

Height and Diabetic Neuropathy

The interesting and stimulating article by Gadia et al. (1) reports a correlation between tallness and the presence of impaired conduction and chronaxie in the lower extremities of diabetic patients that did not occur in non-diabetic people of similar height. In diabetic and non-diabetic subjects, the same hydrodynamic factors are present when venous pressure in the legs increases with height, which is directly related to the vertical distance from the right atrium down to the appropriate vein in the lower extremities. This increase occurs when the person is sitting or standing and is equal to the height of a column of blood extending from the ankle or knee, respectively, to the right atrium. Therefore, in patients with diabetes there must be a special sensitivity to the decreased capillary perfusion resulting from the increased venous pressure. I can think of no other physiological principle that might account for the eloquent results of the study by Gadia et al.

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REFERENCE

- Gadia MT, Natori N, Ramos LB, Ayyar DR, Skyler JS, Sosenko JM: Influence of height on quantitative sensory, nerve-conduction, and clinical indices of diabetic peripheral neuropathy. *Diabetes Care* 10:613–16, 1987

Glucose-to-Insulin Ratio in Intravenous Tolbutamide Test Predicts Long-Term Therapeutic Outcome in NIDDM

Dietary caloric restriction and sulfonylureas have been used to ameliorate hyperglycemia in non-insulin-dependent diabetes mellitus (NIDDM). However, many patients do not respond satisfactorily to these treatments and need insulin in their clinical treatment (1). Whether a long-term prediction of successful management with diet and sulfonylureas in NIDDM can be made by a simple clinical β -cell function test is unknown. To address this problem, we correlated plasma insulin response to an oral glucose tolerance test (OGTT) or an intravenous tolbutamide test (IVTT) before treatment with the modes of therapy employed at the 5th yr of follow-up in NIDDM patients. Thirty-four patients with NIDDM diagnosed according to the reported criteria (2), treated

without insulin or untreated, were admitted to our hospital for the study. After an OGTT and an IVTT, they were treated initially with diet alone or diet plus sulfonylureas according to their individual response. Thereafter, follow-up was made for at least 5 yr. Fasting plasma glucose (FPG) was monitored at our outpatient service once or twice per month. When their glycemic control became poor, patients were advised to be readmitted, and their response to diet plus sulfonylureas was re-evaluated. Institution of insulin therapy was considered when, without intercurrent illness, FPG could not be lowered below 140 mg/dl with diet plus glyburide or chlorpropamide up to 15 or 500 mg/day, respectively, under the surveillance of compliance to these treatments during initial admission or readmission. Before completing the follow-up, 8 patients moved to other hospitals and dropped out of the study. Within 0.5 yr of the follow-up, 4 patients were switched to insulin treatment. The number of patients on insulin thereafter was increased to 6 and 8 by the end of the 1st and the 3rd yr of follow-up. Finally, at the end of the 5th yr, among 26 patients who completed the follow-up, 10 patients (3 men, 7 women, aged 28–72 yr at entry) were on insulin, and they were classified as insulin-receiving (IR) patients. Duration of diabetes for the IR patients from initial diagnosis to the time they started to receive insulin was between 2 and 25 yr. Among the remaining 16 patients who managed either with diet alone or diet plus sulfonylureas throughout the study, 12 patients with FPG <140 mg/dl at >70% of the determinations in the 5th yr of follow-up were considered to be in good glycemic control. These 12 patients (7 men, 5 women, aged 28–66 yr) were defined as non-insulin-receiving (NIR) patients. Four patients with higher FPG than the above criteria were not included in the NIR group.

IR patients showed higher plasma glucose values than NIR patients with comparable insulin values during OGTT. In contrast, lower insulin responses to IVTT were seen in IR patients. To discriminate between individual patients in both groups, parameters obtained in both OGTT and IVTT were compared as shown in Fig. 1. Higher FPG and lower peak plasma insulin with the IVTT (mean \pm SD 12.0 ± 6.4 vs. 26.8 ± 15.5 μ U/ml, $P < .05$) were seen in IR patients. Peak plasma insulin and a ratio of incremental insulin area above the basal level to the corresponding incremental glucose area (IIA/IGA) were comparable. Individual values of peak plasma insulin with IVTT and FPG showed considerable overlap despite significant difference between the mean values of both groups. A ratio of FPG to peak insulin with an IVTT gave a clear separation of individual patients with different therapeutic outcomes; i.e., all but 1 IR patient had values of this parameter >11, and none of the NIR patients exceeded 11 (Fig. 1E).

The usefulness of measurements of basal and glucagon-stimulated levels of C-peptide to differentiate diabetic patients requiring insulin therapy from those who do not has been suggested (3,4). However, in these studies, long-term follow-up for outcome of the selected