malities with the exception of some neuronophagia at the rostral pole of one ganglion.

Recently Morgan and Goland[2] have presented evidence that tends to show that there are postganglionic parasympathetic fibers which arise in the nodose ganglion of the dog to pass to the heart. Heinbecker and O'Leary[3] failed to show any decrease in heart rate but normal results were obtained from the fibers responsible for certain motor effects in the lungs and duodenum of the cat. Ranson, Foley and Alpert[4] report observations which show no histological evidence that the nodose ganglion contains synapses between pre- and postganglionic parasympathetic neurons in the cat. Our observations in the cat give no support to the presence of postganglionic parasympathetic neurons situated in the nodose ganglion which control the heart. Final conclusions as to the presence of such neurons which may exert an effect on the respiratory and gastrointestinal tracts can not be reached from our observations.

6827

Acetylation Studies.

BENJAMIN HARROW, ABRAHAM MAZUR AND CARL P. SHERWIN.

From the Department of Chemistry, College of the City of New York, and Chemical Research Laboratory, Fordham University.

In an earlier paper[1] experiments were recorded as evidence for the belief that acetylation studies might prove of value in elucidating phases of intermediary metabolism. In this communication we record further experiments dealing with acetylation.

p-Aminobenzoic acid is partially acetylated by the rabbit forming *p*-acetyl-aminobenzoic acid, a substance which can be recovered from the urine. (Qualitative tests make it quite evident that some of the *p*-aminobenzoic acid is also eliminated in the form of its glycuronate salt; but this will be the subject of another communication.) Since carbohydrates—and possibly fats—are probably the

[2] Morgan, L. O., and Goland, P. P., *Am. J. Physiol.*, 1932, **101**, 274.

[3] Heinbecker, P., and O'Leary, J., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 506.

[4] Ranson, S. W., Foley, J. O., and Alpert, C. D., *Anat. Rec.*, 1933, **55** (suppl.), 33.

[1] Harrow, Power and Sherwin, PROC. SOC. EXP. BIOL. AND MED., 1927, **24**, 422.

source of the acetyl group furnished for the acetylation process, it became of interest to determine the effect of the injection of insulin upon the amount of acetylated product produced.

The results show that the injection of 0.5 units of insulin per kilo wt. raised the normal acetylation value of 20-25% to that of 44-47% of the original *p*-aminobenzoic acid injected. Although the rabbits showed individual variations in their ability to acetylate, insulin, in each case, practically doubled the output of the acetylated product.

We next determined what effect the simultaneous injection of insulin and reduced glutathione would have upon the output of the acetylated product. The belief is prevalent that the -SH group has an inhibiting action in so far as insulin activity is concerned. For example, Du Vigneaud[2] has shown that insulin can be inactivated if it is mixed with cysteine or reduced glutathione in a neutral solution in an atmosphere of nitrogen, because the blood-sugar of the rabbit does not drop when the insulin-glutathione (or insulin-cysteine) solution is injected. Instead of mixing our reduced glutathione and insulin *in vitro,* we decided to inject them separately, but simultaneously. Five mg. of reduced glutathione and 0.5 units of insulin (per kilo of body weight) were injected. The results showed an acetylation value of 23-34%, as compared with 44-47% when insulin alone was used.* These *in vivo* experiments confirm Du Vigneaud's *in vitro* results in tending to show that reduced glutathione inhibits insulin action.

Glutathione itself had no effect upon the amount of acetylated product produced. We must, therefore, regard the action of glutathione as being confined to its action on insulin.

The results point to the following conclusions: insulin markedly increases the acetylation process in the body; reduced glutathione does not affect it; but the combined action of insulin and glutathione inhibits the increased output of *p*-acetyl-aminobenzoic acid obtained when insulin alone is used. This supports the view that the -SH group has an inactivating effect upon insulin action.

---

[2] Du Vigneaud, *J. Biol. Chem.*, 1931, **94**, 233.

* We are indebted to Professor Du Vigneaud for certain details of his experiments.