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ACETONAEMIA IN THE DAIRY COW

By C. R. TOOP, B.V.Sc., Chief Veterinary Surgeon

ACETONAEMIA—sometimes called ketosis or acidosis—occurs quite frequently in dairy herds, but is rarely recognised as such by dairy farmers. Many cases of the disease have been encountered in the field and many others have been reported in which the description furnished has left little doubt concerning the identity of the condition. The available information suggests that the incidence of the disease has increased during recent years. For the most part, acetonaemia occurs during the first six weeks of lactation and is characterised by loss of appetite, marked reduction of milk yield, rapid loss of condition, listlessness and constipation, and at times by the presence of a peculiar sweetish odour which may be detected in the breath, milk and urine. In some cases there are nervous complications.

The mortality rate is low and recovery is the rule. Since the disease occurs during lactation when it greatly depresses the milk flow it may become a source of considerable loss. The majority of cases respond promptly to appropriate treatment.

CAUSE

The actual cause of the disease has not been determined but there is little doubt that it is a nutritional disorder. It is believed to be due to a derangement of metabolism in association with which the blood sugar falls below the normal level and abnormal amounts of toxic substances known as ketone bodies accumulate in the blood and tissue fluids. This may be due to a failure on the part of the animal to derive a sufficient amount of carbohydrate from its diet or to an inability to properly utilise the carbohydrate that is available. Carbohydrates are represented by such substances as starch, fibre and sugars and provide the principle source from which an animal derives its energy requirements. When the carbohydrate falls below the required level the animal is forced to draw upon its fat reserves. When fat is completely burned or oxidised in the body the end products are carbon dioxide and water which are excreted and cause no ill effects.

For the complete combustion of fat an adequate supply of carbohydrate is necessary. When the supply of carbohydrate is inadequate the oxidation of fat cannot

proceed to completion but becomes arrested at an intermediate stage wherein the fat has become converted to substances known as ketone bodies which accumulate in the blood and are excreted in the breath, milk and urine to which they impart a characteristic odour. Ketone bodies are acid in reaction and possess toxic or 'poisonous' properties. Acetone is one of these and it is from the presence of this substance in the blood that the name acetonaemia is derived.

The latest information available suggests that the actual cause of the upset in carbohydrate metabolism is due to a glandular deficiency. It is thought that there is a temporary failure of the adrenal gland (a small gland located near the kidneys) so that one of the hormones essential to carbohydrate metabolism is produced in insufficient amounts.

The functions of the adrenal glands are in turn controlled by the pituitary of "master gland" of the body. This gland located just below the brain secretes, amongst others, a hormone called the anterior pituitary corticotrophic hormone, which is now commonly known as A.C.T.H. and which regulates the output of hormones from the cortex of the adrenal gland.

The production of A.C.T.H. is increased when the body is subjected to strain, cold, heat, exhaustion or high body temperatures caused by fever. The adrenal gland goes

to work to help the body meet the needs caused by such stress but continued heavy strain or stress however, causes certain changes in both the pituitary and adrenal glands as well as some of the organs of the body. The resultant upset in carbohydrate metabolism probably produces the symptoms of the disease.

SYMPTOMS

Although the disease may occur at any time during the lactation period, the majority of cases occur from about a week to six or eight weeks after calving. Cows of any age may be affected but the condition is most frequently observed in well-nourished high-producing animals. Two forms of the disease may be recognised—the digestive form and the nervous form. The former is most commonly encountered.

Digestive Form.

In this form of the disease there is a sudden or gradual loss of appetite and this is accompanied by a rapid loss in condition and a marked fall in the milk yield. The bowels are usually constipated, droppings being passed infrequently and in small amounts. Diarrhoea has been observed in some cases but this is unusual. The cow is dull and listless and a sweetish chloroform-like odour of acetone may in some cases be detected on the breath, milk and urine.

The detection of this characteristic odour may depend on the keenness or otherwise of the investigator's sense of smell. While it will be immediately noticed by some people on entering a shed occupied by an affected animal, others will fail to detect anything unusual.

Nervous Form.

In addition to the symptoms occurring in the digestive form of the disease, cows affected by acetonæmia may show a variety of nervous symptoms. In some cases there may be constant licking of the skin or licking or biting at the manger and other objects. There may be sucking of the tongue, slobbering from the mouth, grinding of the teeth, champing of the jaws and rolling of the eyes. The gait may be staggering and unsteady and the cow may have difficulty in remaining on

her feet; she may fall and may remain down for some time before being able to regain her feet. Other symptoms which have been described include marked excitement with wild expression and staring eyes, violent movements, walking in circles, stringhalt and convulsions.

It should be recognised that the digestive form of the disease predominates and that only a small proportion of cases may become complicated by one or more of the nervous symptoms above described.

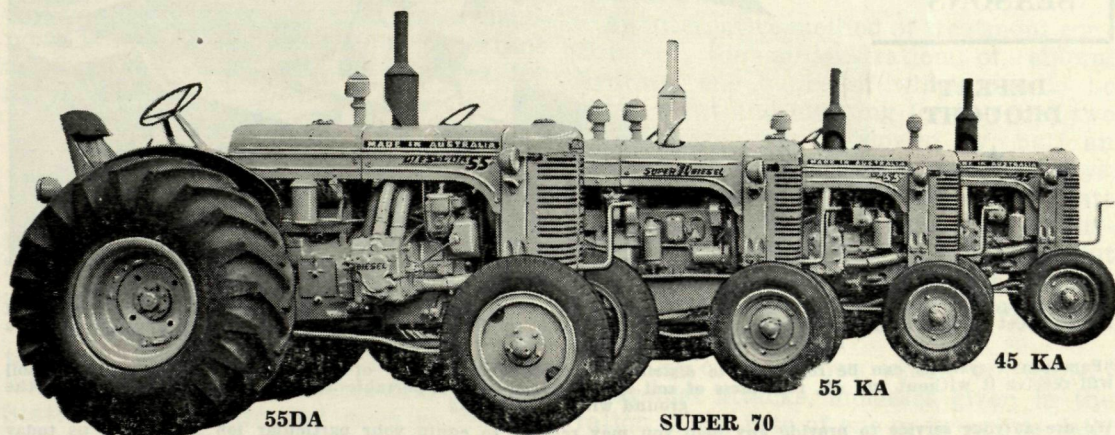
COURSE OF THE DISEASE

The majority of cases of acetonæmia terminate in recovery. With appropriate treatment recovery will usually occur within the space of a few days. Untreated cases or those which fail to respond to treatment, however, may persist for several weeks during which period the animal becomes emaciated and the already diminished milk flow may cease entirely. In prolonged cases the cow may be rendered worthless until the next lactation period. Severe emaciation and debility may result in the death of the animal. The mortality rate, however, is very low. Some animals are subject to recurrent attacks of the disease, an apparent recovery accompanied by a return of appetite and rumination being followed shortly afterwards by a relapse.

DIAGNOSIS

From a consideration of the history of the attack and the symptoms exhibited, the diagnosis of the disease will usually present little difficulty. When symptoms of loss of appetite, rapid wasting, greatly diminished milk yield and constipation occur during early lactation, acetonæmia should be suspected. If in addition the characteristic odour of acetone is detected in the breath or should any of the nervous symptoms described make their appearance this will provide confirmatory evidence. It is, however, necessary to mention that an odour of acetone may occasionally be present in the breath of apparently healthy cows or in cows suffering from some other diseases and it is consequently unwise to place absolute reliance on this feature alone. In order to confirm the diagnosis, samples of milk or urine from an affected animal may be submitted

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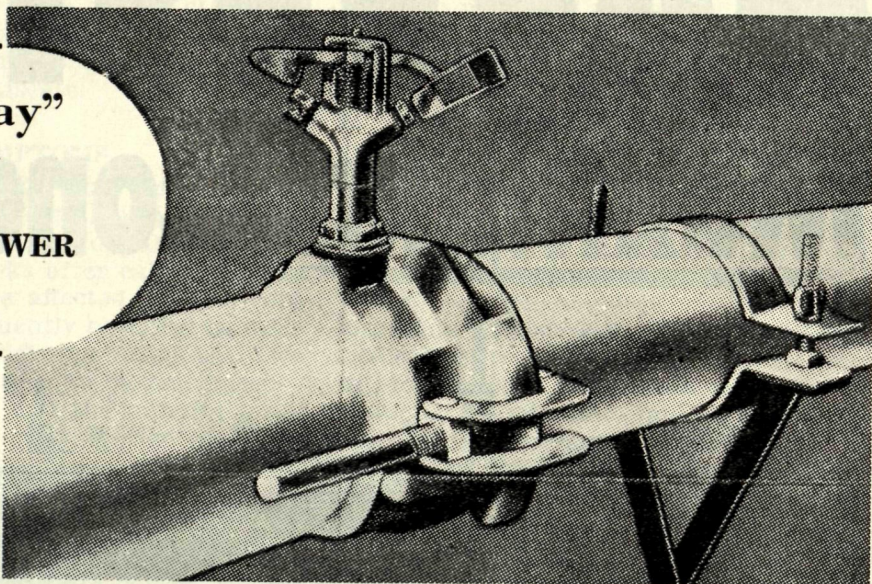
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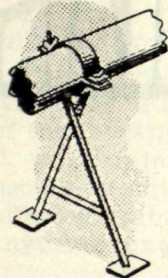
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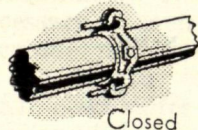
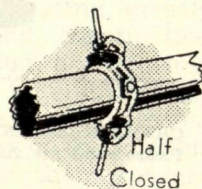
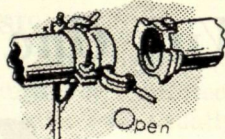
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to a simple chemical test in which a positive result is denoted by a colour reaction. A strongly positive reaction considered in conjunction with the symptoms will serve to place the diagnosis beyond doubt. When confirmation is desired milk or urine samples may be forwarded to this Department for testing. The amount of each need not exceed a fluid ounce and the sample should be despatched immediately after collection, otherwise it may prove unsuitable.

Acetonaemia may be a complication of other metabolic diseases such as milk fever, or other conditions may be secondary to acetonaemia. In such cases diagnosis is not always easy but is important as treatment may also be required for those conditions.

TREATMENT

Many treatments have been described for acetonaemia and whilst most give reasonable results they have not always been successful in re-establishing appetite and milk production rapidly.

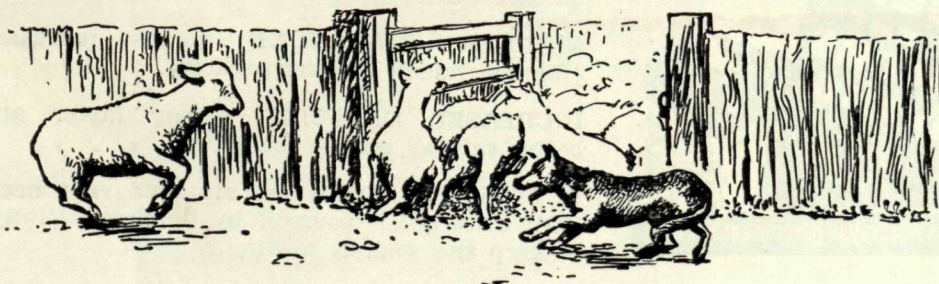
However, basing the treatment on the theory of glandular upset, A.C.T.H. injections have been most successful in this State and elsewhere and cows so treated mostly regain their appetite within 24 to 48 hours. In most cases one injection is

all that is required. The use of A.C.T.H. is confined to veterinarians but its cost is not now so high as to preclude its use.

A treatment which can be adopted where veterinary services are not available is the injection of a 40 per cent. solution of glucose (approximately 7 oz. of glucose dissolved in 17 oz. of boiling water and allowed to cool). This is injected subcutaneously (under the skin) preferably in three or four different sites. A single injection may result in recovery but four or five injections repeated on successive days may be necessary before the desired result is obtained.

An alternative method of treatment consists of the administration of chloral hydrate, one ounce of which should be given night and morning for the first two days of treatment followed by half an ounce once daily for the next six days. In the administration of chloral hydrate the prescribed dose of the drug is dissolved in a pint of hot water and a pint of treacle or molasses added, the mixture being given to the cow in the form of a drench.

In the case of cows which are subject to annual attacks, molasses given in the ration at the rate of one or two pints daily for the first few weeks following calving may be beneficial.

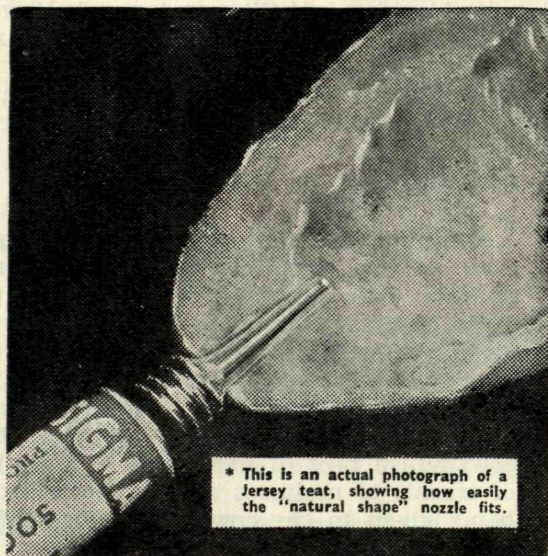


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