from Matthews et al. (3), the formula for HOMA (*R*) is presented as insulin/($22.5e^{-\ln \text{glucose}}$). Therefore, we described this formula in the METHODS section of our article according to the original paper. However, in recent publications from other investigators, the formula (insulin × glucose)/22.5 is chosen for HOMA (*R*) (4,5). The latter formula makes it simpler to calculate insulin resistance index than does the original formula. Thus, we are in agreement with your valuable comment.

KAZUNARI MATSUMOTO, MD
MAYUMI YANO, MD
YUKITAKA UEKI, MD
SEIBEI MIYAKE, MD

From the Department of Internal Medicine, Sasebo Chuou Hospital, Sasebo City, Nagasaki, Japan.

Address correspondence to Kazunari Matsumoto, MD, Department of Internal Medicine, Sasebo Chuou Hospital, 15 Yamato-chou, Sasebo City, Nagasaki 857–11, Japan.

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Screening for Gestational Diabetes

What is the no-show rate?

The recent report from the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (1) has recommended retention of the two-step process for diagnosing gestational diabetes. The process involves using a 50-g glucose load as a screening test, which, if positive, is followed by a 100-g diagnostic test. One of the increasingly heard criticisms of this process is the so-called "no-show" rate (2). Since Magee et al. (3) drew attention to this phenomenon when 91 of 456 positive screened individuals refused the diagnostic test, others have used this to argue against the use of a screening test. They have suggested instead a one-step process involving only a diagnostic test for all pregnant women (2). We have been surprised by the high patient no-show rate quoted (2–4).

The Antenatal Clinic at Royal North Shore Hospital, Sydney, Australia, follows the two-step screening/diagnostic process. The Clinic serves a population of women that broadly reflects the multicultural background of the larger Australian cities. Of the women, 57.7% were not born in Australia, and 11.5% require interpreters. All women receive information about the screening process at their booking-in visit (16–20 weeks). At 26–28 weeks, they are offered the 50-g glucose challenge test at their routine clinic visit. The testing is done by midwives who attend and supervise the running of the clinic. Positive tests are followed up by the midwives. Women with a positive screening test (a 1-h value ≥7.8 mmol/l) are offered the diagnostic test.

We have examined our records from 1 January 1995 to 30 June 1997. Of 2,133 patients who underwent screening, 477 were positive. All these patients attended for the diagnostic test. This gives us a simply calculated no-show rate of 0%. We experienced our first no-show in July 1997.

These data, which involve a number of patients similar to those included in the original Magee et al. publication, show that the two-step screening/diagnostic process can be made to work effectively. A no-show rate in one setting cannot be assumed to apply elsewhere. Strategies to limit the no-show rate are an alternative to proceeding to a diagnostic test in all patients.

ROSEMARY HITCHMAN, RN
GITA MATHUR, BSC, MB, BS
AIDAN MCELDUFF, MB, BS, PHD, FRACP

From the Department of Endocrinology, Royal North Shore Hospital, St. Leonards, Australia.

Address correspondence to Aidan McElduff, Department of Endocrinology, Royal North Shore Hospital, St. Leonards, NSW 2065, Australia.

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Response to Hitchman et al.

The purpose of glucose tolerance testing in pregnancy is to detect women with gestational diabetes mellitus (GDM). The purpose of a preliminary glucose challenge test is to reduce the number of women who must have a full glucose tolerance test (GTT). When the full GTT, as widely practiced in the U.S., involves a minimum commitment of 3 h, 100 g of a glucose solution, and four blood tests, this is both understandable and commendable.

The number of women who test positive on a challenge test and who do not proceed to a full GTT has not been specifically examined in the literature but can be gleaned from incidental data. In Western societies, it appears to be ~15–25%. That around one-fifth of all women who have a positive challenge test, of whom about a quarter could be expected to have GDM, do not proceed to the diagnostic GTT must be a cause for concern. In average hands, a two-stage testing procedure will not only underdiagnose GDM but will delay treatment of diagnosed women by the time taken for the second test to be done.

With the above reservations in mind, the results reported by Hitchman et al. (1) are outstanding and demonstrate what can be achieved by a dedicated and enthusiastic staff. To achieve total compliance over a 30-month period with one test is remarkable; to maintain this for a two-stage pro-

cedure is quite extraordinary. A challenge test followed by a definitive GTT can remain a viable option if compliance approaching this figure could be achieved in all centers.

However if this is not possible, then any modification of the existing glucose tolerance testing procedure for pregnancy to one that increases the compliance and reduces the inconvenience should not be dismissed.

ROBERT G. MOSES, FRACP

From the Illawarra Area Health Service, Wollongong, New South Wales, Australia.

Address correspondence to Robert G. Moses, 4/393 Crown St., Wollongong, NSW, 2500, Australia. E-mail: robert_moses@uow.edu.au.

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Antibodies to GAD in Elderly Patients With Previously Diagnosed NIDDM

Clinical significance of seropositivity for antibodies to GAD (anti-GAD) has been extensively investigated in middle-aged patients with NIDDM (1,2). To our knowledge, however, there have been no studies on the prevalence and clinical significance of anti-GAD in elderly patients with NIDDM, despite the fact that the number of elderly diabetic patients is increasing. Thus, we examined anti-GAD by a radioimmunoprecipitation assay with recombinant human GAD65 (3) in 183 consecutive elderly patients (78 men and 105 women) ≥65 years old (mean age, 72.3 ± 6.1 years) with previously diagnosed NIDDM.

The prevalence of anti-GAD in elderly patients with NIDDM was 5.5% (10 of 183). It was 9.2% (7 of 76) in insulin-requiring NIDDM and 2.8% (3 of 107) in NIDDM well controlled by diet and/or oral hypoglycemic agents. Impaired pancreatic β-cell function was a major clinical characteristic of insulin-requiring elderly patients with NIDDM who were positive for anti-GAD (anti-GAD$^+$) compared with those negative for anti-GAD (anti-GAD$^-$) (fasting plasma C-peptide, 0.12 ± 0.10 vs. 0.56 ± 0.39 nmol/l, respectively; $P < 0.01$ by unpaired Student's t test), as is observed in middle-aged anti-GAD$^+$ NIDDM patients (4). Interestingly, seropositivity for anti-GAD alone was not predictive of the later development of insulin deficiency in elderly patients with NIDDM, but a genetic factor (HLA-DRB1) might also be a prognostic indicator in elderly anti-GAD$^+$ NIDDM patients (Table 1). In brief, anti-GAD$^+$ elderly NIDDM patients with DRB1*0405 and/or DRB1*0901, alleles that are associated with susceptibility to IDDM (5), tended to develop insulin deficiency within a short period. Even insulin-requiring anti-GAD$^+$ elderly NIDDM patients without those alleles developed insulin deficiency after a long period. In contrast, β-cell function was preserved in two patients with the DRB1*1502 allele, which protects against IDDM.

In conclusion, anti-GAD appeared to be a useful marker for predicting later development of insulin deficiency in elderly NIDDM patients and for determining their appropriate treatment when insulin is required because of secondary failure of sulfonylurea agents, which occurs in elderly as well as younger patients.

MICHIAKI FUKUI, MD
NAOTO NAKAMURA, MD
KOJI NAKANO, MD
MANABU SAWADA, MD
OSAMU AOJI, MD
HIROSHI OBAYASHI, PHD
HIROFUMI SHIGETA, MD
MOTOHARU KONDO, MD

From the First Department of Internal Medicine (M.F., N.N., K.N., H.S., M.K.), Kyoto Prefectural University of Medicine; Kyoto Microbiological Institute (H.O.), Kyoto; and Daijyukai Hospital (M.S., O.A.), Osaka, Japan.

Address correspondence to Michiaki Fukui, MD, First Department of Internal Medicine, Kyoto Prefectural University of Medicine, 465 Kajii-cho, Kawaramachi-Hirokoji, Kamigyo-ku, Kyoto 602, Japan.

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Table 1—*Clinical profiles of elderly anti-GAD$^+$ NIDDM patients*

Patient number	Sex	Age (years)	Age at onset (years)	Disease duration (years)	BMI (kg/m^2)	sCPR (nmol/l)	uCPR (μg/day)	Current treatment	Daily insulin dose (U/kg)	Interval* (years)	HbA_{1c} (%)	HLA-DRB1 genotype
1	M	79	52	27	18	0.10	7	Insulin	0.27	26	6.6	0803/0901
2	F	65	44	21	21	0.03	ND	Insulin	0.52	16	7.2	0406/1403
3	F	71	52	19	22	0.17	15	Insulin	0.31	15	8.0	1001/1401
4	F	66	40	26	19	0.30	ND	Insulin	0.3	25	7.4	0803/1501
5	F	69	45	24	23	0.17	14	Insulin	0.46	20	8.6	0410/1302
6	F	75	66	9	23	0.03	ND	Insulin	0.19	3	7.8	0803/0901
7	F	68	64	4	25	0.03	ND	Insulin	0.94	1	11.5	0405/0901
8	M	72	57	15	19.5	0.63	41	Diet	—	—	5.5	0901/1101
9	M	71	55	16	22	0.96	ND	SU	—	—	8.3	0802/1502
10	M	67	51	16	19	0.76	ND	SU	—	—	7.0	1502/1502

*Interval between diagnosis of diabetes and initiation of insulin therapy. ND, not determined; sCPR, fasting plasma C-peptide; SU, sulfonylurea; uCPR, 24-h urinary C-peptide excretion.