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DISCUSSION

- Professor Wodzicki: Were there any experiments on the palatability and digestibility of dehydrated meat by physiologists on rats or humans?
- Dr. Andrews: Yes, Dr. Cunningham of the Agriculture Department is working on rat tests, and satisfactory human tests have been carried out in Australia on the Australian product.
- Mr. W. G. Bonner: What, if any, are the by-products of dehydrated meat?
- Dr. Andrews: The by-products received from dehydrated meat, are the bones from the boning out of the carcasses, and a certain amount of surplus rendered fat which is converted into a satisfactory edible tallow.

"KETOSIS OF DAIRY COWS"

by

M. G. Armstrong, Veterinarian, Masterton.

Synonyms:- Acetonaemia, Aceturia, Acidosis,
or Post Partum Dyspepsia.

Ketosis is a nutritional complaint arising under conditions of inadequate carbohydrate or sugar intake by the animal system and it is characterised by the presence of certain highly poisonous abnormal fatty acids called ketone bodies in the blood.

It is a well recognised condition in human physiology as a certain state of ketosis exists in Diabetes mellitus - a disease affecting the utilisation of carbohydrates; it is also precipitated in patients for special therapeutic purposes by giving a diet low in carbohydrate and rich in fat. It also appears in conditions of starvation when the body has depleted its reserves of glycogen and is utilising its own fat reserves.

Before discussing this condition as it affects dairy cows it is expedient to review a certain aspect of fat metabolism and its dependence on carbohydrates.

When an animal is called upon to utilise fat from its reserves, the fat must first be desaturated in the liver to simpler acids which are then finally oxidised in muscles and other tissues. The complete oxidation of fat to carbon dioxide and water, however, is dependent on the presence of carbohydrate in the simple form of glucose in the tissue cells.

When the tissues are deprived of carbohydrate - for example during starvation or on a diet free from carbohydrate - the oxidation of fat is incomplete and intermediate metabolic products are formed in the tissues and pass to the blood and urine. The chief of these ketone bodies or intermediate products of fat metabolism are diacetic acid, beta-oxybutyric acid, and acetone.

The presence of these ketone substances in the urine indicates that the supply of carbohydrate to the tissues is inadequate.

To prevent a state of ketosis it is estimated that a given amount of fat to be oxidised requires twice that amount of sugar present in the tissues for complete metabolism.

Actual measurement of ketosis cases often fails to reveal any increased H-ion concentration of the blood because of the perfect compensatory processes brought into play by the body. The term acidosis by which this condition is often named is therefore not as satisfactory as ketosis.

Diacetic acid readily disintegrates in the bladder and lungs yielding carbon dioxide and acetone. Acetone can be detected in the urine, milk or breath, hence the name aceto-naemia which is frequently used.

OCCURRENCE:

Ketosis in cattle is chiefly a complaint of mature high producing dairy cows and it is evident that the incidence is on the increase as a result of the general selection during recent years for high production. In the later years it has been widely recognised in the United States, Canada, and in Europe; while in New Zealand it has been diagnosed with varying annual severity in the Waikato, Taranaki, and Wairarapa Districts. If more information on accurate details of cattle complaints were obtained the condition would doubtless be existent in all dairying districts of New Zealand in the spring and autumn.

The actual occurrence of ketosis in a dairy herd can be classified into parturient and non-parturient types. One attack may follow another in the same or succeeding lactations and it is known to occur at any time of the year, although the greatest incidence falls at, or shortly after, calving.

Following extensive investigation in U.S.A., Udall estimated that of the total incidence, 40% is parturient, occurring within the first two weeks after calving. Several of these cases are complicated with or follow Milk Fever (Hypocalcaemia) or Grass Stagger cases or may be associated with uterine affections.

The remaining 60% of cases are classed as non-parturient and it is estimated that at least one half of them occurs between two and six weeks after calving, while the remainder occurs chiefly in the late summer and autumn. Although no extensive survey has been made yet in New Zealand it is becoming more clear that the incidence is similar to that recorded by Udall in U.S.A.

A state of ketosis is therefore most frequently recognised in dairy cows in the earlier stages of lactation and, except for odd cases resulting from the devitalising influence of certain persistent diseased conditions (septic metritis, degeneration of the liver, etc.), the condition is precipitated by a dietary deficiency of digestible carbohydrate when the animal is forced to maintain itself or produce off its body reserves.

SYMPTOMS:

The symptoms of ketosis show such a great variability that a diagnosis is uncertain without a positive test for ketone bodies in abnormal quantities in the urine, milk, or blood.

The ketones, particularly diacetic acid, are highly poisonous substances resulting generally in a depression of the higher centres.

Symptoms can be classified generally into three main syndromes - Digestive, Nervous and Comatose.

1. Digestive:

These symptoms are recognised in most of those cases in the non-parturient class. In severe cases the first alarm is a sudden cease of milk production when the cow is near her peak of production. Almost total loss of appetite is followed by a rapid loss in condition which greatly intensifies the ketosis. There is a varying degree of mopingness or lassitude and affected cows are often observed wandering in a dazed and cataleptic state. There is usually a thin diarrhoea. The temperature remains normal. In mild cases the onset of these symptoms is gradual.

2. Nervous Syndrome:

These symptoms are so typical of grass staggers that this syndrome is either liable to confusion with, or is a complication of grass staggers. Treatment for grass staggers is insufficient without a simultaneous treatment for ketosis. Symptoms usually observed are complete loss of milk flow, inappetence, a varying state of excitability with convulsions or twitchings of muscles, and varying degrees of nervous staggers leading quickly to prostration. This syndrome is a common type to meet shortly after calving.

3. Comatose Syndrome:

In this type of ketosis the symptoms observed are practically identical with those usually seen in milk fever, and, as it is frequently in association with milk fever, it is evident that an accurate diagnosis is essential for successful treatment.

In the U.S.A. another type of ketosis called "Licking Mania" has been recognised.

AETIOLOGY:

As pointed out previously ketosis results from a lack of available carbohydrate or sugar for the complete oxidation of the fats which are being converted to energy.

In New Zealand the fat content of pasture and supplementary fodders is very low, and therefore this condition is generally confined to cattle maintaining themselves or producing from the fat reserves of their bodies. It is, therefore, observed most in herds improperly fed during the winter - at a time when the cow has an added demand of a rapidly developing foetus plus a sudden plunge into high milk production, when grass growth is still at its lowest ebb.

The liver store of carbohydrates in the form of glycogen is low during the later stages of pregnancy and the depletion is accelerated by the onset of milk production.

Susceptible cows are those which are in low condition at calving time and which are fed silage, poor quality hay, or feeds poor in sugars.

An important predisposing factor is a long cold winter when cows are losing condition rapidly at the calving period. Other higher incidences are recorded on overstocked farms and in herds fed excessive quantities of silage.

During the spring and summer the complaint is frequently observed in high yielding cows on low carbohydrate rations.

In Canada and U.S.A. ketosis may become malignant in stall fed cattle when they are fed poor quality hay and supplements low in available carbohydrates.

VITAMIN BI:

It is known that the animal system must have adequate Vitamin BI to make full use of carbohydrates. Vitamin BI can be synthesised in the digestive tract of ruminants and digestive disturbances in odd cases may cause ketosis as a secondary complication. Pasture, hay, turnips and molasses contain fair amounts of Vitamin BI but what part this vitamin may play has yet to be determined.

DIAGNOSIS:

The disease can be readily recognised by the veterinarian with the aid of Rothera's Test by which he can test urine and milk for the presence of diacetic acid and acetone in abnormal amounts. The addition of alkali and sodium nitroprusside to the sample gives a wine red colour in a positive case.

Gerhardt's Reaction for diacetic acid in urine, using 10% ferric chloride, can also be used easily in the field. After adding acetic acid to fix the phosphates, a few drops of ferric chloride are added to the urine - a wine colour indicates a positive ketonic urine.

Before treatment the urine is darker than normal, of low specific gravity, acid in reaction and low in phosphate. With improvement, the specific gravity rises, the reaction tends to become alkaline, and in the later stages of recovery, is loaded with phosphates.

TREATMENT:

The principles of treatment are based on a relief of alarming symptoms, a fixation of the ketone bodies and a correction of the low sugar factor. A powerful narcotic such as chloral hydrate is indicated for nervous cases - while strychnine and calcium injections for comatose cases. As soon as the animal can swallow, liberal frequent doses of molasses are of outstanding value. If the animal is unable to swallow, large intravenous injections of glucose are administered. Chlorides are claimed to fix ketones and render them harmless. Large doses of common salt and sodium bicarbonate are indicated in severe cases, to check acidity and fix ketones. A good proportion of cases are back to full milk within three to five days after the start of treatment.

PREVENTION:

The prevention of this complaint is worthy of serious consideration, owing to the fact that a serious loss of milk production, at a time when a cow is reaching its maximum peak, is involved. In addition to this serious loss of milk production, there is a death toll of high producing cows if they are unattended by skilled veterinary aid. The farmer's usual paralysis, milk fever, grass stagger, or indigestion treatments fail, owing to an incorrect diagnosis.

There is direct evidence from the field that an abundant supply of sugars or sugar-forming feeds in the winter and spring, will prevent serious calving complaints, in particular ketosis and complicated milk fever, which result from a low carbohydrate intake at that time.

As our dairy herds are being raised to high production levels, certain complaints such as ketosis arise as further warnings that our dependence on pasture and certain winter supplements needs revision, to prevent wastage to the point of serious economic loss in production.

From the foregoing data, it is learned that the greatest incidence of ketosis falls within the first six weeks of calving, and it is known that the condition of the cow at this time is most important, since the stimulus to milk production is so strong that most animals will milk off their body reserves of fat and protein if the plane of nutrition is inadequate. The loss in body weight under these conditions can be very rapid. Kellner records losses in weight to the extent of 100 lbs. per cow per day when producing milk on starvation rations.

It is known that a 10 cwt. cow producing 4 gallons of milk per day at a 5 per cent test requires 19 lbs. of starch equivalent, and 3.5 lbs. digestible protein per day in a ration within the daily limit of 30 lbs. dry matter.

High testing milk with 5-6% fat contains, in addition, 5-6% carbohydrate, and 4-5% protein.

The problem of feeding heavy milkers is to get sufficient protein and starch equivalent in a ration that is within their appetite.

By what methods can we overcome the deficiency of sugars in the winter and early spring and thereby overcome the serious incidence of ketosis and its complications?

METHOD (1) - MOLASSES:

Extensive observations since 1940 by the Morrinsville Veterinary Association on 50,000 cows on 700 farms led to the conclusion that a low incidence of fatty cow ailments at, and shortly after, calving, is associated with a high consumption of molasses in the winter and spring months.

The lowest incidence of calving ailments is recorded in cattle fed in the winter and early spring with an adequate supply of good quality hay sprayed with an abundance of molasses.

Satisfactory prevention is obtained by feeding molasses alone in quantities as small as $\frac{1}{2}$ -lb. solidified block per cow per day.

The feeding of sugar supplements is a factor in reducing the high incidence of milk fever which is associated with the high protein, low carbohydrate intake of rapid spring pasture growth - a factor based on evidence that an abundance of sugar is necessary for the maximum utilisation of minerals, especially calcium.

A surprising number of ketosis cases precipitated in herds in January and February when pasture is good, suggests that the balance in certain pastures may be too much in favour of the protein.

METHOD (2) - SUGAR BEET:

Since the quantity of molasses available is likely to fall short of the sugar requirements of our dairy herds, it is suggested that sugar beet be grown in preference to other crops or silage to supplement good hay.

There may be no doubt that good quality hay, by virtue of its relatively high carbohydrate analysis, is essential, but, it is considered inadvisable in the interests of the animal's digestive system to feed more than 20 pounds per head per day.

The feeding of sugar beet in preference to other root crops is worthy of serious consideration since its advantages over the mangel alone are numerous, viz:-

1. It has $2\frac{1}{2}$ times the sugar content and feeding value of mangel. For the same energy value $2\frac{1}{2}$ times the bulk must be grown and handled when feeding mangels. It is relished by stock and there is a greater chance that the requirements of stock will be met within their normal appetite.
2. The bulbs can be fed immature, straight from the paddock, or in contrast, from storage heaps as late as November after being out of the ground for 4 to 5 months.

Sugar beets will grow wherever mangels grow. Crops of 40 to 50 tons of roots and 20 to 30 tons of tops have been grown in South Taranaki, Manawatu, Hawke's Bay and Waikato. With the aid of the single furrow plough there is little difficulty in pulling the beet.

Dried sugar beet leaves have a feeding value equal to good hay and dairy cows do extremely well on the roots and/or dried leaves up to $\frac{1}{3}$ rd their total ration, i.e. up to 20-30 pounds roots, or 15-18 pounds leaves per day.

It is known that the sugar beet tops contain oxalic acid up to 3 to 4% of the dry matter. The tops can be fed in larger amounts to cattle and sheep than to horses or pigs, since the fermentations in the paunch of ruminants destroys some of the oxalic acid. If it is desired to feed the maximum amounts of tops, it is well to add 1 ounce of fine ground limestone or chalk to each 50 lbs. of tops to precipitate the oxalic acid.

In conclusion, it may be stated in addition, that a state of ketosis with considerable quantities of acetone in the blood and urine is pronounced also in the disease Pregnancy Toxaemia or Sleepy Sickness (Anti-partum paralysis) of Ewes, and in the prevention of this complaint in the ewe, more consideration should be given to the value of feeding rich sugar-forming feeds such as hay and molasses or sugar beets.

APPENDIX.

COMPOSITION OF FEEDS: T. B. Wood and H. E. Woodman - "Rations for Livestock":-

Feed	Dry Matter	DIGESTIBLE NUTRIENTS		Starch Equivalent.
		Sol. Carbo-hydrates	Fibre	
Mangels	13.2	9.4	0.3	6.8
Swedes	11.5	7.5	0.8	7.3
Soft Turnips	8.5	5.2	0.3	4.4
Mangel Leaves	11.0	3.5	0.9	5.3
Sugar Beet Tops (wet)	16.2	7.2	1.1	8.6
Pasture (spring)	20.0	7.3	2.6	11.2
Sugar Beet	23.4	19.3	0.4	15.0
Grass Silage	32.0	7.6	5.6	10.5
Good Hay (meadow)	85.7	25.7	15.0	37.0

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N.Z. Journal Ag. August, 1940, 61, 87 - Sugar Beet.
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 N.Z. Journal Ag. March, 1941, 62, 173 - ditto.
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DISCUSSION

Mr. Webster: deprecated the modern trend to describe Acetonaemia or Ketosis as a disease entity, and maintained that the presence of Ketone bodies in the blood was no more than a pathological symptom of a disturbance in carbohydrate metabolism. Any slight disturbance in the normal equilibrium of the cow is sufficient to produce a transient Ketosis without any clinical symptoms whatever. It would be preferable to revert to the older terms such as pregnancy, toxæmia, parturient paralysis, post-parturient dyspepsia, etc.

Reply: Mr. Webster has misunderstood the text of the paper in that it is dealing with the presence of ketone bodies in the blood and urine in such abnormal quantities that a constitutional disease entity with disturbance of function of the system and recognisable symptoms is the result. The physiological upset which may be a primary or secondary disease is correctly called ketosis or acetonaemia. This must be distinguishable from ketonuria or acetonuria in which the ketone bodies are not in abnormal quantities in the blood but can be satisfactorily excreted by the kidneys.

It has long been known that certain devitalising influences or physiological disturbances can result in a transient ketonuria which is not accompanied by clinical symptoms.

To say that ketosis is only the result of a disturbance in carbohydrate metabolism, is to neglect recognised physiological knowledge on the various causes and to choose a great exception rather than the rule.

Considering the feed and production conditions under which this disease appears in a primary state in various districts of New Zealand, and in view of the outstanding value of the carbohydrate therapy using molasses as treatment and prevention, it is clear that the incidence particularly in the winter and spring is due to insufficient carbohydrate or sugar intake for the complete metabolism of fats.

The name pregnancy toxæmia is not included as a synonym as little is known of its true aetiology or whether the pronounced ketosis is a primary or secondary diseased condition.

Post-parturient dyspepsia is a misleading term of the old school, which describes one of the three syndromes of ketosis only.

Acetonaemia and ketosis are both correct names although ketosis is more satisfactory since it includes the presence of any one of the numerous ketone bodies.
