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Sudies on the Alimentary Tract of the Merino Sheep in South Africa. XXII.—The Effect of the pH of the Ruminal Contents on Ruminal Motility.

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Introduction.

IN the preceding paper of this series Clark, Oyaert and Quin reported the occurrence of ruminal stasis accompanied by a high pH of the ruminal contents in sheep dosed with urea. Further investigations have been carried out into the effect of the reaction of the ingesta on ruminal motility.

The Buffering Powers of the Ingesta.

The literature contains considerable data as to the pH of the ruminal contents on different diets but no reports on investigations into the buffering powers of ingesta could be traced. This question was studied in the following manner.

Ingesta was withdrawn from sheep with permanent ruminal fistulae from which two 100 c.c. samples were taken. To one of these, measured amounts of normal hydrochloric acid were added and the pH taken after each addition. The other sample was similarly treated with normal sodium hydroxide. A Beckmann glass electrode pH meter was used throughout these and the subsequent experiments. These experiments were conducted on ingesta from several sheep on both lucerne hay and grass hay rations. Two typical results are depicted in Graph 1.

Graph 1.—As will be seen the ingesta showed strong buffering powers on the acid side but the addition of alkali caused a rapid rise in the pH. Contrary to expectations ingesta from sheep on lucerne hay showed weaker buffering action than that from sheep on grass hay.

The Effect of Dosing Alkali on the pH of the Ruminal Ingesta.

Eight sheep with permanent ruminal fistulae were used. Of these four were kept on lucerne hay and four on grass hay. The alkali was administered through the fistula tube.

From numerous trials it was found that the rise in pH from a fixed dose of alkali varied considerably, not only from sheep to sheep but also from day to day in individual animals. For instance sheep No. 8 (lucerne hay ration) was on one occasion given 75 gm. of sodium carbonate with a resultant maximum pH

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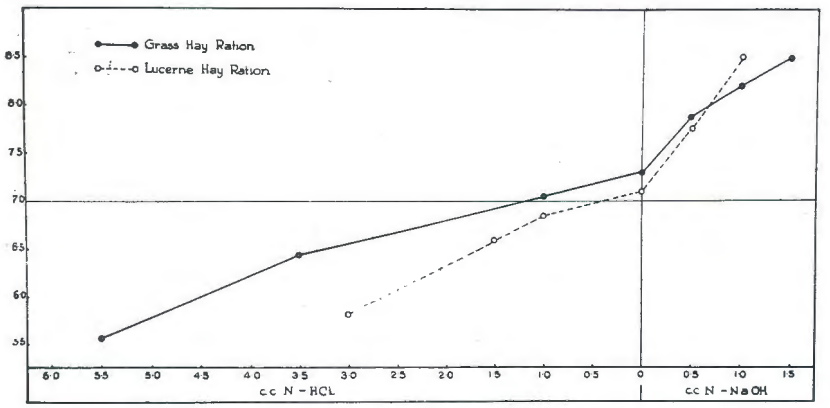
of 8·1. In another trial a dose of 40 gm. of sodium carbonate caused the pH to rise to 9·1. So marked was this variation in response that no definite conclusion could be drawn as to the effects of the different diets but, as will be seen later, sheep on lucerne hay appeared to be more susceptible to the effects of alkali dosing that those on grass hay.

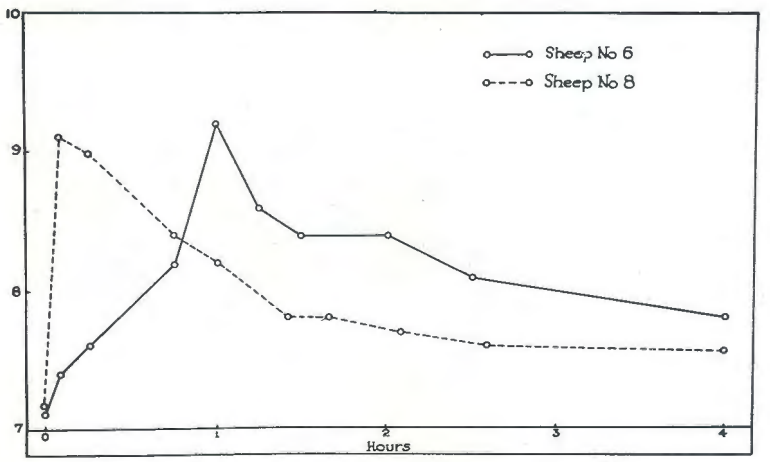
GRAPH 1.—*The buffering action of ruminal ingesta* in vitro.



Furthermore it was found that the administration of a single dose of alkali could cause either an immediate or delayed rise in pH. Graph 2 illustrates these two types of reactions.

Graph 2.—Both sheep were on a ration of lucerne hay at the time of the trials and each was dosed with 40 gm. of sodium carbonate. As will be seen the ingesta of sheep No. 8 showed a prompt rise in pH five minutes after dosing. Sheep No. 6, on the other hand, showed a delayed rise which only reached its maximum after one hour.

GRAPH 2.—*The effect of a single dose of* 40 *gm. sodium carbonate on the pH of the ruminal ingesta.*



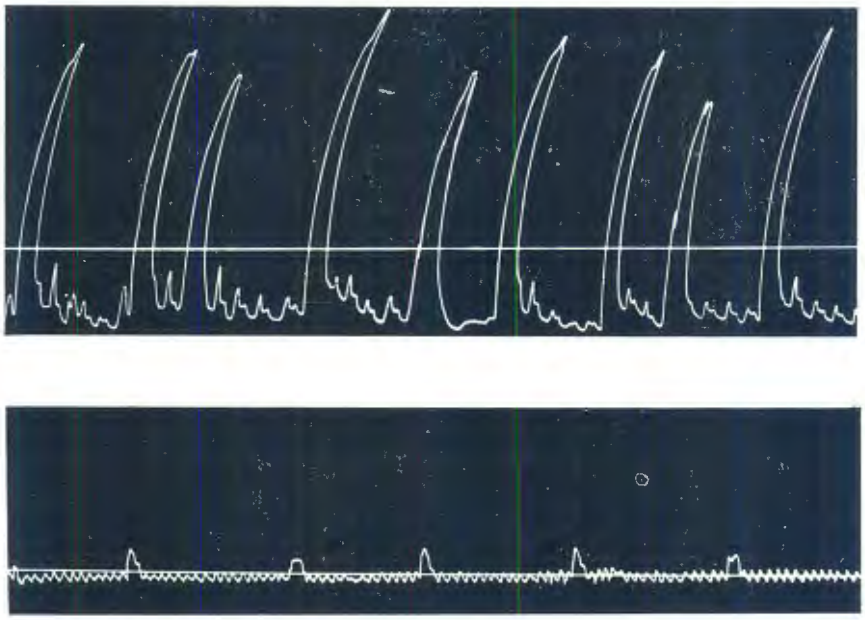
As pointed out by Smith (1941) the pH of the ruminal ingesta may vary considerably in different parts of the organ but by adopting a standard method of sampling we found that the variations between samples taken at short intervals were not important. Furthermore a gradual rise in pH after a single dose of alkali was found on several occasions and therefore it would not appear possible that the readings were due to chance selection in a badly mixed ingesta. No explanation can be offered for this finding nor could it be shown to occur *in vitro*.

The return to normal from a pH of 8 to 9 usually took 6 to 8 hours. In some cases, however, it was remarkably rapid as for instance a drop from 9·2 to 7·1 in one hour.

The Effect of pH on Ruminal Motility.

It was found that alkalinity of the ruminal ingesta was invariably accompanied by decreased motility of the organ. A typical tracing is shown in Figure 1.

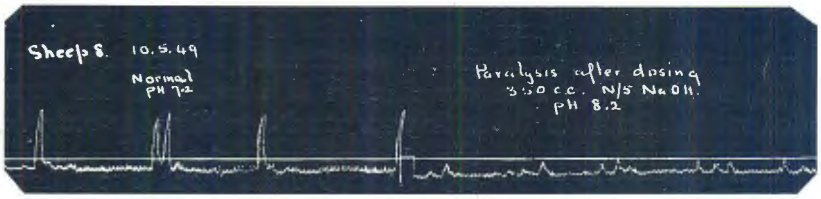
FIG. 1.—*The effect of dosing sodium carbonate on ruminal motility.*



Top.—Normal contractions at pH 7·0.

Bottom.—Ruminal paresis 6 hours after dosing 40 gm. Na_2CO_3. pH 7·8. Time of each tracing 5 minutes.

FIG. 2.—*The effect of dosing sodium hydroxide on ruminal motility.*



Normal contractions.
pH 7·2.

Paresis after dosing
350 c.c. N/5 NaOH.
pH 8·2.

Time of each tracing 5 minutes.

As will be seen from Figure 1 the excursion of the contraction was markedly decreased but the rhythm was not significantly altered. This was typical of the findings throughout the experiments. Even when the rumen was almost completely paralysed small abortive movements at regular intervals could still be discerned on the tracings.

Exactly the same reaction occurred if sodium hydroxide was given (see Fig. 2).

Considerable variations in susceptibility was found but in general it can be stated that a diminution in the size of the contractions occurred at pH 7·5 or over.

The Cause of the Ruminal Paresis.

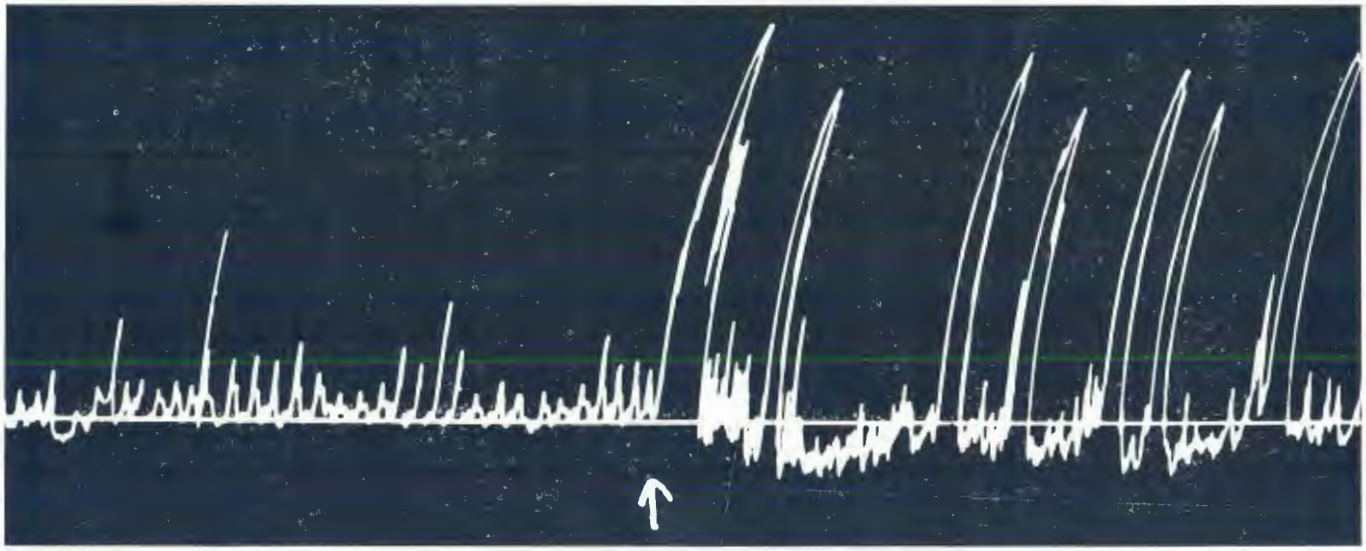
Several observations led to the conclusion that the decrease in the force of the contractions was due to inhibition of the vagal centres and not to any influence on the musculature itself. These were as follows:—

(*a*) *The Response to Feeding.*—Sheep standing in the recording stocks and showing ruminal paresis subsequent to being dosed alkali could seldom be induced to feed but on two occasions this was accomplished. In both instances the rumen immediatly responded with frequent contractions of normal excursion as illustrated in Figure 3.

Increased motility during feeding is initiated by stimulations of nerve endings in the mouth and pharynx acting via the vagus. The fact that the ruminal musculature could respond normally to such stimuli indicated that it was not affected. The rumen again became quiescent a few minutes after the food was removed.

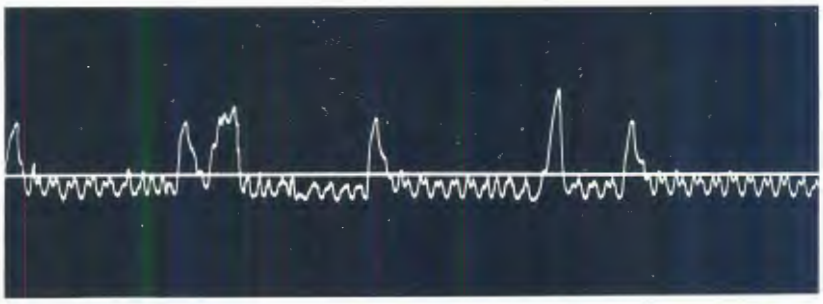
(*b*) *The Reaction to Carbamyl-choline-chloride.*—As seen in Figure 4, the injection of carbamyl-choline-chloride subcutaneously brought about hypermotility in the rumen paralysed by an alkaline content. This is further evidence that the muscle itself is not affected.

FIG. 3.—*The restoration of ruminal motility on feeding.*



Ruminal paresis following administration of NaOH. pH 8·2 Animal commenced feeding lucerne hay at arrow. Note immediate strong ruminal contraction.

FIG. 4.—*The reaction to carbamylcholine chloride.*



Small contractions due to dosing 40 gm. Na_2CO_3 pH 7·8.

Hypermotility 15 minutes after subcutaneous injection of 1 mgm. carbamylcholine chloride. pH 7·7.

(*c*) *The Effect of the Intravenous Injection of Alkali.*—The intravenous injection of small amounts of alkali (sodium hydroxide and ammonia) furnished convincing proof of the central origin of the ruminal paralysis. As shown in Figure 5 such treatment resulted in immediate cessation of movements.

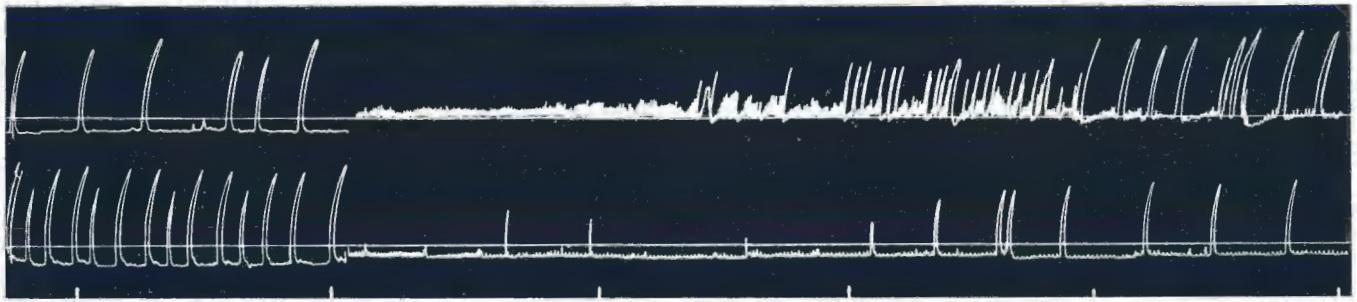
That the ruminal paralysis was not due to psychic disturbances caused by the handling and injecting was proved by the administration of equal amounts of normal saline under identical conditions with entirely negative results.

The Relationship between Ruminal pH and Motility.

The ruminal movements were not affected for some 10 to 20 minutes after the administration of alkali despite an immediate rise in the pH of the ingesta. In view of the prompt paralysis after the intravenous injection of alkali this would also support the hypothesis that the cause of the paresis is central.

The trial recorded in Figure 6 is of interest.

FIG. 5.—*The effect of the intravenous injection of alkali on ruminal motility.*



Above—

A. Normal.

B. Paralysis immediately after intravenous injection of 100 c.c. 1·3 per cent. NaOH.

C. and D. Gradual return of movements with hyperpnoea and restlessness 5 to 15 minutes after injection.

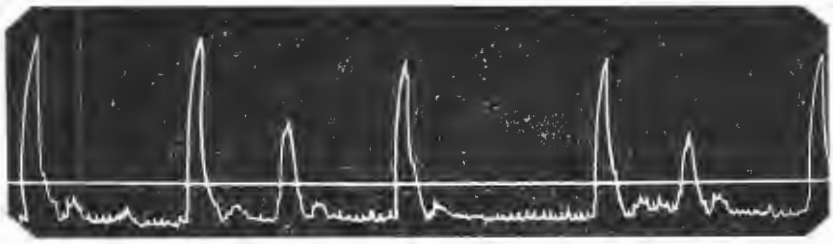
E. Recovery one hour later.

Below—

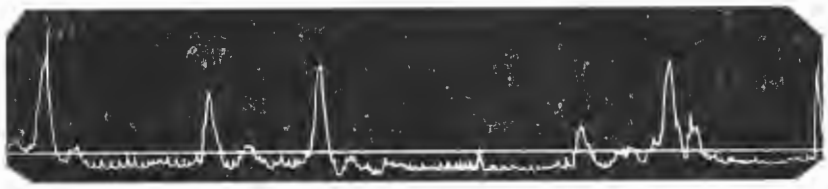
A. Normal.

B. to E. Continuous recording after intravenous injection of of 150 c.c. of 1 per cent. *ammon. fort.* in saline. Note immediate paralysis and rapid recovery.

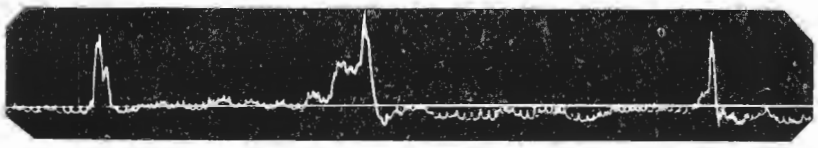
FIG. 6.



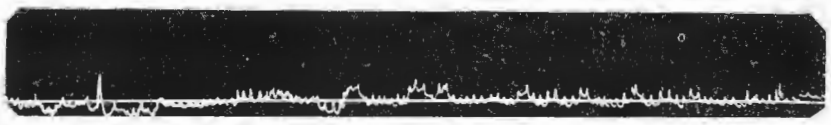
A. (9 a.m.) Normal movements before dosing. pH 6·9.



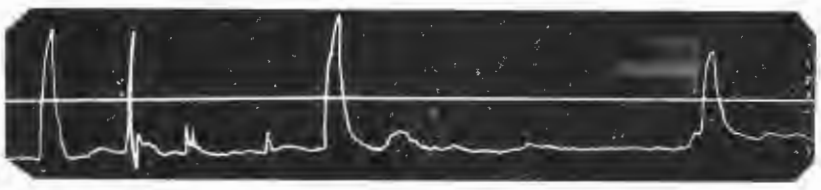
B. (9.10 a.m.) 10 Minutes after dosing 250 c.c. N/5 NaOH. pH 7·2. Note reduction in size of movements.



C. (9.30 a.m.). 5 Minutes after dosing further 100 c.c. N/5 NaOH. pH 9·2.



D. (10 a.m.). Complete paralysis pH 7·1 .



E. (10.30 a.m.). Recovery. pH 7·1.

It will be noted from tracing B that the administration of 250 c.c. of N/5 NaOH caused a diminution in the strength of the ruminal contractions although the pH of the ingesta was still well within normal physiological limits. This effect was presumably due to the absorption of alkali.

Referring to tracing C it will be noted that the dosing of a further 100 c.c. of NaOH solution resulted in a rapid rise in the pH to 9·2, yet ruminal movements were still present.

Half an hour later (tracing D) the pH had returned to normal but ruminal paralysis was complete. Recovery took place after a further 30 minutes.

Changes in the reaction of the ruminal ingesta were therefore reflected in an alteration in ruminal motility only after a latent period. Had only one recording been made at 10 a.m. (tracing D) the relationship between pH and motility would not have been apparent.

These findings again indicate that the paresis is not directly due to the presence of alkali in the rumen but to an upset in the acid-base ration of the blood which appears and disappears some time after the corresponding changes in the reaction of the ruminal ingesta.

The Effect of Acid on Ruminal Motility.

Having established the fact that an alkalosis caused ruminal paresis it was decided to investigate the corresponding effect of an acidosis. Owing to the powerful buffering action of the ingesta on the acid side already demonstrated, large amounts of acid caused little change in the pH of the ingesta. A dose of 100 c.c. of normal hydrochloric acid caused a shift from 7·3 to 6·7 in one sheep and 7·4 to 7·1 in another. In neither instance was there any effect on ruminal motility.

Likewise the intravenous injection of 150 c.c. of a 1 per cent. solution of both lactic and acetic acids failed to affect the ruminal contractions.

It could, therefore, not be demonstrated that an acidosis affected ruminal motility.

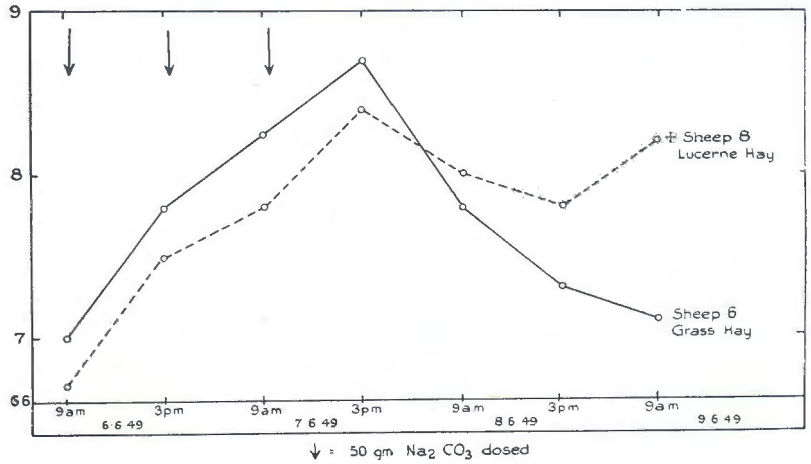
The Effect of Repeated Dosing of Alkali.

In view of the above findings it was decided to investigate the effect of repeated dosing with alkali with a view to the possible production of chronic ruminal stasis.

In the first trial two sheep were used, the one being on a grass hay ration and the other on lucerne hay. Three consecutive dosis of 50 gm. sodium carbonate were given at 9 a.m. and 3 p.m. on the first day and at 9 a.m. on the second day. Tracings of ruminal movements and pH readings were taken twice daily before dosing. The changes on the pH of the ruminal contents are shown in Graph 3.

It will be noted that in both sheep the pH of the ingesta rose sharply as a result of the treatment. After dosing was stopped the sheep on grass hay showed a rapid return to normal but the ingesta of the other animal remained highly alkaline. This sheep died two days after the last dose and showed an acute abomasitis at autopsy.

GRAPH 3.—*The effect of repeated dosing of alkali on the pH of the ingesta.*



Sections of the tracings of the ruminal movements are shown in Figure 7. The sheep on grass hay was highly resistant to the effects of the alkali whereas the animal on lucerne hay showed ruminal paralysis almost throughout the experiment.

It would therefore appear that, in this case, the repeated dosing of alkali broke down the powers of recovery but that the presence of the acute abomasitis prevented the development of the typical syndrome of chronic ruminal atony.

In a further similar experiment the dose of sodium carbonate was reduced to 25 gm. given twice daily over a period of 5 days. Once again the sheep on lucerne hay proved to be much more susceptible but typical chronic ruminal atony could not be produced.

During these experiments it was found that the maintenance of a high pH in the rumen caused inappetence and inhibition of both cellulose digestion and sugar fermentation.

The Treatment of Alkali Paresis by the Administration of Acid.

In several trials sheep showing ruminal paresis as a result of the administration of alkali were treated by dosing acid. It was found that although the pH of the ruminal contents could be corrected, this was not followed by an immediate restoration of motility. Owing to the great variation in the speed of recovery already noted in untreated cases, it was impossible to assess the beneficial effects of the acid. The intravenous injection of acid also had no immediate effect.

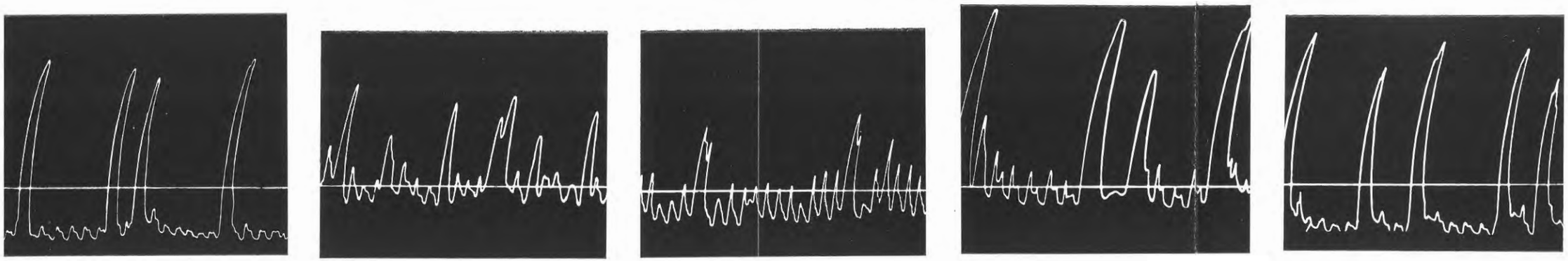
Discussion.

It has been shown that an alkaline reaction of the ruminal contents causes ruminal paresis. As far as the authors are aware this has not been reported previously.

It is difficult to assess the practical significance of this finding but it may well prove to be of great importance.

FIG. 7.—*The effect of chronic alkalosis on ruminal motility.*

Sheep 6.–Grass Hay Ration.



pH 7·0 — 9 a.m. 6-6-49

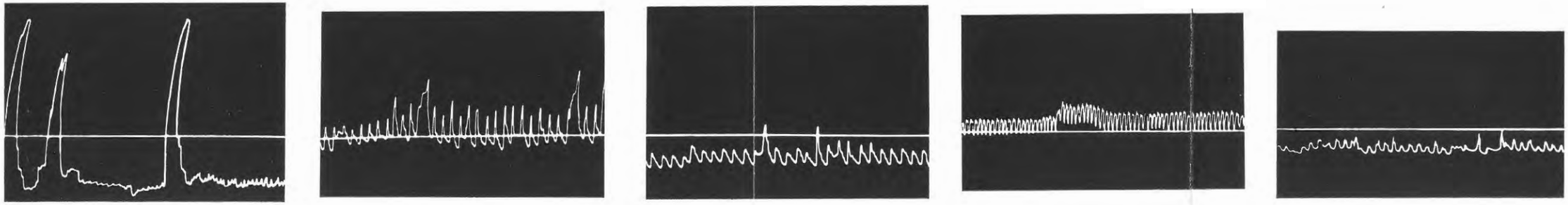
8·3 — 9 a.m. 7-6-49

8·7 — 3 p.m. 7-6-49

7·8 — 9 a.m. 8-6-49

7·1 — 9 a.m. 9-6-49

Sheep 8.–Lucerne Hay Ration.



pH 6·7

7·8

8·4

8·

8·2

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The normal regulation of the pH of the ruminal ingesta would appear to depend on two main factors, viz.:—

1. The interaction between the organic acids formed by the microbial breakdown of carbohydrates and the sodium bicarbonate of the saliva.
2. The selective absorption of volatile fatty acids from the rumen (Barcroft, McAnally and Phillipson, 1944).

It would therefore appear that the system is adapted mainly to prevent the development of excess acidity. The question that now arises is—does an alkaline reaction develop within the rumen under certain circumstances? De Boom (personal communication) has reported a high ruminal pH in cases of "Enzootic Icterus" associated with ruminal atony and impaction.

As the acid in the rumen is derived from the fermentation of carbohydrates, a cessation of this process might be expected to lead to an alkalinity of the ruminal contents. With this possibility in view two sheep were starved for 72 hours but this caused no rise in the pH of the rumen.

Oyaert, Quin and Clark (1951) showed that the administration of sulphanilamide into the rumen resulted in the inhibition of carbohydrate fermentation. Accordingly two sheep were dosed with 5 gm. sulphanilamide through the fistulae twice daily. On the morning of the fourth day, i.e. after 6 doses, one of the animals showed a ruminal pH of 7·8 and complete ruminal atony. The animal was completely off its feed and extremely weak. It was treated with acetic acid, sugar and normal ruminal ingesta all administered directly into the rumen. The following day the ruminal pH was 6·5 and the motility was normal.

This case is of interest in that it indicates that suppression of the ruminal flora may lead to an alkaline reaction of the ingesta and consequent ruminal stasis. On the other hand the second sheep was dosed twice daily for five consecutive days without showing any reaction except inappetence.

The true significance of alkalinity of the ruminal ingesta in the aetiology of ruminal stasis can only be assessed by direct observation on naturally occurring cases. This will present considerable practical difficulties for the following reasons:—

1. The ingesta must be withdrawn without contamination with saliva. Trials were made to compare the pH of samples of ingesta withdrawn from the same sheep (*a*) by stomach tube and (*b*) through the fistula. The following table gives the results obtained:—

	Sheep No.				
Sample.	1.	2.	3.	4.	5.
Per Stomach Tube.....	7·7	7·1	7·2	7·1	7·8
Per Fistula.....	7·1	6·5	6·7	6·7	7·3

It will be noticed that the samples taken per stomach tube all showed a higher pH than the controls taken per fistula. The stomach tube samples from sheep 1 and 5 appeared to be abnormally alkaline whereas the fistula samples were within the normal range. When taking the samples with the stomach tube the end of the tube was plugged with cotton wool before insertion, the plug being blown out after the tube had entered the rumen. This was done to avoid the collection of saliva in the tube. The higher alkalinity in the fore portion of the rumen is probably due to the presence of a greater

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proportion of saliva. In practice, therefore, samples taken per stomach tube will not give a reliable indication of the pH of the general ruminal mass.

2. Owing to the dark colour of the ingesta pH meters depending on colour reaction cannot be used. Ordinary litmus paper is useless as the colour of the ingesta masks the reaction. Even acid ingesta gives a blue colour.

3. The pH of ruminal ingesta tend to alter after removal. If left open it becomes more alkaline, due to the escape of carbon dioxide (Olsen 1941). If there is active fermentation taking place and the sample is tightly corked the pH drops. A variation of ·25 to 1 pH unit in either direction was recorded in a period of 24 hours.

An indirect method of approach would be to observe the effect of the administration of acid to natural cases of ruminal atony. Owing to the strong buffering action against acid exhibited by the ingesta there is little danger of causing excess acidity.

Acetic acid in the form of vinegar is physiological, cheap and easily obtainable and can be used in doses of 200 c.c. to sheep and 1,500 c.c. to cattle. As it has been shown that sugar fermentation is inhibited in an alkaline medium, sugar should only be dosed after the administration of acid. Treatment with acid would, of course, be contra-indicated where the ruminal paresis is due to cyanides.

Summary.

1. It has been shown that the administration of alkali (sodium carbonate or sodium hydroxide) into the rumen causes ruminal paresis if the pH exceeds approximately 7·5.

2. Ruminal paresis can also be caused by the intravenous injection of alkali (sodium hydroxide or ammonia) indicating that the paresis associated with alkalinity of the ruminal ingesta is of central origin.

3. No corresponding effect could be demonstrated following the dosing or injection of acid.

4. The paretic ruminal musculature is still capable of normal contraction as shown by the reaction to feeding and the response to carbamylcholine.

5. The possible significance of alkalinity of the ingesta in the aetiology of ruminal stasis is discussed.

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