BLOOD LACTATE BEHAVIOR AFTER GLUCOSE LOAD IN DIABETES MELLITUS

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The increase in blood lactate concentration after an oral glucose load in normal subjects is well known 8. 14. On the contrary, conflicting data on blood lactate behavior in diabetes mellitus have been reported, probably in relation to different degrees of metabolic control and to residual B-cell function 8, 14, 30. The increase in lactate concentration after glucose load depends essentially both on inhibition of splanchnic extraction and increase in hepatic and muscle production of lactate 8, 9, 10, 25, 30. The peripheral behavior of blood lactate therefore represents an indirect evidence of both tissue glucose metabolism and gluconeogenesis inhibition.

The aim of the present study was to evaluate the relationships between the increments in blood lactate, plasma glucose, insulin (IRI) and C-peptide (IRCP) concentrations, during the first hour of an oral glucose load, in normal subjects and in non-insulin-dependent (NIDDM) and insulin-dependent (IDDM) diabetics.

MATERIALS AND METHODS

An oral glucose tolerance test (OGTT, 100 g) was performed in 32 subjects (tab. 1): twelve normal subjects (10 males and 2 females), aged between 16 and 58 years, within 10% of their ideal body weight (IBW) according to Metropolitan Life Insurance Company tables; sixteen non-insulin-dependent diabetic subjects (8 males and 8 females), aged between 20 and 68 years, 13 obese and 3 with normal body weight; and four insulin-dependent diabetic subjects (3 males and 1 female), aged between 15 and 30 years, with normal body weight.

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Glucose tolerance was evaluated according to the National Diabetes Data Group criteria. In 12 NIDDM subjects the duration of diabetes ranged from 5 to 19 years; in the other 4, diabetes was diagnosed during their hospitalization. Eleven NIDDM subjects had been following a therapy consisting of oral hypoglycemic agents for 7 ± 2 years, i.e. sulfonylureas (6), biguanides (1), or combinations (4); the remaining 5 had been following a low carbohydrate diet (150-200 g/die). In IDDM subjects the duration of diabetes ranged from 1 to 12 years with intercurrent episodes of severe hyperglycemia (fasting blood glucose values above 12 mmol/l) and ketonuria; they had been on a regimen of short- and long-acting insulin mixtures twice daily for about four years (mean dose: 25 ± 15 IU).

All the patients hospitalized without hepatic or renal alterations were invited to maintain their normal physical activity. Moreover, normal subjects were placed on an unrestricted diet (about 250 g carbohydrate) for at least three days preceding the test; all the diabetic subjects had followed their previous low carbohydrate diet (150-200 g/die). Informed consent was obtained from all the subjects.

In the diabetic subjects the study was performed at 0800 after a 12h overnight fast and 12h after the last therapeutic administration. Blood samples were obtained by means of a butterfly needle inserted into an antecubital vein and kept patent by a saline solution at -15, 0, 15, 30, 45, 60, 90, 120, 150 and 180 min after the oral glucose load. Blood samples were drawn without stasis and collected in iced EDTA-containing test tubes; blood aliquots were immediately deproteinized by adding equal volumes of chilled HClO4 and neutralized with KOH. After centrifugation, plasma and perchloric extract were stored at -20 °C until assay.

Plasma glucose was measured by the enzymatic glucose oxidase method (Biochemia kit); plasma insulin (Dow-Lepetit kit) and C-peptide (Byk-Mallinchrodt kit) by radioimmunoassay. The intra- and inter-assay variations observed in the insulin and C-peptide assay were 2.4% - 5% and 5.9% - 7.9% respectively. Lactic acid was determined on perchloric extract by an enzymatic method. All samples were measured in duplicate within the same assay.

The incremental areas under the glucose, lactate, insulin and C-peptide curves during 60 min after glucose load (Δ0-60) have been calculated by the geometrical method of triangulation. Results were expressed as mean ± SEM. Student’s paired and unpaired t-test were used for the assessment of statistical significance; correlation between two variables was assessed by linear regression analysis.

RESULTS

Fasting plasma glucose concentrations were raised in NIDDM and IDDM patients: none had a fasting glucose value exceeding 8.3 mmol/l (150 mg/dl). There was no statistically significant difference between the fasting blood lactate level in diabetic subjects and controls. Basal C-peptide concentrations were significantly reduced in IDDM patients compared to controls and moderately elevated in NIDDM patients (tab. 1).

After oral glucose load (fig. 1) the increment of plasma glucose above the basal value was significantly higher in NIDDM subjects from 30 min onward.