an unpleasant, inconvenient, time-consuming, and expensive procedure.

A number of points in the current report merit specific comment. First and most important is the fact that while we developed the screening questionnaire using the diagnostic criteria for diabetes that were accepted at the time as the "gold standard," Knudson et al. defined a fasting glucose >109 mg/dl or a random glucose >159 mg/dl as the gold standard. They did not state what proportion of subjects actually had diabetes by old or new ADA criteria. In addition, Knudson et al. performed definitive follow-up for only 396 of >1,000 subjects and did not state whether they were representative of the population. In contrast, the NHANES-2 provided follow-up for all subjects and subjects were representative of the U.S. population. When patients with positive screening tests are preferentially referred to receive verification by the gold standard test, work-up or verification bias may occur and may substantially distort sensitivity and specificity (3). The lower specificity of the test in the Onondaga County population may relate either to the fact that the gold standard was defined differently or to work-up bias. Sensitivity and specificity are not constants of nature but depend on the population to which the test is applied (3).

Even despite these differences, the performance of the test was in fact quite similar in the two populations. We also found that the sensitivity of the questionnaire was somewhat higher and the specificity somewhat lower among blacks, Hispanics, and Native Americans compared with whites (2). Although minority populations are more likely to have undiagnosed diabetes than whites, race and ethnicity did not enter into the classification trees (2). This suggests that although individuals conducting screening might want to target high-risk minority populations, the instrument is generally valid because the selected risk factors have the same predictive value in different racial and ethnic populations.

We acknowledge that we did not include other important risk factors, such as history of impaired glucose tolerance, hypertension, dyslipidemia, and history of gestational diabetes, in the questionnaire. This was a conscious decision based on our desire to develop a screening instrument that could be used in all populations, including the medically underserved (as was the population of Onondaga County) (2). The advantage of this approach is that the accuracy of the questionnaire does not depend on the respondents having had prior medical evaluation or care.

We certainly concur with de-emphasizing the numeric scores in the questionnaire, since they have no intrinsic meaning but were merely devised to identify subjects in the terminal leaves of the classification tree.

Although we also concur with the careful assessment of symptoms as a part of any medical evaluation for diabetes, published studies suggest that screening based on symptoms is not of value, since up to one-third of all individuals screened report frequent urination, extreme fatigue, and blurred vision (4–6). Clearly, to the extent that the screening questionnaire serves as an educational tool, it should describe the symptoms of uncontrolled diabetes (as the ADA questionnaire does). More sophisticated probing may, however, be necessary to make sense of these symptoms.

We certainly recognize that with the use of any screening test, false negatives will occur. Generally, this is addressed by establishing a screening threshold with a high sensitivity (7). In addition, periodic rescreening of the population can identify false negative screenees over time (7).

Finally, although we concur with the authors and the ADA that periodic screening is desirable, we continue to believe that further applied research is needed to rigorously evaluate the "who, where, when, and how of screening" and to assess cost-effectiveness (7). Careful predissemination evaluation of screening tests is vital to eliminate useless tests before they receive widespread application (3).

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Ketoacidosis During Gestational Diabetes

Case report

Gestational diabetes mellitus (GDM) presenting with ketoacidosis is highly unusual. Clinical reports of diabetic ketoacidosis (DKA) during pregnancy relate either to undiagnosed type 1 diabetes (1), to GDM complicated by stress (prolonged labor or infection) (2), or to the use of high doses of glucocorticoids or β-adrenergic receptor agonists for premature labor (3,4). We describe a woman who had GDM complicated by ketoacidosis without any identifiable precipitating factors. She remains nondiabetic 9 months after delivery.

A previously healthy 25-year-old Mauritian woman of African ethnicity, gravida 1, para 0, presented at 32 weeks of gestation with a 2-day history of vomiting, vertigo, polydypsia, and polyuria. She had undergone an O'Sullivan test (50 g of glucose by mouth) with a glycemia of 9.0 mmol/l at 60 min 1 week earlier. There

was no family history of diabetes. She was on no medication and took no alcoholic beverages. On admission, she was hyperventilating, afebrile, and dehydrated and weighed 76 kg at a height of 160 cm (BMI before pregnancy was 26.9 kg/m^2); her pulse was 120/min, and her blood pressure was 100/50 mmHg. Physical examination was otherwise normal for the gestational age. The pelvic ultrasound was normal. Urinalysis revealed 4+ ketone bodies and glucose, as well as 4 leukocytes per field. Urine and cervical smear cultures were negative. Plasma glucose was 23.7 mmol/l, ketone bodies 2+, lactate 1.9 mmol/l, Na 130 mmol/l, K 4.8 mmol/l, urea 4.8 mmol/l, creatinine 88 μmol/l, HCO_3 10.7 mmol/l, hemoglobin 12.4 g/dl, and leukocytes 6,200/μl; arterial pH was 7.29, P_{CO_2} 3.03 kPa, and P_{O_2} 14.8 kPa. HbA_{1c} was 8.5%. Islet cell antibodies (ICA) were negative. The diagnosis of DKA was established. Routine therapy for DKA with intravenous normal saline, potassium, and insulin corrected the metabolic abnormalities. She was treated subsequently with daily subcutaneous insulin until delivery (37 2/7 weeks of gestation). Spontaneously, she gave birth to a macrosomic boy (4,060 g) without any complication. Immediately after delivery, fasting and postprandial glycemias returned to normal levels. At 9 months postpartum, ICA and GAD antibodies were negative. An oral glucose tolerance test (OGTT) (75 g) was compatible with glucose intolerance from fasting and 2-h plasma glucose levels of 4.6 and 8.1 mmol/l, respectively (5). Plasma C-peptide rose from 1.2 to 4.5 nmol/l during the OGTT.

To our knowledge, this is the first case of DKA during gestation without a clearly identified event predisposing to ketoacidosis. Negative urine culture excluded a suspected urinary tract infection. The O'Sullivan test performed 1 week before DKA could have precipitated the event, although this has not been previously reported in the literature. Banerji (6) described in 1994 a unique form of diabetes among black adults with severe DKA at onset of diabetes and a clinical course resembling that of NIDDM (residual C-peptide secretion capacity, GAD and ICA antibodies negative). Our patient might fit into this category because she is of African ethnicity, overweight, and insulin-resistant. We conclude that DKA, although rare, may complicate GDM without clearly identifiable precipitating factors.

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Erratum

Yosipovitch G, Hodak E, Vardi P, Shraga I, Karp M, David M, Sprecher E: The prevalence of cutaneous manifestations in IDDM patients and their association with diabetes risk factors and microvascular complications. *Diabetes Care* 21:506–509, 1998

An incorrect spelling of Elliot Sprecher's name was published in the above article. Also, Pnina Vardi and Moshe Karp each hold an MD degree.