experiments. In 6 of the 10 positive experiments, there was an actual increase in the amount of reducing substance during the course of the experiment.

The results may be summarized as follows:

Mixture.	Average Loss of Reducing Substance.
Muscle-extract alone	− 0.096 gm. in 100 c.c.
Muscle-pancreas	− 0.225 gm. in 100 c.c.
Muscle-pancreas-adrenalin	+ 0.01 gm. in 100 c.c.

These experiments then show that the antagonistic action between adrenalin and pancreas, as schematized by Falta, Eppinger and Rudinger, may be demonstrated in vitro independently of any possible nervous influence. King in 1910 showed that a similar retarding influence upon the disappearance of reducing substance in muscle pancreas mixtures was exerted by thyroid extract.

The question as to whether there occurs in this reaction, a true glycolysis, or as Levene and Meyer hold, merely a condensation of the sugar molecule, is left untouched by these experiments. The antagonistic action of adrenalin and pancreas in regard to the disappearance of reducing substance in muscle extracts seems to be clearly demonstrated.

58 (667)

The characteristic course of the rise of blood pressure caused by an intraspinal injection of adrenalin.

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An intravenous injection of adrenalin causes a rapid steep rise of the blood pressure with a gradual fall. An intramuscular injection produces a similar effect. A subcutaneous injection either produces practically no effect or it causes a very slow rise which rarely exceeds fifteen millimeters. In recent years adrenalin was injected into the spinal canal in conjunction with some local anesthetic. The question as to the nature of the effect of these injections upon the blood pressure has to our knowledge never yet been investigated. On the basis of the generally accepted assumption that the absorption from the spinal canal into the circulation

is not very prompt we might expect that the intraspinal injection of adrenalin will have no stronger effect upon the blood pressure than that of a subcutaneous injection. We studied this question experimentally and may say at the outset that our results did not bear out this anticipation. Our experiments were made on six monkeys, using each monkey two or three times. The amount injected was either 1 c.c. or 1.5 c.c. of the commercial adrenalin. Most of the injections were made in the lumbar region; but in a few instances the adrenalin was injected in the thoracic region in the fifth intervertebral space. During the experiments the animals were under fairly profound anesthesia and we are unable to state whether the injection had any other effect besides the change in the blood pressure. But it is important to point out that even doses of 1.5 c.c. of adrenalin had no recognizable after-effects upon the animal.

The action upon the blood pressure was in most cases very characteristic. The pressure would begin to rise slowly but steadily, so that in a few minutes it would reach a maximum varying between 150 and 190 millimeters, and would then commence to go very gradually down. As a rule the entire course of the rise lasted longer than in intravenous injections, in some instances even longer than half an hour. The fall of blood pressure occurred so slowly at times that the original level was not reached during the entire time of observation, a fact which might be of considerable practical importance.

Twenty-one injections were given to these six monkeys at intervals; of these thirteen gave the typical rise described. In six cases the rise was preceded by a moderate fall (9–52 mm.) of short duration (½–4 minutes) and in one case this fall was the only effect of the injection. In the seven instances the injection brought on a rise similar to that of an intravenous injection but of longer duration. It is possible that in these cases part of the injection entered indeed into a vein.