INTRAVENOUS GLUCOSE TOLERANCE IN KWASHIORKOR AND MARASMUS*

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The clinical difference between the extremes of kwashiorkor and marasmus is considered to be due to the protein-calorie ratio of the dietary intake. The diet of a child with kwashiorkor is protein deficient with an adequate or near adequate caloric intake (mainly as carbohydrate) whereas the diet of the marasmic infant is deficient in both proteins and calories.[1] The possibility that the higher carbohydrate intake of the child with kwashiorkor may account for the differences in clinical picture, e.g. fatty liver, has been considered.[2] However, comparatively little has been done on the handling of a glucose load in these children. Oral glucose-tolerance tests have been performed,[3] but since malabsorption is a feature of malnutrition[4] the interpretation of the results obtained is difficult.

In the present study glucose tolerance has been measured by the technique of rapid intravenous injection of glucose. Glucose in a dose of 1 G/kg. body weight was injected as a 25% solution within 2 minutes. Capillary blood was obtained before (fasting) and 5, 10, 20, 30, 45, 60, 90, and 120 minutes after injection. Blood sugar was estimated by the Somogyi-Nelson technique.[5, 6]

Blood-glucose disposal, measured by the rate of fall of glucose after an intravenous load, has been related to either the total blood-glucose concentration or to the increment above the fasting value.[7-12] The present data have been analyzed in terms of the total blood-glucose concentration. A semi-logarithmic plot of such data permits the determination of a rate constant (k) which graphically describes a straight line for the first 60 minutes of the test. The constant (k) expresses the fraction of glucose which disappears in one minute.

Standard values for normal adults vary from 1·67 - 1·8%/min.[7, 9, 12] Adult diabetics are usually below 0·95%/min.[12] and normal newborn infants 1·03%/min.[13] No published standards exist for older infants and children. Table I summarizes the data obtained:

Group I—normal controls. All were normal weight for age. The rate constant of glucose disappearance averaged 2·41%/min.

Group II—children suffering from kwashiorkor. All were oedematous, hypo-albuminaemic and underweight for age. The mean rate constant of glucose disappearance in this group is 1·55%/min. This group was further subdivided into two groups. Group IIa consisted of 15 kwashiorkor children on whom the

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TABLE I. INTRAVENOUS GLUCOSE TOLERANCE

Group	No. of patients	Fasting blood sugar	Mean blood-sugar values (mg./100 ml.) Minutes after injection 5	10	20	30	45	60	90	120	Kt %/min.
I	5	83	378	322	253	208	147	102	70	76	2·41
II	20	82	345	305	261	227	188	153	91	67	1·55 (±0·73)
IIa	15	86	351	312	266	230	190	153	83	61	1·61 (±0·78)
IIb	5	67	329	286	250	219	183	152	112	81	1·38 (±0·62)
III	10	72	335	289	229	175	115	76	67	72	2·82 (±0·64)
IV	4	77	291	268	225	199	158	120	76	48	1·66

tolerance test was performed after at least 3 days on milk feeds with added sugar. IIb consists of the other 5 cases who were not only fed for 3 days but received 50 G of glucose by intravenous infusion on each of 3 days preceding the tolerance test.

Group III—children with nutritional marasmus. All were underweight for age and had hypo-albuminaemia, but none had oedema. The mean rate of blood-glucose disappearance was 2·82%/min.

Group IV—children with kwashiorkor on whom combined intravenous glucose- and insulin-tolerance tests were performed. The insulin was given in a dose of 0·25 units/kg. body weight. The mean rate constant of glucose disappearance for this group was 1·66%/min.

All the children were non-White and the groups are of comparable ages.

Results

The salient points of the results are:

1. Normal infants have a faster rate of glucose disappearance than normal adults.
2. There is no significant difference between normally nourished and marasmic children. Marasmic children deal with a glucose load as efficiently as well-nourished children.
3. There is a significantly slower rate of glucose disappearance in kwashiorkor than in both normal and marasmic children. This does not appear to be a manifestation of 'starvation diabetes' since adequate doses of intravenous glucose before the tolerance test failed to alter the rate of glucose disappearance significantly.
4. The slow rate of glucose disappearance in kwashiorkor is not improved by exogenous insulin.

Conclusions

Children with kwashiorkor are thus unable to dispose of an exogenous glucose load as efficiently as normal or marasmic children. The data do not provide an answer as to why this is so, but two likely possibilities are suggested.

Gross fatty infiltration of the liver is a striking feature of kwashiorkor, but not of marasmus. The poor glucose tolerance of the former may be a reflection of the inability of a malfunctioning liver to take up and store glucose.[14]

Muscle biopsy studies in kwashiorkor have shown diminished activity of several of the enzymes concerned in glucose metabolism.[15] The poor tolerance may be due to diminished peripheral uptake.

Failure or imbalance of hormonal control may also be involved. Diminished insulin production seems unlikely since exogenous insulin failed to improve the rate of glucose disappearance. Growth hormone excess in kwashiorkor and not in marasmus also seems unlikely since failure of growth is common to both conditions.

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