

## Destruction of Ascorbic Acid in the Rumen of the Dairy Cow.\*

C. A. KNIGHT, R. A. DUTCHER, N. B. GUERRANT AND S. I.  
BECHDEL.

From the Department of Agricultural and Biological Chemistry, and the Department of Dairy Husbandry, The Pennsylvania State College, State College, Pa.

Dutcher and coworkers,<sup>1</sup> Hart, Steenbock and Ellis,<sup>2</sup> and Hess, Unger and Supplee<sup>3</sup> presented evidence to show that the diet of a dairy cow influenced the antiscorbutic potency of the milk produced. Using guinea pigs to test the antiscorbutic potency of the ration which was fed and to assay the milk which was produced, each of these groups of workers found that milk obtained from cows on a vitamin-rich ration was definitely superior in antiscorbutic value to the milk derived from cows on a vitamin-poor diet. These findings, though widely accepted, were disputed by Hughes and coworkers,<sup>4</sup> who concluded from a series of experiments that the ration received by cows had no influence on the antiscorbutic property of their milk.

Since the development of chemical methods for the quantitative determination of the antiscorbutic factor, which was shown to be ascorbic acid, differences of opinion have arisen concerning the factors which have the greatest influence on the amount of vitamin C in milk. It is now generally agreed, however, that the vitamin C content of milk is independent of the season of the year and the ration of the cow.<sup>5</sup> This fact has led to the present investigation of the fate of ingested ascorbic acid in the cow.

A rumen fistula was made in a Holstein cow. Experiments were performed in which this cow was fed (A) 100 g (2,000,000 International Units) and (B) 150 g of synthetic ascorbic acid mixed with corn silage; 100 g of ascorbic acid were also placed directly in the rumen through the fistula opening.

Similar results were obtained in all of the experiments. No increase was observed in the ascorbic acid values of the blood plasma and of the milk when compared with those values obtained while

\* Authorized for publication on March 19, 1940, as paper No. 963 in the Journal Series of the Pennsylvania Agricultural Experiment Station.

<sup>1</sup> Dutcher, R. A., Eckles, C. H., Dahle, C. D., Mead, S. W., and Schaffer, O. G., *J. Biol. Chem.*, 1920, **45**, 119.

<sup>2</sup> Hart, E. B., Steenbock, H., and Ellis, N. R., *J. Biol. Chem.*, 1920, **42**, 383.

<sup>3</sup> Hess, A. F., Unger, L. J., and Supplee, G. C., *J. Biol. Chem.*, 1920, **45**, 229.

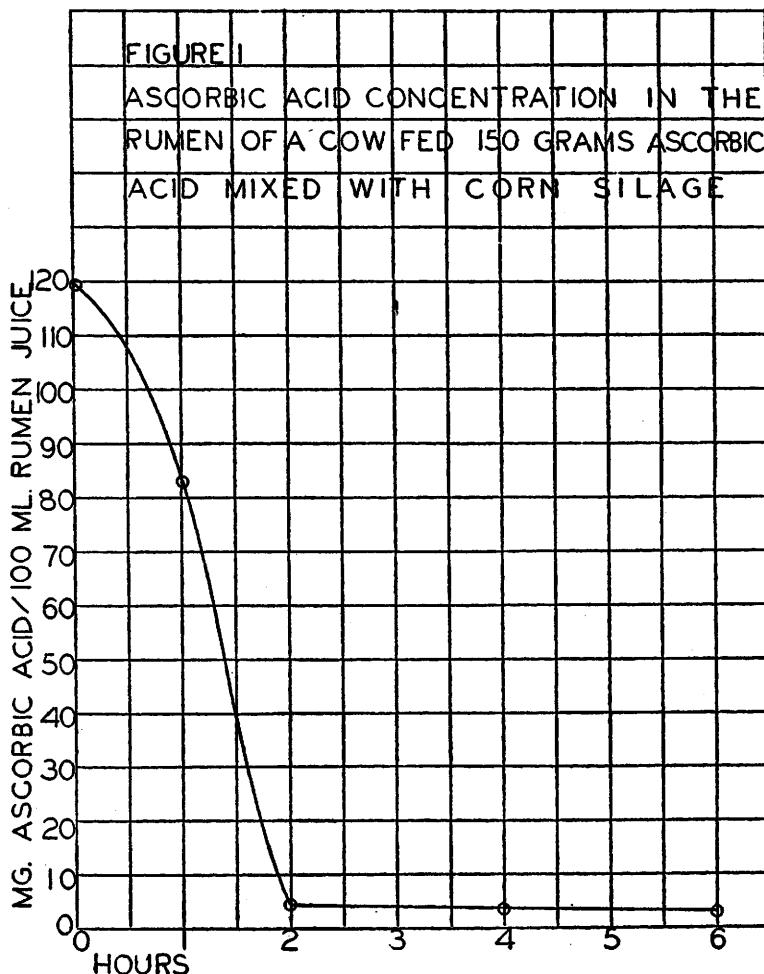
<sup>4</sup> Hughes, J. S., Fitch, J. B., and Cave, H. W., *J. Biol. Chem.*, 1921, **46**, L.

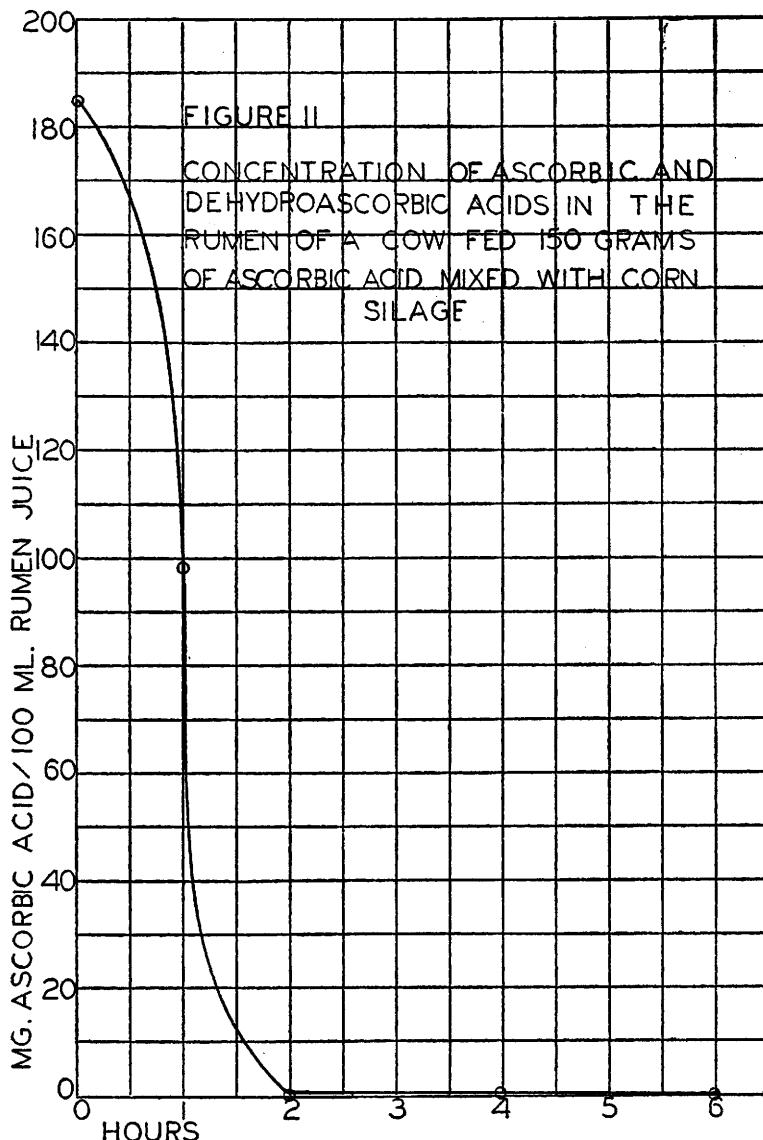
<sup>5</sup> Kon, S. K., *The Journal of Dairy Research*, 1938, **9**, 242.

the cow was on a standard ration unsupplemented with the vitamin. A slight increase was noticed in the amount of ascorbic acid found in the 24-hour sample of urine for the periods during which the vitamin was administered.

A rapid and pronounced destruction of ascorbic acid in the rumen was demonstrated by removal and analysis of samples of the rumen contents at regular intervals after the cow had been fed (Figs. 1 and 2). Ascorbic acid added to rumen contents *in vitro* and stored in a dark-glass, stoppered bottle at 39°-42°C disappeared at much the same rate as that of the *in vivo* experiments.

These results are not in accord with the conclusions of Riddell and





Whitnah<sup>6</sup> who suggested that the rapid disappearance of vitamin C from the rumen of a cow fed large amounts of green rye was due to a quick absorption of the vitamin.

In making the above analyses, both the indophenol titration and the Roe furfural method were employed.<sup>7</sup> The latter method was useful

<sup>6</sup> Riddell, W. H., and Whitnah, C. H., *J. Dairy Science*, 1938, **21**, 121.

<sup>7</sup> Roe, J. H., and Hall, J. M., *J. Biol. Chem.*, 1939, **128**, 329.

in detecting dehydroascorbic acid as well as the reduced form of the vitamin.

The authors wish to acknowledge the assistance of Dr. J. F. Shigley, who performed the fistula operation, and to express their appreciation for the generous supply of ascorbic acid furnished by Chas. Pfizer and Company, New York.

### 11365 P

#### Effect of Electrotonus on Accommodation in Nerve.

ABRAHAM M. SHANES. (Introduced by Robert Chambers.)

*From the Department of Biology, Washington Square College, New York University.*

Hill<sup>1</sup> has proposed an approach to the response of excitable tissues involving two processes, one a rise of the "local potential" and the other a change of threshold called "accommodation", the rates of which are represented by the time constants "k" and " $\lambda$ " respectively. Blair<sup>2</sup> has pointed out some theoretical inadequacies arising from investigations of the effects of electrotonus on rheobase and chronaxie (theoretically  $.693k$ ), but in the absence of similar studies on  $\lambda$ , the extent of such limitations is not clear. Consequently, the present investigation of  $\lambda$  was undertaken.

The technic described by Solandt<sup>3</sup> employing exponentially rising currents was used to determine the  $\lambda$  of the sciatic nerves of *Rana pipiens*. The same nonpolarizable electrodes, 2 cm apart, were employed to produce a 2-second electrotonus and to apply the exponential currents. Special precautions were taken to minimize residual and progressive effects. Most experiments were performed at 20°C.

The chief results obtained are summarized in the accompanying figure. The ordinate represents the relative change in  $\lambda$  (i.e., the ratio of  $\lambda$  during electrotonus,  $\lambda_e$ , to  $\lambda$  of the normal nerve,  $\lambda_n$ ) and in rheobase (i.e., the rheobase during electrotonus,  $V_e$ , divided by its normal value,  $V_n$ ), while the abscissa is the intensity of electrotonus ( $E/V_n$ ) in rheobases. It can be seen from the continuous

<sup>1</sup> Hill, A. V., *Proc. Roy. Soc. London*, Ser. B, 1936, **119**, 305.

<sup>2</sup> Blair, H. A., *Cold Spring Harbor Symposia of Quantitative Biology*, 1936, **4**, 63.

<sup>3</sup> Solandt, D. Y., *Proc. Roy. Soc. London*, Ser. B, 1936, **119**, 355.