Pacific Coast Section

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Effect of Administered Glucose upon Amino Nitrogen Content of the Blood.*

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The experiments here recorded arose from investigations into the influence of adrenalin on the amino nitrogen content of the blood. It was recently shown by Davis and Van Winkle[1] that the amino-acid-lowering property of insulin, which we have been studying for some years, is directly due to adrenalin, secreted in response to the administered insulin. Part of the proof is based upon the observation that insulin is without effect on the blood amino acids in adreno-demedullated rabbits. However, the administration of adrenalin induces in these, as well as in normal animals, the characteristic lowering.

Since the response seems to be due specifically to adrenalin, it is possible to infer, under appropriate conditions, that a lowering in blood amino acids is indicative of a discharge of this hormone. We have now made use of this hypothesis to test the response of the adrenal medulla to administered glucose.

To 15 normal students (13 males, 2 females) from 16 to 30 years of age, who volunteered as subjects, glucose was administered after fasting periods of 15 hours. To 4 others, who served as controls, water was given in quantities comparable to those received with the glucose by the experimental subjects. Blood sugar (Folin) and amino nitrogen (Danielson) determinations were made at zero, one, 2 and 4 hours after the first administration of glucose. Glucose was given at zero, one and 2 hours. The results are summarized in Table I.

* This investigation has been made with the assistance of a grant from the Committee on Therapeutic Research, Council on Pharmacy and Chemistry, American Medical Association.

[1] Davis, B. L., Jr., and Van Winkle, W., Jr., *J. Biol. Chem.*, 1934, **104**, 207.

TABLE I.
Hypoaminoacidemia induced by glucose.

Hrs. after commencement		Glucose administered, gm.	Blood-sugar content, % of initial value		Amino N content, % of initial value		Subjects
			Range	Aver.	Range	Aver.	
0		100	100 (98-110)§	100 (104)§	100 (7.0-8.1)§	100 (7.6)§	
1		50					10
2		25	83-113	92	69-84	77	
4			72-105	91	66-77	70	
0		50	100	100	100	100	
1		25	83-134	100	83-101	89	
2		25	79-104	91	83- 93	88	5
4			83-98	89	77- 93	87	
0			100	100	100	100	
1	Controls		100-106	103	93-103	98	
2	No glucose		97-106	102	99-104	103	4
4			103-104	104	96-102	99	

§ Initial absolute values are given in parenthesis.

As a simple explanation of these observations we suggest the following mechanism. Insulin is first secreted in response to the administered glucose. This is indicated by the absence of sustained hyperglycemia,† the evident trend towards hypoglycemia after one or 2 hours, and is in accord with the now abundant evidence of many investigators. The secreted insulin then evokes a discharge of adrenalin, the agent immediately responsible for lowering the amino acid content of the blood. The evidence pertaining to the discharge of adrenalin in response to insulin is presented in numerous papers cited in our earlier report.[1] Since the reduction in amino acid content is not attended by a low blood sugar, we conclude that insulin, *per se,* in the absence of appreciable hypoglycemia is able to stimulate the adrenal medulla.‡ We propose, eventually, to

† In 2 instances, several blood-sugar determinations were made during the first hour after the initial dose of glucose was given; in confirmation of many similar observations recorded in the literature, there was a very marked but transitory hyperglycemia.

‡ It is pertinent to mention that in the course of other studies by Daniels and Luck (hitherto unpublished) glucose was given as in the experiments recorded here. The total quantities administered were larger and were divided into 5 doses, administered over 4-hour periods. The initial hyperglycemia was more prolonged, the amino acid concentration fell, and so also did the blood inorganic phosphorus. The phosphate decrease, which adrenalin is able to induce, is itself indicative of adreno-medullary stimulation and supports the conclusions drawn from our amino acid studies.

test this hypothesis by the administration of glucose to adrenalectomized animals.

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Stability of Streptofibrinolysin.*

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Quantitative studies of antifibrinolytic immunity are made difficult by the multiplicity of reacting (or conditioning) factors in sterile† streptococcus filtrates and by the non-applicability of simple

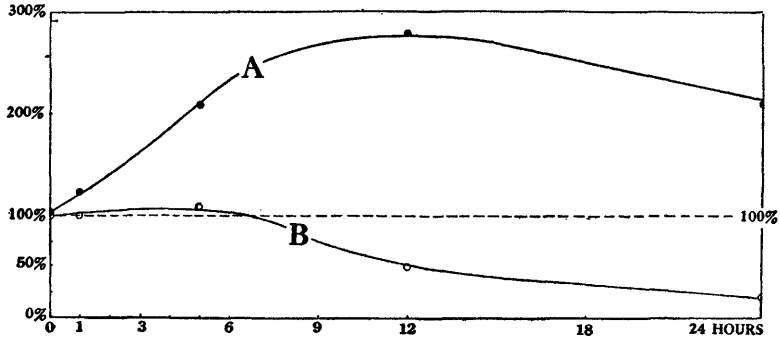

FIG. 1. *Serological Exaltation of Fibrinolytic Titer.*

Chamberland (L-3) filtrates from 24-hour broth cultures of *S. hemolyticus* were diluted with an equal volume of 0.25% to 0.5% normal horse serum, control dilutions being made with the same volume of 0.8% NaCl-solution. The dilute filtrates were then incubated at 37°C. At various times during this incubation, samples were titrated for their antihuman fibrinolytic function.

The lytic unit selected for these titrations was the minimum volume of the original filtrate that would cause demonstrable lysis of the serum-free human-fibrin clot, by the end of one hour, materials, dilutions, etc., being identical with those used by Tillett and Garner.[1] The original titer of the filtrate is recorded as 100%.

A. 50% Streptococcus filtrate A containing 1:600 normal horse serum. (Composite data from two titrations.)

B. Control test with serum-free filtrate.

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† These filtrates gave no demonstrable growth in veal-infusion broth or on routine 5% rabbit-blood agar.

[1] Tillett, W. S., and Garner, R. L., *J. Exp. Med.*, 1933, **58**, 485. Van Deventer, J. K., PROC. SOC. EXP. BIOL. AND MED., 1934, **32**, 366.