already dry. This would be late summer or early fall for many herds. Now is the time to start thinking about preventing the mastitis and breeding trouble that reduced profits during the last lactation. This will enable the high producing cows to get off to a good profitable start at calving this year.

Such information as the calving interval, breeding efficiency from natural and artificial services, number and percentage of cows culled from the herd, reason for culling, replacement cost, cost of purchased cattle, change in inventory, and veterinary costs and return from the veterinary program should be determined. The number of cows and heifers with cystic follicles, retained placentas, calving difficulties, abortions, and those which required three or more services per conception should also be listed.

Conclusions

The procedures and recommendations for a dairy herd health plan are discussed. They are designed to have each calf born healthy and remain that way. They enable the cow to produce to her fullest inherited capability and return the greatest possible profit to her owner.

A healthy, profitable dairy herd is maintained by good breeding, feeding, management, records, and programmed preventive medicine. To make this possible, teamwork and good understanding between dairyman, extension agent, agribusiness personnel, and the veterinarian are essential.

References


Ketosis in Dairy Cattle

L. H. SCHULTZ
Department of Dairy Science, University of Wisconsin, Madison

The primary purpose of this paper is to summarize the current status of the problem of ketosis in dairy cattle, emphasizing the role of the dairyman in its control. An attempt will be made to evaluate present knowledge and make specific recommendations, recognizing that the research data available leave room for differences of opinion.

Definitions. Ketosis, or acetonemia, is a metabolic disorder in which the level of ketone bodies in the body fluids is elevated. These ketone bodies are beta-hydroxybutyric acid, acetoacetic acid, and acetone (possibly also isopropanol). This discussion will refer primarily to total ketone bodies, since the proportion of each may vary.

There is good agreement that the presence of some ketone bodies in body fluids is normal; also, that elevation of ketone bodies is a normal consequence of a situation existing when energy needs exceed energy intake and body fat reserves are mobilized. This occurs in fasting or underfeeding, as well as in high-producing cows that may not be able to eat enough to supply the energy needs for high production in early lactation.

It should also be pointed out that ketone bodies are used by many tissues. In the ketotic cow, there is no major defect in the utilization of ketone bodies, so the problem is primarily one of overproduction. The ketone bodies themselves may not necessarily cause the adverse symptoms, but they do seem to be the best practical indicator of the severity of the problem.

Incidence. Ketosis in cattle occurs in practically every area of the world where there are high-producing dairy cows. Some years ago, the incidence in the U.S. was estimated at a million cases per year, or 4% (29). A recent report from the United Kingdom (18) gave a figure of approximately 2%. Incidence in individual herds may be much higher. These figures would obviously be influenced by diagnostic criteria and the extent to which subclinical cases were included. There appear to be no breed or area restrictions on this disorder. The sheep has a similar condition called pregnancy disease, which occurs prior to lambing in ewes carrying twins or triplets. Other farm animals are little, if at all, affected.

Primary ketosis, in the author's experience,
practically always occurs during the first ten days to eight weeks after calving. Other difficulties are usually involved when problems occur outside of this period. About three weeks after calving is the most critical period. The majority of cases occur in the winter, or barn-feeding period, in northern climates. This may be due mainly to the fact that more cows calve during this period. Although cases appear on pasture, recovery has often been observed when barn-fed cows are turned out to pasture. The incidence is higher in older cows, and certain cows tend to repeat, but it also occurs at first calving (29).

**Diagnosis.** A recent publication (12) used the following diagnostic classifications for ketosis: The degree of ketosis was classified as 1) subclinical, 2) mild, 3) severe acute, or 4) chronic. The clinical type was classified as 1) nervous, 2) digestive, or 3) wasting. The etiological type was classified as 1) silage-butyrate, 2) undernutrition, 3) metabolic-hormonal, 4) indeterminable, or 5) excess protein. Classification for etiological type is the most difficult. It is the author's opinion that there is a lack of sufficient evidence to justify the excess protein classification in this country.

Scandinavian workers (7) classify their cases as underfeeding (small farm) or overfeeding (estate) ketosis, with the major problem being the latter. This division is a logical one, and it appears that the major problem in this country is also in the presumably well-fed herds.

A distinction should be made between so-called primary ketosis, where no apparent pathologic condition other than the true ketotic syndrome exists, and secondary ketosis, in which other factors are involved. The most frequent complicating ailments are metritis, retained placenta, nephritis, and hardware. Since primary ketosis is a metabolic disorder, there is no elevation of body temperature, so elevated temperature accompanying ketotic symptoms immediately implicates other factors. It is estimated that about one-third of the ketosis cases are of the secondary type.

As far as the dairyman is concerned, the first symptom of primary ketosis usually is a loss of appetite. Grain is commonly refused first, and then silage, but the cow may continue to eat some hay. There are all degrees, from small feed refusals to almost complete failure to eat. The cow appears gaunt; sometimes there will be a deprived appetite. Usually there is rumen inactivity and constipation. Most often the cow is depressed and dull, but occasionally there is the nervous type of ketosis, in which she is highly excitable. Sometimes there may be incoordination, particularly of the hindlegs. There is a distinctive acetone-like odor in the breath and fresh milk. Urine tests for acetone become positive first, followed by positive milk tests. These often precede other visible symptoms. Decreased milk production and loss of weight are obvious consequences of the reduced feed intake. Milk fat test is usually above normal. Only an occasional cow dies from ketosis, but when one does, the most obvious finding on postmortem examination is a fatty liver.

**Ketone tests in diagnosis.** The blood level of total ketone bodies is the best indication of the ketotic status of the animal. Roughly speaking, milk levels are about one-half the blood values, whereas urine levels are variable and exceed the blood values by about four times. For many years, the Rothera test, based on the formation of a pink-to-purple color when acetone or acetoacetic acid reacts with sodium nitroprusside, has been used to test urine for the diagnosis of ketosis. The urine test is useful because a negative test rules out ketosis. However, interpretation of a positive test is difficult, since not all positive cows require treatment. Use of the urine test by the dairyman may result in undue concern over the ketosis problem in his herd. The milk test represents a more conservative but accurate indication of the degree of ketosis.

Different preparations have different sensitivities. Emery (3) has developed a test sensitive to 2 mg % milk ketones. About half of 132 cows checked exceeded these values during the first 30 days after calving. The author (25) has worked with a commercial powder that starts to become positive at about 4-6 mg % total milk ketones, representing about 10-15 mg % total blood ketones. Of the 20 cows used in this study, 50% did not become positive during the first month after calving. Forty per cent had at least one positive test, but did not require treatment. Ten per cent showed markedly positive milk tests and required treatment. In another study (21), out of 42 cows positive to this milk test during the first eight weeks after calving, 22 were considered to require treatment by the herdsman or veterinarian. Kronfeld (11) has suggested use of a still less sensitive test positive only to milk levels of 10 mg % acetone or higher.

These tests do not measure betahydroxybutyric acid (BHB). This is probably desirable in milk, because BHB is used by the mammary gland for milk fat synthesis and its content in milk is rather constant at a low level of about 2 mg %, regardless of blood level (25). Milk and jugular vein blood have similar levels of the acetoacetate plus acetone fraction. The ratio of BHB to
increased gluconeogenesis from body protein with insufficient feed intake to replenish the per 100 ml. Values below 40 can be considered normal levels in ruminants are about 50 mg in blood components is a decrease in glucose. It becomes too severe. and permit corrective action before the cases become too severe.

**Blood changes.** One of the major changes in blood components is a decrease in glucose. Normal levels in ruminants are about 50 mg per 100 ml. Values below 40 can be considered subnormal. Ketotic animals may have levels as low as 25. The glucose decrease is presumably due to the large amount of glucose removed by the mammary gland to make lactose, coupled with insufficient feed intake to replenish the glucone supply. Although there is presumably increased gluconeogenesis from body protein (9), this is not sufficient to maintain a normal blood level.

A second major blood change is an increase in ketone bodies, already mentioned in relation to diagnosis. Following previous suggestions, normal levels would be considered something less than 10 mg %. Ketotic animals may have levels as high as 50. In nonketotic animals, the main source of these ketone bodies appears to be butyric acid from the rumen, with conversion to ketone bodies occurring in the rumen wall or liver.

As the condition progresses, the cow is forced to mobilize body fat, resulting in an increase in plasma free fatty acids (FFA). Normal levels are something less than 10 mg %. Ketotic animals may have levels as high as 50. They are carried in the plasma as an albumin complex. Under fed conditions, correlations between glucose and FFA or ketones and FFA are low (1, 22). However, under fasting conditions, or in cows positive to the milk test for ketosis, there is a significant negative correlation between glucose and FFA as well as between glucose and ketones, with a significant positive correlation between FFA and ketones (21). It seems clear that under fasting or ketotic conditions, FFA become an important source of ketones. This conversion takes place mainly in the liver. The negative correlation between blood glucose and both FFA and ketones in early ketosis suggests that the low availability of glucose is an important factor in the development of the condition. There is evidence in goats that a glucose-drain condition imposed by phlorizin causes increased conversion of butyric acid to ketone bodies in the rumen wall, as well as increased mobilization of FFA, with their subsequent conversion to ketone bodies in the liver (16). There is also evidence that the mammary gland produces acetoacetate under ketotic conditions (11), but the origin is not clear.

The significance of a fourth change in the blood of cows in early ketosis, a small but significant decrease in triglycerides (TG), is not apparent. Levels in cows positive to the milk test were about 15 mg % compared to 20 mg % when negative (21). High FFA levels are generally considered to result in an increase rather than a decrease in TG (30).

**Rumen changes.** The fact that, of the farm animals, ketosis is a practical problem only in ruminants has led to considerable research regarding the role of the rumen and rumen metabolites in the condition. Administration of the individual volatile fatty acids (VFA) produced in the rumen (26) suggests that rumen acetate is of only minor importance as a ketogenic agent. Oral sodium acetate may even have some beneficial effect for treatment because of a glucose-sparing action. Propionate is definitely glucogenic and a major source of blood glucose for the ruminant. Low blood sugar levels in the ruminant, compared to non-ruminants, is usually attributed to reliance on production and absorption of propionate from the rumen and conversion to glucose in the liver, rather than direct absorption of glucose from the digestive tract. Butyric acid is definitely ketogenic, with major conversion to ketone bodies in the rumen wall. Administration of butyric acid usually results in an initial increase in blood glucose, followed by levels below normal. The mechanism involved here is not well understood. Initial stimulation of gluconeogenesis from other sources, followed by increased insulin secretion, may be involved. There is reasonable agreement that butyrate is not glucogenic and its administration does not result in a net gain in glucose, even though some of the label from labelled butyrate may appear in glucose. Of the other minor acids, valeric is glucogenic and isovaleric is ketogenic.

Measurements of levels and proportions of volatile fatty acids in the rumen of cows in the early stages of ketosis have shown no significant
changes from normal (21). In a more advanced stage, the total concentration of VFA is decreased, with the percentage of acetate increased and propionate decreased. The same results can be obtained by fasting, so it appears that these changes are due to a reduced feed intake at the time of ketosis, and cannot be considered a causative factor of ketosis.

Certain types of rations may alter rumen VFA production, but the importance of this in the development of ketosis is not clear. High-grain rations tend to increase the proportion of propionate and decrease the proportion of acetate. Hay-crop silages compared to hay tend to do the same thing, but the magnitude of the change is not great. High-moisture hay-crop silage tends to be high in butyric acid, which would be metabolized in a ketogenic fashion and would be undesirable from the standpoint of ketosis. The predominant acid in high-quality corn or hay-crop silage is lactic. The rather confusing picture regarding lactate metabolism in relation to ketosis seems to become clearer in view of recent in vitro studies (5). These studies suggest that the rumen metabolites formed after lactate feeding depend upon the previous feeding regime as well as the rumen pH. When lactate was absent from the ration of animals from which inoculum was obtained, there was rapid formation of rather large amounts of butyric acid in flasks containing lactate. This resulted primarily from stimulation of the conversion of acetate to butyrate, rather than conversion of lactate to butyrate. Reduced rumen pH accentuated the condition. About seven times as much butyrate as propionate was produced. Inoculum from cows fed corn silage, on the other hand, resulted in the formation of about equimolar quantities of butyric acid in flasks containing lactate. Administration of propionate-butyrate mixtures to normal goats (31) suggested that propionate successfully prevented accumulation of blood ketone bodies from butyrate when present in equimolar amounts.

The conclusion which seems warranted is that lactate administered to cows with no lactate in the previous ration may result in large temporary increases in butyric acid and subsequent increases in ketone bodies. Milk tests for ketosis in fistulated cows can be made positive by this procedure. On the other hand, in administration of lactate to cows on a previous ration containing lactate, increased propionate may balance increased butyrate production. Thus, lactate feeding (20) or corn silage in the ration over a period of time would have neither a beneficial nor a detrimental effect from the standpoint of ketosis.

It should be pointed out that, unless administered in large amounts as soluble salts, the lactate produced in or added to the rumen does not appear to be absorbed as lactate. It is rapidly metabolized in the rumen, normally returning to negligible levels within four hours after feeding.

From the standpoint of ketosis, an ideal ration might be one that would produce high levels of propionate and acetate with low levels of butyrate. Attempts to find practical rations to accomplish this have been unsuccessful. High-grain rations tend to increase propionate, but acetate is reduced, with resultant depression of milk fat test. These rations would theoretically be helpful from the standpoint of ketosis, however, since butyrate is not changed greatly.

Predisposing factors. Much difference of opinion exists on the major cause(s) of ketosis. A number of possible predisposing factors will be discussed, with full recognition that there is a lack of controlled research in dairy cows to support many of the ideas.

1. Glucose drain for lactose production. There is reasonable agreement that this is an important factor, with estimates that over 1 kg of glucose per day may be needed for lactose synthesis by high-producing cows (29). The author considers this to be the primary factor involved in initiating the problem. This is supported by the fact that the problem is limited primarily to high producers in peak lactation. Attempts most successful in producing experimental ketosis are those involving glucose-drain conditions such as phlorizin administration, which causes loss of glucose in the urine (16). Not all high-producing cows get ketosis, however, so obviously this is not the only factor involved.

2. Endocrine disorders. Shaw (29) has suggested that there is exhaustion of the pituitary and adrenal cortex, resulting in insufficient ACTH and glucocorticoids to respond properly to the stress of high production. This is supported by histological studies of the two glands and response to treatment with glucocorticoids or ACTH. Satisfactory response to other therapy and failure to find low blood levels of glucocorticoids in ketotic cows argue against this disorder as the sole cause.

Thyroxin has been implicated both as a successful treatment as well as a causative factor in ketosis. Treatment data are inconclusive. Recent work on the induction of ketosis with high-protein rations and thyroxin injection (6) appears to represent an unphysiological situation. Large amounts of thyroxin were injected. Marked symptoms resulted in five days. The

J. DAIRY SCIENCE VOL. 51, NO. 7
blood ketones were exceptionally high, but the glucose was within normal ranges. Fatty livers were present. In a Michigan study (4), implants of tri-iodothyronine at calving time increased either the severity or the incidence of ketosis symptoms. These two studies would suggest that excess thyroxin is undesirable.

Growth hormone is a potent mobilizer of FFA, and results in increased blood ketones (10). Its role in the development of ketosis is uncertain.

3. Excess condition at calving. This suggestion is based on theoretical considerations. The reasoning is that excess fat results in greater and more prolonged mobilization of fat after calving and a greater chance for the accumulation of fat in the liver, with possible increases in the rate of ketogenesis (30). Livers from ewes with experimental pregnancy toxemia formed ketone bodies at ten times the normal rate (17). Lipid contents of the liver and rates of ketone body formation were directly related to plasma FFA concentration, and thus dependent upon fat mobilization.

5. Hepatic dysfunction. There is evidence of abnormal liver function in ketotic cows. This may be related to the fatty infiltration previously mentioned. Normal fat content of the liver is considered to be less than 10% on a wet basis. The author has found levels as high as 30% (68% on a dry basis) in an advanced case of ketosis. The development of fatty livers is presumably related to the balance between formation of lipid in the liver from FFA and its release as lipoprotein. The observation of elevated FFA but reduced TG in the blood of cows in early ketosis (21) might suggest decreased release of TG, although the liver lipid content of these cows was not determined. More research needs to be done regarding the possible relationship of development of ketosis to the accumulation of fat in the liver.

5. Inadequate energy intake after calving. The reasoning here is simply that an inadequate energy intake forces the high-producing cow to mobilize excessive amounts of body fat, with the undesirable effects previously mentioned. There is a tendency for some dairymen to reduce feed after calving, because of unfounded worries regarding udder edema and mastitis. It is often impossible for a high-producing cow to consume enough of a practical ration to meet energy needs; in these cases, subclinical ketosis may be unavoidable.

6. Deficiencies or excesses of protein. Obviously, a gross protein deficiency in the ration would be undesirable from many standpoints. The fact that one of the characteristics of protein malnutrition in humans is a fatty liver makes it tempting to relate protein or specific amino acid deficiencies to the ketosis problem in cows. One suggestion in humans is that there is impaired lipoprotein synthesis, which interferes with release of lipids from the liver. Methionine tends to prevent certain types of fatty livers. However, Shaw (28) used methionine for treatment of ketosis in cows without success. The fact also remains that there is a lack of evidence that protein malnutrition is involved in the problem in the field. It is well established that rumen organisms can synthesize essential amino acids. Most dairymen with a ketosis problem appear to have adequate total protein equivalent in the ration to meet requirements when the ration is consumed at normal levels. Evidence that excess protein under U.S. feeding conditions can create difficulties is lacking. Swedish workers (7) suggest that protein-poor rations fed during the dry period are often shifted quickly to protein-rich rations after freshening. Under these conditions, “abnormal, often toxic, protein decomposition products may be formed in the rumen and absorbed. The liver, already greatly overtaxed, can be injured when forced to detoxify them.” Russian workers (14) report a syndrome resembling ketosis when cows are fed either too little or too much protein. They indicate that the main cause is a disturbance of protein metabolism, so it might more properly be named protein auto-intoxication. Hibbitt (6) suggested that protein level of the grain was important in the development of experimental ketosis following thyroxin administration. When the grain contained 31% crude protein, seven out of ten cows developed ketosis, while in another group of five cows on a 20% protein mix, only one case appeared. It is suggested that this may be due to the extra ketogenic amino acids supplied by the high protein.

7. Mineral or vitamin deficiencies. There is a lack of clear-cut evidence for a direct involvement of mineral or vitamin deficiencies in the development of primary ketosis. It is obvious that a deficiency of cobalt and the possible subsequent deficiency of vitamin B12 would likely be detrimental. The depressed appetite and the specific involvement of B12 in the conversion of propionate to succinate on its way to glucose argue strongly for adequate cobalt. But most dairymen with ketosis problems are feeding adequate cobalt. Responses of ketotic cows to propionate administration with increases in glucose also argue against a specific metabolic defect here.

It has recently been observed in other species
that nicotinic acid has the property of reducing FFA mobilization and blood ketone levels. The
author and associates (27) have observed the same phenomenon in cases of experimental ketosis in goats and subclinical ketosis in a cow. It is unlikely that any deficiency is involved, because the doses used are considerably above any therapeutic levels used to treat vitamin deficiency. The mechanism involved in this action is also unclear at this point. Some workers have suggested an effect through alteration of tissue levels of cyclic AMP. Kronfeld and Raggi (13) found lower-than-normal levels of nicotinamide coenzymes in the mammary gland of ketotic cows, and have suggested that a shortage of these enzymes may interfere with the operation of the citric acid cycle. Whether these observations of lowered FFA and ketone levels following oral nicotinic acid will have any practical value in ketosis therapy in cows remains to be seen. The effects are very temporary and the mechanism unknown. Shaw (28) tried B vitamins, including nicotinic acid at much lower levels, for therapy a number of years ago without success.

8. High intake of ketogenic materials. It seems likely from previous discussion that a high intake of butyric acid from silage would be undesirable. In a Wisconsin experiment comparing high- and low-moisture grass silage, cows on the high-moisture silage had 22 mg/100 ml blood ketones and 42 mg/100 ml blood sugar, compared to 4.5 and 53 on the low-moisture material. Butyric acid content of the wet silage was 3.7%, compared to .05% for the drier material. No clinical ketosis was observed, but the cows were beyond the ketosis-susceptible period of lactation.

Available evidence suggests that lactic acid in silage or from other sources should not be considered a predisposing factor, particularly when the animal previously had been fed a ration containing lactic acid.

Although protein contains ketogenic amino acids, it also contains glucogenic amino acids. It seems unlikely on usual dairy rations that an excess of ketogenic amino acids would be an important predisposing factor in ketosis.

Normal dairy rations are low in fat. Addition of fat to dairy rations is not common. It seems logical to conclude that under normal conditions, the fat content of the ration is an unimportant factor in ketosis. Theoretically, addition of fat to the ration of a ketosis-susceptible cow, although supplying a concentrated source of energy and a glucogenic material in the glycerol, would seem to place an additional load on lipid metabolism. Presumably, it would, after hydrolysis and re-esterification, be absorbed primarily through the lymphatic system. The adipose tissue would not be depositing fat, so some of it would likely be deposited in the liver as triglyceride or be converted to ketone bodies, either of which would be undesirable.

Metabolic changes. A number of metabolic changes occur in the ketotic cow (19). They include increased fat mobilization and reduced lipogenesis, presumably resulting in overproduction of Acetyl-CoA. It has also been suggested that there is increased gluconeogenesis with reduced oxalacetate, limiting the oxidation of Acetyl-CoA through the citric acid cycle and diverting it to ketone bodies (9). There is evidence (16) that the increased level of ketone bodies eventually exerts a feedback effect that reduces fat mobilization and prevents this process from getting completely out of hand. The mechanism involved is not clear.

With the cow off feed, one might also expect a reduction in the availability of some of the precursors, such as the B-vitamins, of co-factors involved in intermediary metabolism. Various research workers have suggested specific co-factor or metabolite deficiencies as the major defect in ketosis. It is difficult to establish whether they are a cause or a consequence of the ketosis.

Treatment. At least two-dozen treatments for ketosis have been suggested (12). Because of the well-known phenomenon of spontaneous recovery (29) and the lack of blood analyses in some cases, much of the treatment data is difficult to evaluate. Only brief comments regarding treatment will be made here because the veterinarian, rather than the dairyman, should play the predominant role.

Most of the accepted treatments attempt in some manner to increase blood glucose levels. The following are the most commonly used:

1) Intravenous glucose injections. Usually about 500 ml of a 50% glucose solution is used. This is the most rapid way of supplying an exogenous source of glucose. It has the disadvantage that the glucose is used up rather readily, with blood glucose falling below normal levels within two hours (29). Some may spill over into the urine and be lost. Relapses are frequent when glucose is used as the sole treatment. Slow, continuous intravenous infusion of glucose represents a rather ideal type of therapy, but is too cumbersome to be practical under field conditions.

2) Hormone treatment. Glucocorticoid injection introduced by Shaw (29) has become a
The major beneficial effect appears to be due to the fact that it increases blood glucose, primarily through stimulation of gluconeogenesis from amino acids. The effect lasts several days. It also causes decreased glucose utilization peripherally in muscle and adipose tissue. Initially, this may cause an undesirable mobilization of body fat and increased ketone body formation, particularly if the supply of insulin is low. The major disadvantages appear to be the possibility of upsetting the hormonal balance and the fact that the glucose is being formed at the expense of body protein. A recent report (12) suggests there may be some advantage in using glucocorticoids plus insulin. The reasoning here is that this would insure adequate insulin, which would tend to prevent the decreased peripheral uptake of glucose and thus prevent the undesirable mobilization of body fat and resultant ketone body formation. Adrenocorticotropic hormone (ACTH) is also used for treatment, functioning in an indirect manner by stimulating glucocorticoid output of the adrenal cortex.

3. Oral glucogenic materials. The two materials on which the most information is available are propylene glycol (8, 13) and sodium propionate (23, 31). The usual dose of these materials is one-half to one pound per day. It is best to divide the dose into two administrations per day. The usual treatment period is five to ten days. Because the cow with ketosis is not eating well, it is usually necessary to give the treatments by drench. Some danger of inhalation pneumonia, of course, accompanies drenching. In the earlier stages, they may be consumed in the feed at lower levels. Propylene glycol is a three-carbon compound that appears to be absorbed without alteration, with a rumen disappearance half time of about one hour (3). Loss from the body is minor. It is definitely glucogenic and presumably is converted to glucose in the liver via pyruvate and oxalacetate (2). Propionate is a normal product of rumen fermentation also absorbed from the rumen and converted to glucose in the liver.

The advantage of these compounds is that an exogenous source of glucose is being supplied in a gradual manner. Disadvantages are the necessity for frequent drenching and the long treatment period. The sodium in the sodium propionate is not useful to the cow, and levels above 1 lb per day may upset the electrolyte balance. Similar high levels of propylene glycol may result in incoordination and scouring.

Sugar or molasses fed or given as a drench is not particularly effective because it is not absorbed as glucose, but is metabolized to fatty acids in the rumen. Lactates are also acted upon in the rumen with somewhat unpredictable results.

It is difficult to conclude that any one treatment is best. Each type of treatment has its place in individual situations and none is 100% effective. Often a combination is desirable.

Prevention. We are not familiar with any set of recommendations that would guarantee 100% freedom from ketosis. However, based on our present state of knowledge, a number of suggestions can be made. Most of them are not based on controlled research, which is very difficult in this preventive area, but simply stress certain desirable feeding and management practices. In general, they are based on attempts to balance feed intake with milk production so blood glucose level is maintained, along with minimizing the ketogenic materials the cow is forced to handle.

1) Do not have cows excessively fat at calving time. They have to be in good condition for maximum production, but excess fat means potentially more fat mobilization and ketone-body formation.

2) Increase the level of concentrates rapidly after calving. Starting in the latter part of the dry period appears to be a good idea. The rate of increase and the ultimate level depend upon the individual cow, the type of roughage, and economic considerations. Common sense needs to be used to prevent the cow from going off feed. And it should be recognized that some high-producing cows will not eat enough to prevent drawing on body reserves.

3) Do not make abrupt changes in the ration during the ketosis-susceptible period. Normal changes in amount of concentrate and minor changes in composition should not give trouble, but major changes in roughage type, or to roughage of poor quality, would seem undesirable.

4) Keep cows full of good-quality roughage. Feeding both roughage and grain more often than the usual 2× daily may be helpful.

5) Avoid the feeding of poor-quality, bad-smelling grass or legume silage high in butyric acid during this period.

6) Feed sufficient protein, minerals, and vitamins to meet the needs of the cow.

7) Provide facilities for adequate comfort, exercise, and ventilation.
8) In problem herds, take weekly milk tests of susceptible cows for the first six weeks after calving, to obtain a more specific evaluation of the problem.

9) In problem herds, feeding propylene glycol (3) or sodium propionate (24) to selected cows after calving may be useful. Selection could be made on the basis of the milk test.

10) There is evidence that susceptibility to ketosis is inherited, but because it is so closely related to high production, selection away from it is difficult. Production being equal, it would seem a good idea to favor strongly pure cows with good appetites in the selection program.

References