
- D. increased digestibility of the ration.
- E. an increase in the amount of protein stored.
- F. a reduction of free ammonia levels in the rumen.

4. It must also be recalled that the end products of rumen fermentation include the energy sources for the cow: the volatile fatty acids, acetic, propionic, and butyric. It can be anticipated that these acids will normally be present in rumen fluid, when rations are fed, that include large amounts of highly digestible roughage, at molar percentages of approximately 65% acetic, 20% propionic, and 15% butyric (13).

5. The lactating dairy cow is under a production burden that predisposes to energy-deficit disorders. A lactating cow may, as a consequence of lactose synthesis, lose two to four pounds of glucose daily (11). In order to maintain homeostatic mechanisms, this loss must be offset by adequate dietary intake.

6. Pathologic alterations need not be grossly discernible.

With these factors in mind, we can begin to examine some abnormal (pathologic) conditions in the dairy cow, whose incidence may be rather dramatically influenced by the composition of the ration and/or its physical structure and/or the way it is fed. These abnormal states could conceivably involve a myriad of conditions ranging from simple indigestion to torsion of the abomasum, including, as well, the metabolic-deficiency disorders. To discuss each in the detail necessary to fulfill the definition of "pathology" (20) would be excessively time-consuming. I would prefer, therefore, to select certain disorders and use these as models to illustrate, in a practical way, the possible connections between the ration and the inducement of a pathologic condition.

Rumenitis

Rumenitis has been variously referred to as toxic indigestion, rumen overload, grain overload, engorgement toxemia, rumen acidosis, and lactic acidosis. Rumenitis, however, is the preferred term (22) and the disorder can be viewed as a model of inadequate dietary adaptation. The disorder occurs as a consequence of the ingestion and the rapid intra-ruminal fermentation of unusual amounts of dietary carbohydrate (grain, etc.) with the subsequent production of excessive amounts of lactic acid. The salient pathophysiologic features following the ingestion of the carbohydrate substrate are (1,7,19,22):

A. an initial decrease in rumen pH (from near 7.0 to approximately 6.0) probably as the result of the accumulation of organic acids (acetic, formic, valeric, and succinic) other than lactic.

B. a decline in the numbers of rumen protozoa, an increase in the numbers of rumen gram positive organisms (*Streptococcus bovis*), pronounced increased production of lactic acid, and a fall in the numbers of rumen gram negative organisms.

C. an increasing concentration of intra-ruminal lactic acid, progressive decrease in pH of the rumen fluid (pH 4.5-4.8), depletion of intraruminal bicarbonate, loss of rumen protozoa, pronounced increase in the osmotic pressure of ruminal fluid and the initiation of chemical rumenitis.

D. an increase in the fluid volume of the rumen at the expense of circulating volume, and because of increasing intra-ruminal osmotic pressure; the absorption of lactic acid from the rumen; rumen histamine concentration begins to increase (and may be present for several days).

E. As the process continues and the pH of the rumen fluid continues to decline (4.5 - 4.0) the concentrations of *S. bovis* decline and are replaced by Lactobacilli species (gram positive rods) whose optimum pH is below 5.0; endotoxin is probably present; chemical rumenitis is pronounced and severe dehydration (hematocrit values may exceed 50%) and uncompensated metabolic acidosis are present.

The clinical course of the disorder is quite variable and apparently dependent on the degree of acidosis (rumenitis) present. In peracute cases death may occur within 12 to 24 hours. In affected animals that survive the acute phase of rumenitis, the course is dictated by the degree and nature of the pathologic alterations. Recovery from rumenitis will be delayed until a normal rumen flora is established. In mild cases, with prompt correction of the ration, the animal will usually begin to eat normally within a few days; however, milk production and milk fat content will frequently take several weeks to return to original levels (7).

Anatomic Pathology of Rumenitis

At necropsy the blood may appear dark and thick, suggestive of dehydration. The rumen is usually distended by fluid that has a sour odor, and rumenitis is usually located in the anterior-ventral sacs (22).

In experimentally induced rumenitis (22) histological changes were evident at 6-10 hours. The lesion began with hydropic change in the cells of and below the stratum lucidum. These cells subsequently ruptured to form microvesicles that became extensively infiltrated by neutrophils and sloughing of the epithelium occurred. The inflammatory reaction proceeded to ulceration in areas of small villi covered by thin epithelium.

In natural cases of rumenitis, once the ruminal epithelium becomes ulcerated, bacteria, principally *Fusiformis* (*Spherophorous*) *necrophorous* and fungi invade the lesions. These organisms may cause deep and extensive ulceration, a microtizing vasculitis (fungal elements can be viewed, histologically in blood vessels) and subsequent metastatic infection of other organs (liver). Severe and extensive mycotic lesions may also involve the omasum and abomasum. Frequently, the ulcerative

process extends into the abdominal cavity, producing a fibrino-hemorrhagic peritonitis.

We have presented rumenitis, perhaps too superficially, from a somewhat traditional point of view: an acute process due to the introduction of a carbohydrate substrate to a population of rumen organisms not adapted to the nature of the substrate; the progressive loss of homeostatic mechanisms so that intra-ruminal pH is uncontrolled. Thus, severe pathologic alterations occur because of an undesirable rumen fermentation process—a process that fosters the production of increasing concentration of lactic and propionic acids but depression of acetic acid elaboration.

In dairy cattle the history associated with acute rumenitis frequently demonstrates an accidental exposure to large quantities of grain: an individual cow wandering into a grain storage area and “supplementing” her ration, or cows permitted prolonged access to automatic feeders in a milking parlor. There is, however, evidence that ruminal hyperacidity of a less severe form than that responsible for acute rumenitis exist (1,13); although this acidity may not always produce apparent pathologic change, it may be responsible for certain “clinical” disorders.

Low-Fat Milk Syndrome

Dairy cattle fed rations high in carbohydrate (grain) but low in fiber (fibrous material, roughage) experience significant reductions in milk-fat secretion (6). This reduction in milk fat may be, in part, associated with marked elevations of rumen propionate (6). Acetate (and beta-hydroxybutyrate) plays a prominent role in the synthesis of milk fat by supplying carbon for the formation of C₄ to C₁₆ fatty acids (6). With decreasing pH of ruminal fluid, the concentration of acetic acid progressively declines, but the level of propionic acid increases (13,19). When cattle were fed a diet consisting of alfalfa hay plus grain, at a rate of two lbs./5.5 lbs. of milk produced, milk-fat was maintained at 4%. Under this regimen the proportions of volatile fatty acids (VFA) were acetic, 69%; propionic, 21%; and butyric, 10%. When the ration consisted of grain free choice and only five lbs. of alfalfa hay, milk fat declined to 1.8% and the proportions of VFA developed as: acetic 45%, propionic 46%, and butyric 9% (6). Hence, the benefits of increased amounts of roughage incorporated into the ration are apparent.

“Off-Feed” Syndrome

Transient anorexia (“off-feed”) is a relatively common occurrence among dairy cows fed large

quantities of grain. Although these cattle may be accustomed to a specific grain ration, daily fluctuations in amount and/or density ingested, may lower rumen pH and cause reduced or irregular appetite. It has been demonstrated (3) that even moderate depression of rumen pH (6.0) may decrease voluntary intake.

Traditionally, dairy cattle are supposedly fed twice daily but in most herds this isn’t really done. Usually, in an attempt to achieve higher levels of milk production, large quantities of grain are fed at each milking (in parlors, out of necessity, cattle must consume that amount rapidly); forage is fed later. When the quantity of grain ingested is great enough, cows may not eat the forage ration for several hours. The effect of this pattern of ingestion is the development of four distinct rumen fermentations (two for grain and two for forage) (13) with the tendency to reduce rumen acetate but to elevate propionate.

In certain cows this type of “slugging” ingestion and “batch” fermentation, perhaps, could cause loss of reticulo-rumen contractions (2) and the development of rumen atony.

Laminitis, Concrete, Lameness

Lameness, causing significant economic loss, may occur in dairy herds as a consequence of subacute/chronic alminitis and ulceration of the sole with subsequent sequelae. The relationship of lameness resulting from laminitis associated with acute rumenitis has been recognized (22). Because rumen histamine levels increase in acute rumenitis (1), released histamine was thought to cause laminitis. However, laminitis is probably a dyskeratosis (12) (abnormality of development with changes in epidermal cells [20]) caused by an allergic reaction to protein (4). Subacute/chronic laminitis, contributing to sole ulceration, has been associated with grain rations composed of nearly 50% high protein substances (5).

The optimum rumen pH range for normal proteolytic activity and protein synthesis is between 6.3 and 7.4 (13). It is possible that high concentrate rations, that have the potential to diminish rumen pH and alter VFA metabolism, could also dramatically affect rumen protein metabolism and predispose to laminitis.

In certain areas dairy cattle are maintained, for prolonged periods, on slurry-covered concrete that is frequently cracked and broken. This environment, in association with rations having a tendency to contribute to subclinical laminitis with eventual weakening of hoof horn, causes significant lameness.

Fatty Liver Syndrome

For the past several years, a syndrome has been recognized (23) in recently fresh, unusually heavy, dairy cattle. This condition is characterized clinically by severe, non-responsive ketosis, progressive weakness, occasional vague CNS signs, and death frequently associated with an infectious disease. Pathologically, the disorder is characterized by severe hepatic lipidosis (lipidosis will affect other organs as well). Cattle that have been "led and challenged" on high-grain rations including finely-chopped corn silage, are susceptible.

The following herd situation may serve to illustrate certain points concerning the fatty liver syndrome:

1. In late June and early July 1973, eight deaths occurred among recently postpartum, obese cows in a large, well-managed (average milk production of 17,000 lbs.) Holstein herd. Each cow that died had produced in excess of 17,000 lbs. of milk her previous lactation; each had marked, non-responsive ketosis.

- a. Routinely, the milking herd (210 cows) were fed a well-blended concentrate and corn silage ration three times a day. The corn silage, blended from a bunker silo, was finely chopped and the concentrate ration contained wet brewer's grain and urea (60 lbs./ton).
- b. Each morning all refusal from the previous day was removed from the feed troughs, weighed, and fed free-choice to the cows.
- c. All cattle had access to extensive pasture.

2. In late spring and early summer, pasture growth (Ladino clover and Orchard grass) was considered to be lush and the intake of the blended ration fell. The increased quantities of refusal were fed to the dry cows.

- a. Monthly average milk production and percent milk-fat progressively declined. In an attempt to enhance intake and to conserve the cost of protein, additional wet brewer's grains ("the cows really love them") were incorporated into the ration. By late June the blended ration consisted of:

corn silage, 6.9% crude protein (CP): 51.9%
 wet brewer's grain, 26% CP: 33.6%
 grain mix and urea, 24% CP: 14.5%

An analysis of this blended ration indicated approximately: crude fiber: 13.5%; crude protein: 17.0%; moisture: 56.6%; calcium: 0.5%; phosphorous: 0.5%; magnesium: 0.3%.

Intake continued to be depressed, and the excessive refusal continued to be fed to the dry cows, who were considered to be fat!

3. The owner was obviously concerned about the death losses but also complained about diminished production and ketosis in the other fresh cows and the overall declining production in the herd.

4. A diagnosis of the fatty liver syndrome was made on the basis of history, the clinical signs described and observed, physical findings in dry and fresh cows, and the gross and microscopic examination of tissues from several of the cows that died.

The actual pathogenesis of the disorder in this herd will never be known. The condition (decreased intake and reduced production) was apparently "controlled" in the milking cows by decreasing the amounts of wet brewer's grain fed and by elevating the fiber content of the ration. The dry cows were placed on a very restrictive intake of blended ration and supplemented extensively with hay.

It would be fun, although perhaps not prudent, to speculate on the possible pathologic-metabolic events:

1. In the summer, because of the additional heat, significant levels of lactic acid could have developed in the stored brewer's grains. When these were fed, this lactic acid could have lowered rumen pH, thus depressing the production of acetic acid but increasing the propionate content. The diminished rumen pH could also have contributed to a reduction in intake. To support the contention that this effect could have occurred, consider the following:

- a. rumen samples obtained by stomach tube had diminished pH values (judged to be just below pH 6.0).
- b. fat test was declining
- c. positive response to diminished feeding of wet brewer's grains.

2. When the elevated levels of refusal were fed to the dry cows, they became fat. Hence, their total intake of energy must have been in excess of all other requirements. Hepatic lipidosis was possibly initiated during the dry period and this may have displaced hepatic glycogen.

3. All of the cows that died were considered to be excellent milk factories. If initial production was sufficiently great, "reserve" supplies of glucose would have been rapidly expended to meet the demands of lactose formation. Also, these cows early in lactation were probably utilizing body reserves of fat (8). The net effect of these circumstances could have been a pronounced increase in the mobilization of free fatty acids with subsequent oxidation to acetyl coenzyme A, and

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Panel Discussion

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Question: What about the feeding of 2% baking soda to reduce acidity and raise fat content?

Dr. Troutt: I don't know. We tried it—incorporating some baking soda in boxes as well as in limited ration and I do not think it stays long enough in the rumen to act as an adequate buffer. Perhaps we will have clearance on bentonite. This may act as a greater buffer. There is no question that you can depress rumen pH with the addition of baking soda but that depression does not persist long enough to be of value. I have tried it, but I would not recommend it.

Dr. Morrison: I have had experience with baking soda and the combinations with magnesium oxide and the results are poor. Same thing can be said of sodium betonite—it takes 5% approximately to have any effect. You might as well cut back on the grain in my opinion and get the same effect. Five percent bentonite is of no nutritional value.

Question: Do you prefer a roughage, particularly a corn silage, that has a high grain content or a lower protein content?

Dr. Jarrett: Before protein supplements went as high as they are at the present time, I would certainly take the high grain yielding silage.

Question: Without including any hay in the diet for a blended total ration, is the dairyman creating more problems than he can financially solve?

Dr. Jarrett: On our total silage programs we are now recommending that cows be taken off silage in the dry period and go to a rough, permanent type pasture with a good, coarse grass or be put on a hay program during the dry period.

Question: What percentage of crude fat limits do you recommend on total ration basis (based on 100% dry matter) and what fats are more digestible—for example, cottonseed, etc.?

Dr. Bath: We really do not build in a crude fat minimum because any ration you can come up with will exceed what the cow really needs. Two percent would be adequate for crude fat. The important thing fat adds to the ration is the energy that comes from it. Energy is one of the main nutrients that is needed and you can get more energy in the ration by adding things like whole cottonseed. We very successfully feed whole cottonseed under our conditions and it is excellent feed. It adds things other than the fat content however. It does have a fairly high fiber content and it is high in energy because of the fat. It is very digestible and because of this it helps to maintain the fat test and still get the energy up for high milk production. As far as minimum fat, you do not have to worry about it. You will fulfill it with almost any normal ration.

Question: What is your opinion of the multiple mineral feeding boxes for supplying adequate amounts of minerals to dairy cows?

Dr. Morrison: This is a good question and one that is not easily answered because trace mineral deficiencies vary from one section of the country to another. We better define the question a little better because in some places we have cobalt deficiencies and in other places we are liable to run into other trace mineral deficiencies. In some places we are running into problems with selenium, zinc problems and others. My general recommendation to my clients is that we do include all mineral supplements. I think they need to be in the feed or in the herd mix that is to be fed with a specific roughage. We analyze roughages for minerals and then we calculate what is necessary. I do not approve, frankly, of feeding cows their mineral requirements on a free-choice basis except