CLIX. CARBOHYDRATE METABOLISM DURING EXPERIMENTAL HUMAN SALT DEFICIENCY

By ROBERT ALEXANDER McCANCE

From the Biochemical Laboratory, King's College Hospital, London

(Received 16 June 1937)

MICHELSSEN [1933] reported that high blood sugars accompanied the salt deficiency which could be produced in rabbits by diuretin. Figures of 0.222 and 0.246 % were obtained as against the normal 0.098 and 0.102 %. Similar results have been obtained by Wilkinson [1937]. It was decided, therefore, to investigate this matter in human subjects. Five subjects, three of whom have already been described, were made salt-deficient by diet and sweating [McCance, 1936, 1, 2]. The subjects were all adult males in good physical condition. Glucose tolerance tests were carried out when they were salt-deficient. The control tests were made either before any deficiency had been produced or at least a week after it had terminated. In some subjects the control and the experimental tests were made in the morning after an overnight fast, in others both tests were made in the afternoon after the omission of lunch. The dose of glucose was 50 g. by mouth. Capillary blood was taken and the glucose in duplicate portions determined by Folin and Wu's method after precipitation of the whole blood proteins by sodium tungstate and sulphuric acid.

The results are shown in Table I. In comparison with Michelsen's results the changes are small, but it will be observed that in each individual during salt

Table I. *Blood sugar curves in health and during salt deficiency*

Subject	Condition	Fasting Glucose %	Minutes after 50 g. of glucose			
			30 Glucose %	60 Glucose %	90 Glucose %	120 Glucose %
R. B. N.	Normal	0.095	0.128	0.120	0.110	0.112
	Deficient	0.101	0.151	0.149	0.110	0.134
R. A. M.	Normal	0.099	0.136	0.130	0.090	0.091
	Deficient	0.113	0.178	0.125	0.138	0.105
D. W.	Normal	0.095	0.112	0.141	0.135	0.126
	Deficient	0.120	0.152	0.133	0.107	0.131
J. T. B.	Normal	0.101	0.129	0.099	0.080	0.092
	Deficient	0.105	0.186	0.163	0.137	0.118
R. M. L.	Normal	0.090	0.106	0.084	0.111	0.094
	Deficient	0.100	0.122	0.098	0.105	0.109
Average „	Normal	0.096	0.122	0.115	0.105	0.103
	Deficient	0.108	0.158	0.134	0.119	0.119
Difference between averages		0.012	0.036	0.019	0.014	0.016

deficiency (1) the fasting blood sugar was higher than in health, (2) the blood sugar 30 min. after the dose of glucose was higher, (3) the blood sugar 2 hr. after glucose was also higher. The averaged results show higher blood sugars at all times during salt deficiency.

DISCUSSION

It has previously been shown that in man salt deficiency leads to a disordered nitrogen balance [McCance, 1936, 1, 2]. The present results suggest that it also interferes with carbohydrate metabolism. The changes are small in comparison with those in the rabbit, but they are in the same direction and contrary to those produced by adrenal cortex deficiency. It is therefore just possible that they are produced by over-activity of the cortex brought about by the salt deficiency. It has been suggested that these small abnormalities might be due to carbohydrate starvation since some of the subjects suffered considerably from nausea and found it difficult to take a full diet. This is not the case, because the diets of three subjects were weighed and are known to have contained 300–400 g. of carbohydrate per day. The other two subjects did not eat their normal quantities of protein and fat, but their food was closely supervised and their carbohydrate intake is known to have been adequate. Another possible explanation of the results “after glucose” is that during salt deficiency the sugar was being absorbed into a reduced volume of circulating blood. This, however, would not explain why the fasting values were higher during deficiency and would certainly not explain the animal findings. It might even be considered that since the method employed does not give “true” sugar values, the changes in the blood might not be changes in the concentration of glucose. It is believed, however, that the changes measured were really due to glucose because (a) although the salt-deficient blood contained more red cells per $\mu\text{l.}$ than the normal blood, it can be calculated from the data of Herbert & Bourne [1930] that the change in the concentration of glutathione per 100 $\mu\text{l.}$ would not have been nearly enough to account for the changes observed; (b) the increased rise in the blood sugar which followed the administration of glucose in four out of the five subjects may be assumed to have been due to a change in glucose concentration.

SUMMARY

Glucose tolerance tests have revealed small abnormalities in carbohydrate metabolism during experimental human salt deficiency.

The author is indebted to Dr R. D. Lawrence for suggestions, to Mr H. R. Millar for his assistance with the technical work, and to the subjects for their wholehearted co-operation.

REFERENCES

- Herbert & Bourne (1930). *Biochem. J.* **24**, 299.
- McCance (1936, 1). *Proc. roy. Soc. B*, **119**, 245.
- (1936, 2). *Lancet*, i, 643, 704, 765, 823.
- Michelsen (1933). *Arch. exp. Path. Pharm.* **173**, 750.
- Wilkinson (1937). Unpublished observations.