The primary problem with NIDDM patients seems to be that they do not take their disease seriously and are less aware of possible long-term harmful complications. Educational programs for teaching self-care seldom succeed in NIDDM patients, which is why it is difficult for the diabetologist to convince them to maintain a record book.

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Exaggerated Plasma Catecholamines and Cortisol Responses to Hypoglycemic Stress in Essential Hypertension

An estimated 30–60% of diabetic patients may have associated hypertension, and the simultaneous presence of these two conditions increases the morbidity and mortality in a subgroup already at high risk for atherosclerosis. Many studies have pointed out the importance and the benefits of tight antihypertensive therapy in these patients. Furthermore, early diagnosis of both impaired glucose tolerance and hypertension appears to be crucial in the prevention of cardiovascular attacks, especially brain and myocardial infarction. The association between hypertension and diabetes mellitus has generally been attributed to an abnormal vascular volume (1) and/or to hyperinsulinemia that may per se increase the blood pressure (2). However, an interference of plasma catecholamines and cortisol cannot be completely excluded.

We studied 20 subjects with mild impaired glucose tolerance (detected by 75-g oral glucose tolerance test and diagnosed with National Diabetes Data Group criteria; 3) without (n = 10) and with (n = 10) mild essential hypertension (diagnosed with World Health Organization criteria). In the latter group, no subject had renal impairment or papilledema, and no cause of high blood pressure or any associated disease was detected after complete examination; consequently, all subjects were considered to have benign essential hypertension. There were no significant differences in age (47 ± 8 vs. 53 ± 7 yr), body mass index (23 ± 2 vs. 24 ± 4), basal plasma glucose (86 ± 8 vs. 84 ± 9 mg/dl), or diastolic blood pressure (90 ± 5 vs. 91 ± 4 mmHg) between the two groups. In contrast, basal plasma insulin (13 ± 6 vs. 9 ± 4 mU/L, $P < .01$) and systolic blood pressure (163 ± 22 vs. 138 ± 10 mmHg, $P < .01$) were significantly enhanced in subjects with hypertension. No subjects had taken any drugs for at least 6 wk before the study. All subjects gave informed consent, and the study was approved by the ethics committee of our institution. All subjects were consuming a weight-maintaining diet. After an overnight fast (12 h), both groups of subjects underwent a test for hypoglycemia according to the method of White et al. (4). Plasma glucose was determined by a glucose oxidase technique immediately after the experiment (Auto-Analyzer, Beckman, Fullerton, CA; intra-assay variability 2.9%). Plasma samples for hormone determinations were stored at −20°C until assay. Plasma insulin (Bio-Data kit, Italy; intra-assay variability 3.7%), cortisol (Bio-Data kit, intra-assay variability 4.1%), growth hormone (Bio-Data kit, intra-assay variability 3.9%), and glucagon (with a 30K Unger's antibody, Byk-Gulden mat kit, Cormano, Italy; intra-assay variability 4.9%) levels were all determined by radioimmunoassay methods. Plasma catecholamine levels were determined by a classic radioenzymatic method (5). After preliminary ANOVA, statistical comparison was performed via nonparametric (Wilcoxon's rank-sum test) and parametric (t test for unpaired data) tests and r of Pearson. A P value of .05 was chosen as level of significance. All results were expressed as means ± SE.

As depicted in Fig. 1, basal plasma counterregulatory hormone levels did not achieve statistically significant differences. During insulin infusion, plasma glucose declined significantly lower in subjects with essential hypertension. In the same subjects, exaggerated plasma catecholamine and cortisol responses were also observed, whereas plasma glucagon and growth hormone levels had a similar surge. Furthermore, in subjects with essential hypertension, basal systolic blood pressure was positively correlated with basal plasma insulin (r = .79, $P < .01$) and with plasma epinephrine, norepinephrine, and cortisol responses (evaluated as area under the curve) (r = .65, $P < .01$; r = .68, $P < .01$; r = .71, $P < .01$, respectively).

Our data, in agreement with those reported by Ferriss et al. (6), who studied diabetic subjects under different stressful conditions, are interesting in light of the deranging effects of catecholamines and cortisol on glucose tolerance. These hormones may increase hepatic

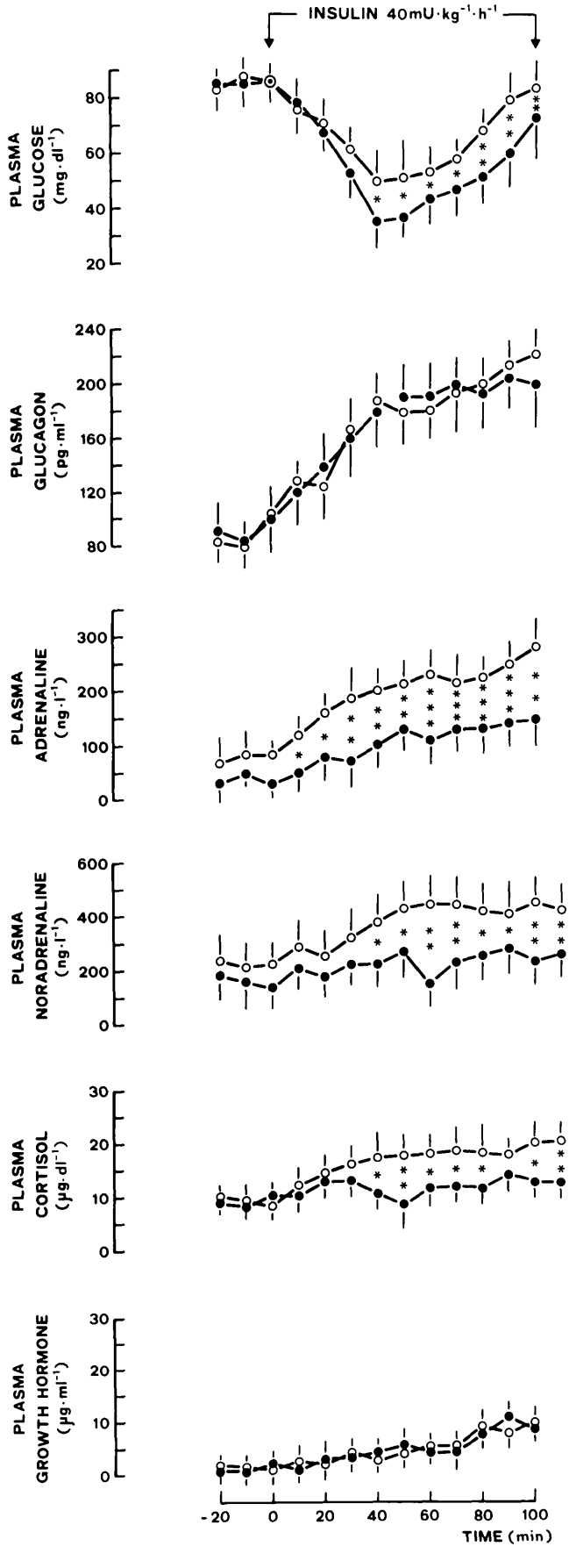

FIG. 1. Counterregulatory hormone responses to hypoglycemic stress in subjects with impaired glucose tolerance with (○; *n* = 10) and without (●; *n* = 10) essential hypertension. All results are means ± SE. Statistically significant differences: **P* < .05, ***P* < .01, ****P* < .001.

glucose production and decrease peripheral glucose uptake (7) by a reduction of insulin binding (8). The pathophysiological response to this changed glucose homeostasis could lead to hyperinsulinemia to counterbalance catecholamine-induced hepatic glucose overproduction. Nevertheless, hyperinsulinemia fails to increase catecholamine-reduced glucose uptake at peripheral-site levels (7). In these subjects a vicious cycle could take place: exaggerated plasma catecholamine and cortisol responses to stressful conditions might induce hyperinsulinemia, which in turn might lead to hypertension and insulin resistance, which is itself a cause of hyperinsulinemia. Moreover, hyperinsulinemia is per se an additional risk for atherosclerosis and cardiovascular diseases. In the pathogenesis of impaired glucose tolerance, a genetic trait may also play a fundamental role; thus, hypertension could facilitate the expression of this genetic trait. Whether hypertension precedes or follows impaired glucose tolerance is still being debated. However, both pathologies when present simultaneously may facilitate a quicker rise of diabetic complications.

In conclusion, in the presence of impaired glucose tolerance and hypertension, a correct antihypertensive treatment should be administered quickly and should include selective β-blockers to simultaneously permit the best cardiovascular protection and the absence of side effects of carbohydrate metabolism.

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Impact of Budget Cuts on Diabetic Control in Urban Adult Diabetes Clinic

Funding cuts are a fact of life of health-care delivery in the United States. Whereas much debate has centered on the impact of such cuts on patients, few, if any, studies have been published that demonstrate an impact or lack of one on health care after services have been eliminated or curtailed. The opportunity to study the impact of cuts occurred in May 1986, when the frequency of visits to our adult diabetes clinic located in a neighborhood family-care center administered by the New York City Health and Hospital Corporation was halved. Appointments were made such that the total frequency of clinic visits remained unchanged. The reduction in diabetes clinic visits was "compensated" by an equal increase in general medical clinic visits. Nonsubspecialty visits took place in primary-care (general medical) teams; patients might be seen by an internist, a family-practice physician, or a nurse practitioner. Well before the frequency of diabetes clinic visits was halved, all primary-care providers were given materials explaining diet calculation and containing algorithms for adjustment of insulin dosage and lectures on diabetes management.

Glycohemoglobin before halving the frequency of subspecialty visits and at least 120 days after was then determined by high-performance liquid chromatography at a reference laboratory. Pre- and postreduction glycohemoglobin were compared by paired *t* test. Correlation of pre- and postreduction glycohemoglobin was by linear regression. The pre- and postreduction glycohemoglobin levels are shown in Fig. 1. Mean (±SE) glycohemoglobin rose from 8.8 ± 0.6 to 12.0 ± 0.8% (normal range 4.4–8.2%) after diabetes-clinic frequency was halved ($P < .001$). Baseline and postreduction glycohemoglobin did not correlate significantly ($r = .319$, $P > .05$).

In humans, the quality of diabetic control is negatively associated with the incidence and rapidity of progression of several complications, including adverse outcome of pregnancy (1), retinopathy (2), neuropathy (3), nephropathy (4), and left ventricular dysfunction (5). Thus, deteriorations in control cannot be taken lightly.

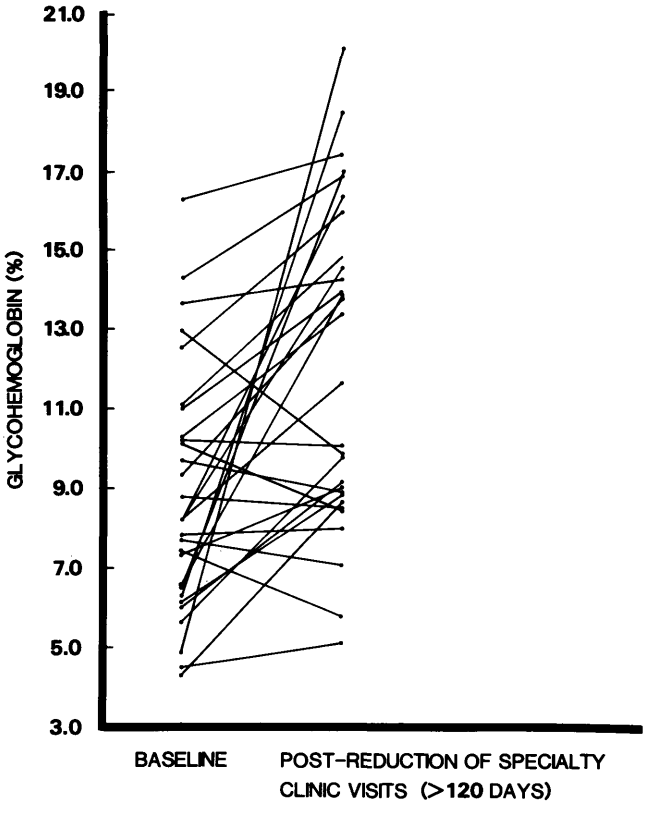

FIG. 1. Change in glycohemoglobin in 29 patients after halving frequency of diabetic clinic visits.

Inspection of the data does not permit prediction of which patients would maintain the same level of control, which patients would deteriorate, and which patients would improve (Fig. 1). Those who performed self-monitoring of blood glucose regularly and those who did not might either deteriorate or improve when subspecialty clinic exposure was reduced.

Review of primary-care provider encounters revealed several factors that might have contributed to a deterioration of control. *1*) Patients were less likely before primary-care provider clinic encounters to have performed our complete laboratory profile, consisting of fasting blood glucose, 1130 and 0400 h serum glucose, glycohemoglobin, serum creatinine, fasting serum total cholesterol, and high-density lipoprotein. In some cases, this was because the provider did not order the profile and in others because the patient failed to have it performed. *2*) There was a greater tendency to order only fasting or casual serum glucose determinations between clinic visits. *3*) There was an apparent reluctance by many, although not all, primary-care providers to adjust the therapeutic plan for mild to moderate hyperglycemia, i.e., serum glucose 140–300 mg/dl. *4*) There