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Factors Affecting Human Potassium Tolerance.

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A relation of the adrenal cortex to potassium metabolism is substantiated by the following facts. In experimental adrenal insufficiency,[1-4] and in Addison's disease[5, 6] there is a definite rise in plasma potassium. This high potassium can be lowered by injection of adrenal cortex extract.[2, 6] In adrenal insufficiency there is an increased susceptibility to exogenous potassium,[7, 8, 9] which has been quantitatively contrasted with normal potassium tolerance in experimental animals.[10] The application of potassium tolerance determinations in the human is here briefly described.

Solutions of potassium salts were administered by mouth, the dose being 10 mg. or 20 mg. of K per pound of body weight. Duplicate determinations of potassium were done for plasma and whole blood taken from the finger or ear immediately before taking the drink, and at intervals of 30 min., 1 hr., and 2 hr., thereafter. Simultaneous hematocrit readings enabled us to calculate the red cell potassium content. If similar amounts of potassium contained in food are taken, slower assimilation makes it necessary to continue the test for 5 hours.

Seventeen human potassium tolerance curves on 12 individuals have been completed to date. Since this preliminary paper does not permit detailed discussion of all the curves, a few typical ones only are shown.

The plasma K of normal individuals is not affected by ingestion of 10 mg. K per pound body weight (Graph 1). In Addison's disease, however, there is a rapid and very considerable rise in plasma K (Graph 2), much greater than can be accounted for by

1 Bauman and Kurland, *J. Biol. Chem.*, 1926, **71**, 281.

2 Zwemer and Sullivan, *Endocrinology*, 1934, **18**, 97.

3 Urechia, Benetato and Retzeanu, *Compt. Rend. Soc. Biol.*, Paris, **119**, 439.

4 Truszkowski and Zwemer, *Biochem. J.*, 1936, **30**, 1345.

5 Maranon, Collazo, Barbudo and Torres, *Arch. Med. Cir.*, 1934, **37**, 893.

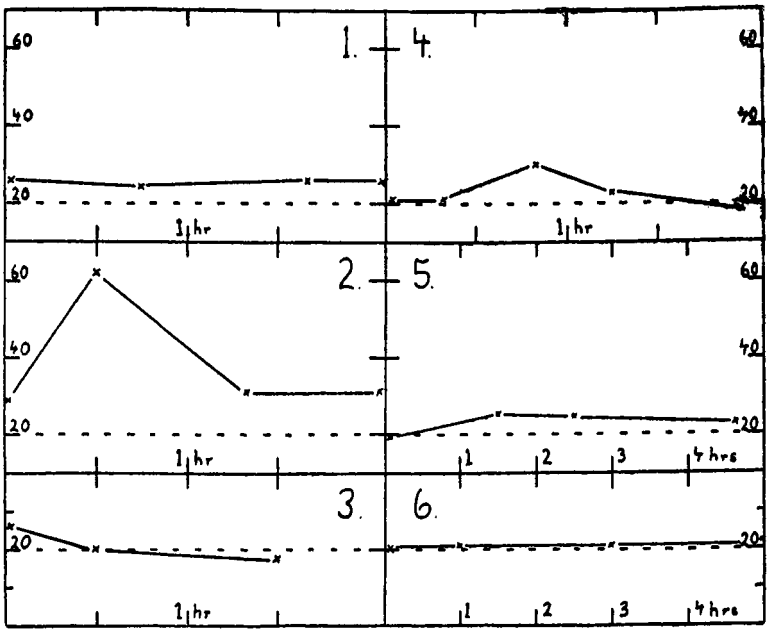
6 Allot, *Lancet*, 1936, **230**, 1406.

7 Zwemer and Truszkowski, *Science*, 1936, **83**, 558.

8 Allers, Nilson and Kendall, *Proc. Staff Meet., Mayo Clinic*, 1936, **11**, 283.

9 Wilder, Snell, Kepler, Rynearson, Adams and Kendall, *Proc. Staff Meet., Mayo Clinic*, 1936, **11**, 273.

10 Zwemer and Truszkowski, *Endocrinology*, 1937, **21** (Jan.), (in press).



EXPLANATION OF GRAPHS.

The mean of duplicate plasma potassium determinations is given in mg. per 100 ml. of plasma. Time is given in hours.

Graph 1. Plasma K of normal person remained unchanged after 10 mg. of potassium per pound body weight given by mouth as a solution of potassium salts.

Graph 2. Rise in plasma K of an Addisonian after a dose identical with that given above.

Graph 3. The 10 mg. per pound body weight dose given to an individual subject to attacks of petit mal did not increase plasma K.

Graph 4. A normal person had a transient rise in plasma K when given 20 mg. K per pound.

Graph 5. Eating of food containing enough K to give an approximate intake of 20 mg. K per pound body weight was followed by an increase in the plasma K of an individual with suspected adrenal insufficiency.

Graph 6. A similar meal to the same individual after treatment.

hemoconcentration. At the end of 2 hours both cell volume and plasma K are again approaching the initial values, but the red cell K content has increased, the increase being perhaps compensatory. Another graph illustrating the 10 mg. dose is from a patient subject to attacks of petit mal (Graph 3). This showed the peculiar phenomenon of a drop in plasma K with a rise in the red cell K content. The constancy of the hematocrit determinations would suggest that the red cell K rise was not due to a shift of fluid between plasma and cells.

Twenty mg. of K per pound body weight gives a definite rise in plasma K in normal individuals (Graph 4). In a few cases in which the test was repeated on the same individual, the character of the

response appeared to be constant. In certain allergic conditions the initial plasma K was found to be higher than normal and the return to the initial level after the 20 mg. dose was somewhat delayed. The relation of the adrenal cortex to allergy has been discussed elsewhere,[11] and in view of its general application we are continuing these studies, in cooperation with the Department of Dermatology.

Although the plasma K of normal individuals does not appear to rise after an average meal, a patient with suspected adrenal insufficiency showed a definite rise in plasma K following a test meal consisting of steak, french fried potatoes and raw cabbage (Graph 5). These were chosen because of their relatively high K content. After a week's rest and treatment with adrenal cortex extract the patient was improved subjectively and a similar test meal produced no rise in plasma K (Graph 6). In contrast to the previous experiment, red cell K fell during the digestion period.

The 5-hour feeding experiments are less satisfactory than the administration of a K drink, both because of the time necessary to complete the test, and of the uncertain K content of the food. They might, however, be used for preliminary tests in certain cases. We feel that either test would be safer and quicker than sodium chloride deprivation as described by Loeb.[12] Any untoward effect can immediately be treated by injection of adrenal cortex extract, which we have found to lower blood K within 15 minutes of injection.

In conclusion, we suggest that the K tolerance test as outlined be used in the diagnosis of corticoadrenal insufficiency.

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11 Wolfram and Zwemer, *J. Exp. Med.*, 1935, **61**, 9.

12 Loeb, *J. A. M. A.*, 1935, **104**, 2177.