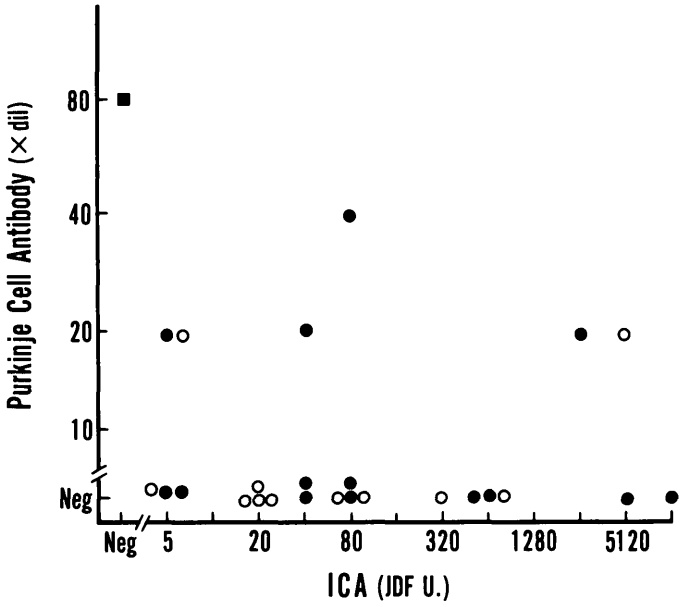

Figure 1—*Correlation between the titer of ICA and the titer of autoantibodies to glutamic acid decarboxylase–containing Purkinje cells in 14 patients with ICA$^+$ IDDM (●), 11 patients with ICA$^+$ NIDDM (○), and 1 patient with stiff-man syndrome (■).*

antibody and ICA did not show any positive correlation (Fig. 1).

In a previous report (2), anti-GAD antibodies (the 64K antibody) in IDDM patients, which reacted with GABA-ergic neurous, rarely recognized denatured GAD on Western blotting analysis. In this study, an immunohistochemical approach that used unfixed and undenatured specimens was adopted to examine the reactivity of anti-GAD antibody to its target antigens. No association between ICA and Purkinje cell autoantibody was observed in terms of positivity and titers in IDDM and stiff-man syndrome, suggesting that the two antibodies are distinct from each other.

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IDDM, INSULIN-DEPENDENT DIABETES MELLITUS; GAD, GLUTAMIC ACID DECARBOXYLASE; ICA, ISLET CELL CYTOPLASMIC ANTIBODIES.

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Glycemic Index Versus Glycemic Response

Nonsynonymous terms

Rasmussen et al. (1) found that most of the variation of glycemic responses in NIDDM subjects is attributable to between-subject variation. It was gratifying that the CVs of blood glucose responses within- and between-subjects that they observed in NIDDM patients treated with diet and oral agents—19 and 33%—respectively, were similar to those we found in 1985, for NIDDM patients on diet and oral agents—16 and 34%, respectively (2). These values also are similar to those for within- and between-subject variation of glycemic responses we found in 1985 for NIDDM patients on insulin—15 and 23% (2), and in 1989 for a mixed group of NIDDM patients (7 on insulin and 4 on diet or oral agents)—16 and 25% (3). Rasmussen et al. then expressed surprise about our report in 1990 that the within-subject variation of the glycemic index was much greater than that between subjects, with SDs of 16 and 6, respectively (4). However, far from being surprising,

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these results are to be expected. It is important not to confuse the terms glycemic index and glycemic response; they mean different things and have different mathematical and statistical properties. The glycemic index standardizes glycemic response areas to each individual's response to a standard food, thus correcting for between-subject variation. The figure, drawn from our 1990 data, shows the percentage of the total variance of the glycemic responses and glycemic index values attributable to the order of testing, the different foods (bread, rice, and spaghetti), order-food interaction,[2] subject (between-subject variation), and error (within-subject variation) (4). Most of the variation of the blood glucose response areas was attributable to between-subject variation. However, expressing the results as glycemic indexes, the proportion of the total variance attributable to between-subject variability is reduced markedly. This is the purpose of the glycemic index.

It also should be pointed out that expressing results as the glycemic index reduces total variability by reducing only between-subject variation without affecting within-subject or between-food variation (Table 1). The between-subject variation of glycemic responses in this study is large because both IDDM and NIDDM subjects were studied and the subjects were selected to be dissimilar so as to increase between-subject variation (4).

Table 1—Sources of variation of glycemic response areas and glycemic index values expressed as SDs and CVs

Source of variance	Glycemic response area		Glycemic index	
	SD	CV (%)	SD	CV (%)
Food	262	31	22.9	30
Within-subject	210	25	15.9	21
Between-subject	408	49	5.8	8

Data from reference 4.

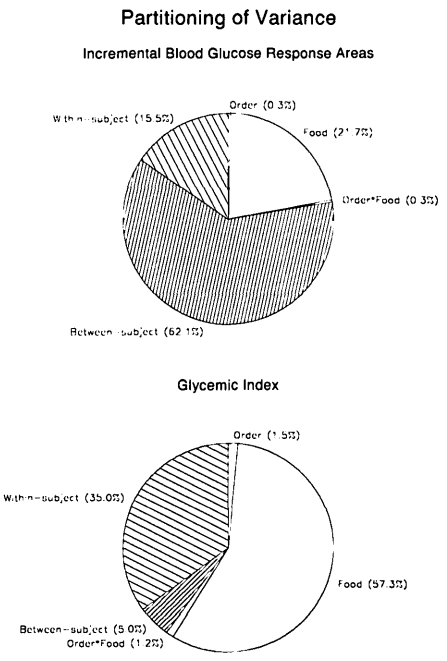

FIG. 1—*Sources of variance of glycemic responses and glycemic index values expressed as percentage of total variance. Data from (4).*

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NIDDM, NON-INSULIN-DEPENDENT DIABETES MELLITUS; CV, COEFFICIENT OF VARIATION; IDDM, INSULIN-DEPENDENT DIABETES MELLITUS.

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Hazard of Glucagon Test in Diabetic Patients

Hypertensive crisis in asymptomatic pheochromocytoma

Plasma C-peptide response to glucagon has been widely applied for a measure of insulin secretory capacity or classification of insulin-requiring or non-insulin-requiring diabetic patients (1,2). On the other hand, glucagon has been used for the diagnosis of pheochromocytoma as provocative test, which can precipitate a severe pressor response and even a hypertensive crisis. Patients with pheochromocytoma are often associated with diabetes mellitus, indicating the danger of glucagon test in such cases. To the best of our knowledge, no such case has been reported. We present a case of asymptomatic pheochromocytoma, which was incidentally diagnosed because of the occurrence of hypertensive crisis during glucagon test for evaluation of β-cell function.

A 50-yr-old Japanese woman (1.64 m, 57 kg, body mass index 21.2 kg/m^2) was referred to our hospital in December 1990 for uncontrolled diabetes mellitus. She had been taking hypoglycemic agents for 6 yr and had no history of hypertension. On admission,

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