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Sugar metabolism in obese children

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In obese children the glucose tolerance curve results in high blood sugar values. A pathologic rise of the serum insulin level may occur even with normal blood sugar curves and the insulin levels tend to be higher in obesity of long standing. In obese children of families with diabetes, post-loading insulin levels were comparatively lower. It is suggested that this weaker insulin response is related to a prediabetic state.

In obese persons a glucose load is often followed by a high, protracted blood sugar curve, usually accompanied by high blood insulin levels [1, 7]. Without diabetic affliction, the phenomenon is closely linked to obesity [8], and the abolition of obesity will lead to the normalization of carbohydrate metabolism [3].

The present paper reports on a study of the incidence of pathologic blood sugar curves after a glucose load in obese children and of the relationship of the changes in the blood sugar insulin levels.

Material and Method

A total of 32 patients, 16 boys and 16 girls, ranging in age from 5 to 15 years was studied. Their body weight as compared to body length was in all cases above the 97 percentile; it exceeded by 52% the mean weight normal for body length.

The patients were divided in two groups according to the duration of obesity: 2 to 5 years and more than 5 years. There were 16 cases in each group. The family history revealed diabetes in the relatives of 9 children. In two cases one of the parents, in two a sibling was diabetic; in two cases there were diabetics among both the paternal and the maternal relatives and in three cases among the grandparents.

The children received a glucose load of 1.75 g per kg of body weight on an empty stomach. Blood glucose and insulin levels were determined before and 60, 120 and 180 minutes after the load, blood sugar from capillary blood by the orthotoluidine blue method, insulin by the Insulin Radio-immuno-assay Kit (Amersham) based on the double antibody separation method of Morgan and Lazarow [6] and Hales and Randle [5].

According to Yalow and Berson [9, 10], the normal blood insulin value of a 70 kg adult ranges from 0 to 66 μU/ml after fasting, and amounts to 139 ± 14 (18 to 342) and 106 ± 10 (21 to 233) at 60 and 120 min, respectively, after the oral administration of 100 g of glucose. Guthrie et al. [4] found the following figures in 40 healthy children: fasting, 0 to

TABLE I

Duration of obesity	Blood sugar curve			Insulin curve	
	normal	slightly pathologic	pathologic	normal	pathologic
2 to 5years	3	7	1	2	9
More than 5 years	3	4	5	—	12

TABLE II

	Normal	Slightly pathologic	Pathologic
Blood sugar curve	1	4	4
Insulin curve	3	—	5

42 μU/ml; 60 minutes after the load, 20 to 105 μU/ml; and at 120 minutes, 20 to 89 μU/ml. Control blood sugar values after loading and their scattering from the 3rd to 97th percentile were determined in 200 healthy children.

The blood sugar curve was arbitrarily considered pathologic if compared to the data of GUTHRIE et al. at least two of the values measured at 60, 120 and 180 min, surpassed the 97th percentile. If two of the values were higher than the 90th percentile, the curve was considered slightly pathological. An insulin level displaying an increasing tendency at 60 minutes after loading, or a value higher than 100 μU/ml was regarded as pathological.

RESULTS

The blood sugar and insulin curves after loading were determined in the first group of 23 obese children without diabetic relatives. Results are seen in Table I. The second group (9 children with diabetic relatives) in view of the small number of cases has not been subdivided according to the length of the history. Pertaining results are seen in Table II. In a case the insulin curve was so irregular that it did not fit into the above classification: the initial value of 270 μU dropped after loading by 100 μU/ml without a further rise.

The blood sugar and insulin curves are shown in Tables III, IV and V. Table III contains the data classified according to the duration of obesity of children with no familial diabetes. In their case the increase in the insulin level was related to the duration of obesity; if it had started not more than 5 years ago, the rise was less marked.

In Table IV the data of the same children are classified according to the character of their blood sugar curve. In cases with a pathologic blood sugar curve the rise of the insulin level was greater than in the other two groups.

Table V illustrates the blood sugar and insulin curves of obese children with diabetic relatives in comparison with the overall values for the other

TABLE III

Blood sugar and insulin levels after glucose load *vs.* duration of obesity (no familial diabetes)

Number of cases	Duration of obesity	Blood sugar level, mg/100 ml			
		Fasting	60 min	120 min	180 min
11	2 to 5 years	92±5	147±6	130±5	113±4
12	More than 5 years	101±8	156±13	132±12	118±9
		Insulin level, μU/ml			
11	2 to 5 years	40± 7	152±29	164±15	111±25
	More than 5 years	58± 7	248±15	192±19	184±24

TABLE IV

Blood sugar and insulin levels *vs.* the type of blood sugar and insulin curves

Number of cases	Type of blood sugar curve	Blood sugar level, mg/100 ml			
		Fasting	60 min	120 min	180 min
6	Normal	88± 5	117± 6	130± 4	101± 2
11	Slightly pathologic	105± 5	155± 9	127± 4	113± 5
6	Pathologic	92± 7	176±14	161±10	134±12
	Type of insulin curve	Insulin level, μU/ml			
6	Normal	54±12	165±47	140±40	160±30
11	Slightly pathologic	44± 9	169±25	146±23	110±20
6	Pathologic	67± 9	273±14	248±39	223±39

TABLE V

Blood sugar and insulin levels after glucose load in children with and without diabetic relatives

Number of cases	Familial diabetes	Blood sugar level, mg/100 ml			
		Fasting	60 min	120 min	180 min
9	yes	86±13	171±27	149± 9	123± 7
23	no	97± 6	152± 8	131± 7	115± 6
		Insulin level, μU/ml			
8	yes	50± 7	118±33	160±34	128±42
23	no	49± 7	220±22*	178±17	147±24

*Significant difference, $p < 0.05$.

group. There was no significant difference in the blood sugar curves of the two groups.

On the other hand, in the group of obese children with diabetes in the family, the insulin curve was flatter, and the rise in insulin level at 60 minutes significantly less pronounced and protracted. The fasting insulin level in these cases was near the upper normal limit.

Discussion

The majority of obese children displayed an abnormal blood sugar tolerance curve. In cases with no diabetes in the family the higher insulin level is apparently a more sensitive indicator of the pathological metabolism than the irregular blood sugar curve.

These data suggest that obesity of short duration is characterized by a high insulin level after glucose loading. The longer the duration of obesity the more significant will be the rise in insulin secretion. According to these observations, normal blood sugar curves may be accompanied by a pathological rise of the insulin level.

More insulin is presumably needed for the maintenance of a normal blood sugar curve in an obese than in a healthy child. The impaired sugar metabolism is balanced with enhanced insulin production but this must not yet lead to diabetes. This is not a unique phenomenon, since some other diabetogenic effects are also compensated by most patients. Thus, despite of the contra-insulin effect, only one fifth of the patients with acromegaly develops diabetes, perhaps because one fifth of the population are diabetic gene carriers.

The situation is different in the case of obese children with diabetes in their family. In this case the rise in the blood sugar curve is accompanied by a comparatively flat insulin curve. Drash [3] reported on similar observations in obese children in whom chemical diabetes was assumed in view of a steep blood sugar curve but their insulin level was relatively low.

Juvenile diabetes is characterized by a reduced or lacking insulin secretion. According to Cerasi and Luft [1] even the prediabetic state is characterized by a reduced insulin response to the rise in blood sugar.

The weak insulin response observed in obese children might also point to a relative deficiency in insulin secretion.

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