kidney extract. In each case it was found that hemolysis was inhibited. The question therefore arises, why are kidneys suffused with blood as a rule more actively hemolytic than the bloodless organs? If their extracts are centrifuged, and all the solid particles, including the red cells, removed, it is found that the extracts are still deeply stained by hemoglobin. This is due to the destruction and solution of red cells, which is inseparable from the process of preparing the extract. The next step, therefore, was to determine the effect of adding red cell constituents to the bloodless organ extracts. This was accomplished by adding red cells to distilled water, and then bringing the solution to the strength of normal salt solution. Such a solution adds very markedly to the hemolytic power of the organ extract. Its manner of action seems to resemble that of complement, inasmuch as it is capable of breaking up the red cells only after a preliminary treatment with the organ extract.

Tumors were investigated in the same manner as the kidneys. It was found that the non-necrotic tumors are somewhat more hemolytic than are the kidneys, owing possibly to their blood content. They act, however, in other ways precisely like the latter, their action being diminished by the addition of serum and of white cells, and of being increased by the red cell extract.

Necrotic areas of tumors are extremely hemolytic, even up to dilutions of two to four hundred. This hemolytic activity is not affected by the addition of the blood components.

An experimental study was made of the action of a necrotic organ, by ligating the vessels and removing the organ after several days. The extract was hemolytic in a dilution of one in 6,000. It acted in other respects like the extract of necrotic tumors.

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The enzymotic properties of diplococcus intracellularis.

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The brief vitality of many of the cultures of diplococcus intracellularis is a point of differential importance. Many strains, grown on a favorable medium, unless transplanted to a fresh

medium, do not survive beyond two or three days. Cultures three days old show marked degenerations, and the latter increase rapidly with age until, at the end of five or six days, or even earlier, no normal cocci persist. As degeneration progresses, loss of staining power and disintegration ensue, until finally, staining capacity is lost and a formless detritus remains.

The changes in the diplococcus are associated with the action of an enzyme which brings about the disintegration. This enzyme does not exhibit the usual properties of a proteolytic ferment: it does not liquify gelatin or coagulated serum. The degree and rapidity of its action varies with its concentration; at least a heavy suspension of the cocci in salt solution, kept at 37° C., undergoes dissolution more rapidly and completely than a weaker suspension. The vitality of the cultures is associated with the degree of autolytic alterations in the suspensions: cocci in the weak suspensions survive longer than in the stronger ones. At lower temperatures — 2°C. — disintegration of the cocci either does not take place at all or progresses much more slowly. Under the latter conditions more cocci survive in the strong than in the weak concentrations, although even here the vitality is a brief one.

Potassium cyanide restrains the action of the ferment which tends to disintegrate the diplococci; after removal of the cyanide, dissolution sets in. Heating the diplococci to 65° C. prevents or reduces the dissolving power of the intracellular enzyme.

The brief vitality which the diplococcus exhibits, as grown upon the usual media, and in salt suspensions, is associated with a deficiency of calcium in the media. If the diplococcus is suspended in Ringer's solution it survives, in concentrated suspensions, for 15 days at least, and if it is grown upon serum-glucose-agar to which calcium carbonate has been added, the period of viability is considerably greater than this. The diplococcus suspended in Ringer's fluid and killed by heat (60° C.) or toluol, undergoes autolysis.

The enzyme acts upon the dead cocci — probably not upon the living germs. Diplococci killed by heat (50° to 55° C.) undergo autolysis; but when the cocci are killed by the addition of toluol autolysis is accelerated. A heavy suspension of the diplococci in salt solution, under toluol and kept at 37° C., may be disintegrated in four hours.

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