due to the action of small amounts of thrombin, formed spontaneously from the prothrombin. It was not appreciated at the time that the product formed from the prothrombin was the principal impurity of the preparation. The thrombin produced a derivative of prothrombin which was without typical activity. It is likely that the presence of this inactive prothrombin derivative is responsible for the polydisperse appearance of the prothrombin preparations examined in the ultracentrifuge.[10]

Summary. When purified prothrombin was allowed to react with a small amount of purified thrombin there was loss in prothrombin activity. Comparison of the electrophoretic patterns of the prothrombin before and after alteration with thrombin showed that protein disappeared from the curve representing prothrombin and appeared as a new component with a lower electrophoretic mobility than that of prothrombin.

[10] Seegers, W. H., and Ware, A. G., *Fed. Proc.*, 1948, **7**, 186.

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Acetylcholine and Blood Sugar. (17396)

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Intravenous injection of acetylcholine (100-500 α/kg, i.v.) produces hyperglycemia (25-60%) in rabbits. High doses of acetylcholine (500-1000 α/kg, i.v.) induce convulsions and a marked increase in blood sugar, up to 200%.

The mechanisms of the hyperglycemia provoked by acetylcholine have been investigated in a series of experiments. The observations may be summarized as follows:

1. Atropine (0.25 - 1 mg/kg, i.v.) does not abolish the acetylcholine hyperglycemia

2. The adrenolytic drug SY 28 (α-naphthyl-methyl-ethyl-β-bromo-ethylamine HBr), in doses of 1 to 3 mg/kg, does not abolish the hyperglycemia induced by acetylcholine or epinephrine.

3. The anticholinesterase drug:dimethyl-carbamate of hydroxyphenyl-benzyl-trimethyl-ammonium (Nu-683) (0.1 - 0.3 mg/kg i.v.) sensitizes the animal very markedly to the hyperglycemic actions of acetylcholine.

4. Tetraethylammonium (20 mg/kg i.v.) protects against acetylcholine-hyperglycemia, if doses of acetylcholine which do not induce convulsions are injected.

5. After removal of both adrenal glands and treatment with desoxycorticosterone-acetate, rabbits do not present hyperglycemia after doses of acetylcholine which do not induce convulsions. Higher doses of acetylcholine inducing convulsions still provoke hyperglycemia.

6. Nembutal (Sodium-ethyl(-l-methyl-butyl) barbiturate) anesthesia prevents acetylcholine-hyperglycemia.

7. Control experiments show that no changes in blood sugar occur after injections of the same doses of atropine, tetraethylammonium SY 28 or Nu-683.

8. High doses of Nu-683 (0.5 mg/kg) induce hyperglycemia, which is also prevented by tetraethylammonium.

Conclusions. In rabbits, doses of acetylcholine which do not provoke convulsions induce hyperglycemia.

This increase in blood sugar is prevented by the synaptic blocking agent tetraethylammonium, by nembutal and by removal of both adrenal glands.

The acetylcholine-hyperglycemia thus is induced by adrenal synaptic stimulation, which increases epinephrine output.

The acetycholine and epinephrine hyperglycemia are not abolished by an adrenolytic agent (SY 28). The hyperglycemia induced by high doses of an anticholinesterase drug (Nu-683) is also prevented by tetraethylammonium.

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