The enzyme of the diplococcus acts energetically upon other bacteria, bringing about their dissolution. It acts upon *B. typhosus*, *B. coli communis*, *B. pyocyaneus*, *B. anthracis*, *M. catarrhalis*, and to a less degree and more slowly upon *Staphylococcus aureus*.

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On the supposed existence of efferent fibers from the diabetic center to the liver.

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To explain the causation of those forms of glycosuria which follow stimulation of the central end of sensory nerves and *piqûre* of the medulla, it is commonly believed that there is a diabetic center in the medulla from which efferent impulses are transmitted to the liver causing the glycogen in this organ to become so rapidly converted into dextrose that hyperglycemia and glycosuria follow. Since section of the vagi does not prevent these forms of glycosuria, it is thought that the efferent impulses travel by the upper portion of the spinal cord and the greater splanchnic nerves.

That increased production of dextrose by the liver is the immediate cause of the glycosuria, there is no doubt, but the evidence that it is by nervous impulses transmitted from the medulla to the liver along the above path that this hyperglycogenesis occurs is very meager.

The evidence *in favor* of such a view is as follows:

1. Puncture of the floor of the fourth ventricle does not cause glycosuria if the splanchnic (greater) nerves, or the upper thoracic spinal nerves, or the spinal cord above the first thoracic nerves be cut (Eckhard, Marc Laffont, etc.).

2. Irritation of the cervical spinal cord, or of the upper thoracic sympathetic ganglia causes glycosuria (Pavy, Schift).

Against such a view stands the fact that stimulation of the splanchnic nerves does not cause glycosuria (Cf. Pflüger).

As has been shown by us, and by other workers, the reducing power of the urine of dogs is, within certain limits, no index of the amount of sugar in the blood. Now, very little of the above

evidence is based on observations of the amount of sugar in the blood, this being assumed to be increased whenever the urine strongly reduces. We have accordingly undertaken a reinvestigation of the foregoing evidence but have examined the reducing power of the blood instead of that of the urine.

Of certain of our results, viz., those relating to the influence of nicotin and of lowered blood pressure on the blood sugar, we have already made preliminary communication.[1, 2]

In the present communication are reported the results which we have so far obtained on the changes in the amount of sugar (reducing substance) of the blood resulting from stimulation of the spinal cord at various levels, and from stimulation of the splanchnic nerves. The sugar analyses were performed by the method of Waymouth Reid.[3]

The following table gives the averages of the results so far obtained:

Nature of Experiment.	No. of Exps.	Blood Sugar in gm. Per cent. Before Stimulating. No. of Analyses Included in Averages.	Blood Sugar in gm. Per cent. After Stimulating. No. of Analyses Included in Averages.
Stimulation of peripheral end of one splanchnic nerve, the opposite splanchnic and the vagi being cut	6	0.133 (9)	0.145 (9)
Stimulation of cut spinal cord below the cervical region	7	0.163 (8)	0.160 (12)
Stimulation of lower cervical region of spinal cord.			
A. With cord cut	1	0.140 (2)	0.236 (2)
B. With cord uncut	2	0.189 (3)	0.276 (4)
C. With oxygen freely administered by Hirch's method[4]	2	0.140 (2)	0.157 (6)

It will be seen that no hyperglycemia is produced by stimulation of the splanchnic nerves, or of the spinal cord below the cervical region. In the cervical region, on the other hand, stimulation produces hyperglycemia except when oxygen is very freely

[1] Macleod and Dolley: Proceedings of the Physiological Society, *Journal of Physiology*, 1905, xxxii, p. lxiii.

[2] Macleod and Briggs: Proceedings of the Toronto meeting of the British Medical Association, *British Medical Journal*, Dec. 22, 1906.

[3] Reid: *Journal of Physiology*, 1896, xx, p. 316.

[4] Hirsch: Ueber Künstliche Atmung durch Ventilation der Trachea. Dissertation, Giessen (1905); ref., *Biophysikalisches Centralblatt*, 1905.

delivered into the trachea. By such administration it has been shown by Hirsch that the blood remains arterial even after the respiratory movements have been inhibited by curare. When the cervical spinal cord is stimulated, and especially when it is cut, the respiratory movements are very considerably interfered with so that a partial asphyxia is produced which may be the cause of the hyperglycemia.

The fact that stimulation of the cervical cord causes glycosuria cannot therefore be taken as a proof of the existence of efferent fibers which control the glycogenic function of the liver. Dyspnea may be the cause of the hyperglycemia in these cases.[1]

Regarding the other evidence, which is supposed to point to the existence of such fibers, we would point out that in all the experiments on which it is based (viz., cutting the splanchnics, or sympathetic chain, or certain nerve roots, or the spinal cord, there must have been induced by the operation, a great fall of blood pressure which, in the cases of dogs with vagal glycosuria, Dolley and the writer have shown usually to cause a marked depression in the reducing power of the urine (*loc. cit.*).

Conclusion. — When every precaution is taken to prevent asphyxia we have been unable, so far, to demonstrate the existence of any efferent fibers whose stimulation causes hyperglycemia.

[1] Underhill (*The Journ. of Biol. Chem.*, 1905, i, p. 113), explains the hyperglycemia produced by the administration of certain drugs on the same basis, viz., that they produce dyspnea by an action on the respiratory center.