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Studies on the Alimentary Tract of Merino Sheep in South Africa. XIII.—The Rôle of Prussic Acid in the Aetiology of Acute Bloat.

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Introduction.

In 1943 Quin showed that bloating in ruminants was frequently associated with the formation of a frothy mass in the rumen, attributed to the rapid fermentation of sugar in the presence of saponin in the lucerne on which the experimental animals were fed. This foamy consistence of the ruminal mass was held to be responsible for the difficulty in eructation of the gas. It was stated that the dangerous fulminating production of gas from green lucerne could be controlled by feeding other materials first, thereby partially satisfying the metabolic requirements of the ruminal flora and retarding the gas formation from the lucerne to within reasonable limits.

A similar method for the prevention of bloat in cattle is advocated by Cole *et al* (1943), namely the feeding of hay or other roughage prior to pasturing on lucerne, but these authors attribute the success of this procedure to the stimulation of ruminal motility by the presence of coarse material in the rumen. This explanation is supported by the word of Schalk and Amadon (1928) who reported that ruminal movements could be initiated by mechanical irritation of the lining of the anterior rumen.

The question that arises is whether the successful eructation of gas depends on ruminal motility or not. In 1942 Cole and his coworkers stated that belching occurred only when the rumen was in an active state of contraction. In their later publication (Cole *et al*, 1943) it is admitted that the mechanism of belching in cattle is not known but it is suggested that it is closely allied to that of rumination. On the other hand Schalk and Amadon (1928) found that no special ruminal contraction is associated with the act of food regurgitation. It must also be remembered, as pointed out by McIntosh (1941), that there are two types of acute bloat, one where free gas is superimposed on the ruminal mass and the other where it remains admixed with the ingesta. In the former type the retention of the gas may be due to a mechanical obstruction of the oesophagus or to the abeyance of the act of eructation and can easily be released by the passage of the probang or by the use of the trocar. In the second type the gas is held in the ingesta as a foam and can neither be eructated nor released by the measures already mentioned.

In view of the doubt as to the connection between ruminal motility and eructation, the present authors decided to ascertain whether a sheep with a paralysed rumen could eructate gas.

Method.

Merino sheep with a permanent ruminal fistula as described by Quin *et al* (1938) were used. Potassium cyanide was employed to bring about ruminal paralysis as Quin and van der Wath (1938) had shown that small doses of this compound caused complete stasis of ruminal movements for a short period and also because of the possible rôle of cyanide in the aetiology of acute bloat on wilted green pasture. Potassium cyanide (1·1000 aqueous solution) was introduced directly through the fistula into the ruminal mass, repeated small doses being given until either ruminal paralysis or symptoms of general poisoning set in. The ruminal movements were counted continuously over five minute periods throughout the experiment. After preliminary trials, the standard rate of dosing adopted was 100, 100, 50, 50, 100, 100 mgm. KCN at five-minute intervals.

Results.

As acute bloat usually sets in during active ruminal fermentation, the first experiments were carried out on sheep immediately after the consumption of from 300 to 500 gm of lucern hay. It was found that, under these conditions, surprisingly large amounts of cyanide could be tolerated, the actual doses required to cause ruminal stasis in four individual sheep being respectively 650, 900, 1,000 and 650 mgm. KCN (average 800 mgm.).

When the experiment was repeated on the same sheep after all food had been withheld for 14 hours, the amounts of KCN which brought about ruminal paralysis were respectively 200, 100, 200 and 200 mgm. (average 175 mgm.).

As the enhanced resistance to KCN after feeding was out of all proportion to the calculated increase in ruminal contents, it was considered that it could not be due merely to a dilution effect. Neither did it appear likely that the explanation could be the neutralisation of HCN by combination with the increased amount of aldehydes and ketones (principally sugars) in the ruminal mass.

After feeding lucerne, sheep normally show a marked increase in the depth of respiratory movements, presumably due to the absorption of additional carbon dioxide from the rumen during active fermentation. Such an increase in pulmonary ventilation would simultaneously accelerate the elimination of a volatile poison such as HCN and it was suspected that this might be the explanation of the greater tolerance to cyanide shown after lucerne feeding. In order to test out this hypothesis the experiments were repeated on the same sheep after again withholding food for 14 hours but carbon dioxide was now introduced into the rumen together with the KCN solution.

The carbon dioxide was allowed to escape from an ordinary pressure cylinder into a gasometer and thence, after being heated to body temperature, was led through the fistula into the depths of the ruminal mass at the rate of two litres per minute. In order to allow its effect on respiration to develop the carbon dioxide was administered for 15 minutes prior to the commencement of dosing with KCN. This introduction of carbon dioxide into the rumen was in all cases immediately followed by a marked increase in the depth and rate of breathing.

Under these conditions it was found that there was a marked increase in the tolerance to KCN, the doses required by the four sheep in order to cause ruminal paralysis being 850, 750, 750 and 200 mgm, respectively (average 638 mgm.). These doses are comparable to those withstood by the sheep after lucerne feeding.

It was, however, realised that, in addition to increased pulmonary elimination, a portion of the HCN might be expelled from the mouth together with the eructated unabsorbed carbon dioxide. In order to assess the signaficance of this possible loss the experiments were repeated on two sheep with air substituted for carbon dioxide. It was found that two litres of air per minute introduced into rumen had no effect on either respiration or tolerance to KCN, the amounts of which that were required to cause ruminal paralysis being 300 and 150 mgm.

For the purpose of comparison the doses of KCN required to cause ruminal paralysis under the various experimental conditions are repeated below:—

TABLE 1.

Sheep No.	KCN required to cause Ruminal Paralysis (Mgm.).			
	After Feeding.	After 14 Hours Fast.	After 14 Hours Fast + CO_2.	After 14 Hours Fast + Air.
1	650	200	850	—
2	900	100	750	—
3	1,000	200	750	300
4	650	200	200	150
Average	800	175	638	225

It will be noted from the above results that the tolerance for KCN varies considerably in different sheep and even in individuals from time to time. Although the toxicity of KCN probably depends on various factors, the above data indicate that the absorption of CO_2 from the alimentary tract of the ruminant exerts a significant effect in causing an increased respiratory exchange and thereby facilitating the elimination of the poison.

The Effect of Ruminal Paralysis on the Eructation of Gas.

In order to ascertain whether the eructation of gas from the rumen could take place in the absence of ruminal motility, air or carbon dioxide was introduced into the rumens of the experimental sheep after ruminal paralysis had set in. In all cases it was found that either air or CO_2 (2 litres per minute) could be belched up despite the ruminal paralysis. In two instances this even took place while the animal was showing general symptoms of acute prussic acid poisoning. These experiments afford direct evidence that, despite the absence of any ruminal movements, none of the animals experienced any difficulty in eructating even excessive amounts of either air or CO_2 when these gases were artificially introduced into the rumen.

This somewhat surprising observation affords direct evidence that eructation can take place independently of ruminal motility. This fact is not in accord with the previously mentioned theory of Cole *et al* (1943), namely, that the feeding of roughage prior to lucerne protects against bloat by promoting ruminal motility through mechanical stimulation of the ruminal wall, thereby facilitating eructation. From observations made at Onderstepoort it would not appear that the absence of roughage necessarily leads to ruminal stasis, at least in the sheep, but even if this were the case the retention of ruminal gases would not be an inevitable sequence. It is more likely that the roughage acts in a mechanical way on the texture of the ruminal contents by preventing the formation of a frothy glutinous mass and allowing the gas to escape from the ingesta.

Summary.

It is shown that more than four times the amount of KCN is required to cause ruminal paralysis in sheep during active fermentation of lucerne in the forestomach than after a fast of 14 hours.

This increased tolerance to KCN after feeding is explained on the basis of an accelerated elimination of HCN from the lungs resulting from the greater respiratory exchange, which in turn is caused by the absorption of CO_2 from the alimentary tract during fermentation.

Similar results are reported by the artificial introduction of CO_2 into the rumen.

Sheep showing paralysis of the rumen, caused by KCN, are able to eructate two litres of gas per minute introduced through the ruminal fistula.

These observations, therefore, afford no evidence for incriminating the cyanogenetic factors in plants as being associated with the aetiology of acute bloat in ruminants.

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