Gestational Diabetes

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Pregnancy has been shown to have a diabetogenic effect on carbohydrate metabolism, increasing the glucose response to a mixed meal or a pure carbohydrate challenge (1). On the other hand, fasting glucose levels in pregnant women are lower than in nonpregnant individuals (2). It appears intuitive, then, that the metabolic changes brought about by pregnancy might unmask incipient diabetes. Because overt maternal diabetes has been associated with adverse perinatal outcomes, it would seem logical to search for such an association in pregnancies complicated by incipient diabetes. Indeed, early studies demonstrated an increased likelihood of perinatal loss in pregnancies among individuals who later developed diabetes (3–5). Such studies left unanswered the question of whether the involved mothers would have demonstrated transient abnormalities of glucose metabolism if they had been tested during their pregnancies.

The seminal work of O'Sullivan and Mahan (6) introduced the concept that GDM was a condition that could be sought among pregnant women and might prove useful in the care of their pregnancies and themselves in the long run. O'Sullivan and Mahan (6) developed criteria for diagnosing GDM based on the predictive value of the glucose response to a 100-g, 3-h OGTT administered during pregnancy for the later development of diabetes (6). These criteria form the basis for the most widely used diagnostic paradigm in the U.S. Because they were based on the epidemiological concept of GDM as a risk factor for subsequent diabetes, and not as a risk factor for perinatal mortality, they have come under criticism in recent years. In fact, some have suggested that testing for GDM be abandoned (7). Indeed, virtually all recently published studies have failed to detect an increase in the perinatal mortality rate among GDM pregnancies. Although it is possible that this finding reflects a lack of increased risk among such pregnancies, it seems more likely that the identification of GDM, with increased pregnancy surveillance, has been successful in lowering the risk of perinatal mortality to background rates.

With current widespread screening programs for GDM, it is difficult to study the risks of this condition in its pristine, untouched state. Numerous studies have demonstrated that women with GDM manifest an increased rate of stillbirths in previous pregnancies, although ascertainment bias (resulting from screening based on risk factors) may have influenced some reports (8,9). Studies of women with undiagnosed GDM, who thus received no special treatment or observation, would appear to offer the best opportunity. The logical consequence of concluding that GDM carries with it no special risk would be an end to population-screening programs, leaving all or most cases of GDM undiagnosed. Only a few such studies exist. O'Sullivan et al. (10) compared 187 gravidas with untreated GDM, who were part of a randomized trial of prophylactic insulin conducted at a time when GDM was not a widely accepted diagnosis, to 259 randomly selected pregnant women with normal glucose tolerance. The perinatal mortality rate was 6.4% in the former group and 1.5% among the control subjects ($P < 0.05$). This fourfold increase in the perinatal mortality rate presumably was attributable to the presence of GDM. The suggestion has been made that confounders such as maternal age and obesity might have explained the observed differences; however, no pathophysiological mechanism exists that links these problems to perinatal death, although they are generally regarded as risk factors. On the other hand, there is a possible pathophysiological mechanism linking hyperglycemia and perinatal death, as is true in women with pre-existing diabetes who become pregnant (11). Pettit et al. (12) administered a 75-g, 2-h glucose challenge to 811 Pima Indian women during the third trimester of pregnancy. The results were not available to the clinicians managing the pregnancies. However, a continuous and significant ($P = 0.001$) increase in perinatal mortality was observed, from 0.5% when the 2-h plasma glucose level was <120 mg/dl (6.7 mM) to 4.4% when the value was 160–199 mg/dl (8.9–11.1 mM). Although the usual 100-g, 3-h OGTT used to diagnose GDM was not given, these data add credence to the hypothesis that increasing

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GDM, gestational diabetes mellitus; OGTT, oral glucose tolerance test; GTT, glucose tolerance test; STZ, streptozocin; PG, phosphatidylglycerol; BMI, body mass index; CI, confidence interval; NDDG, National Diabetes Data Group; ACOG, American College of Obstetricians and Gynecologists; ADA, American Diabetes Association; type I diabetes, insulin-dependent diabetes mellitus; type II diabetes, non-insulin-dependent diabetes mellitus.

Table 1—*Standard diagnostic criteria for GDM*

	O'Sullivan et al. (1973)*	NDDG (1979)†	Corrected data (25)‡
Blood glucose			
Fasting	5.0 mM (90 mg/dl)	5.9 mM (105 mg/dl)	5.3 mM (95 mg/dl)
1 h	9.2 mM (165 mg/dl)	10.6 mM (190 mg/dl)	10.0 mM (180 mg/dl)
2 h	8.1 mM (145 mg/dl)§	9.2 mM (165 mg/dl)	8.7 mM (155 mg/dl)
3 h	7.0 mM (125 mg/dl)‖	8.1 mM (145 mg/dl)	7.8 mM (140 mg/dl)

If any two thresholds are met or exceeded, GDM is diagnosed.
*Whole blood samples.
†Plasma or serum samples.
‡Corrects for change from whole blood to plasma or serum glucose and for the use of glucose oxidase or hexokinase methodology.
§Rounded up from 143 mg/dl.
‖Rounded down from 127 mg/dl.

abnormality of glucose tolerance in pregnancy is associated with increasing perinatal mortality when the results of glucose testing are not used in management.

Other diagnostic criteria, based on perinatal mortality risk, have been developed in Australia (13). Based on data collected from the universal application of a 50-g, 3-h OGTT to 18,679 pregnancies, these criteria are a 1-h whole blood value ≥9 mM (162 mg/dl) and a 2-h value of ≥7 mM (126 mg/dl). These values identified 2.5% of the population who had approximately twice the perinatal mortality risk of the remaining population. Given the fact that a 50-g, rather then a 100-g, load was given, the criteria are remarkably similar to the values described by O'Sullivan et al. (6). Gillmer et al. (14) have derived criteria for GDM based on the likelihood of neonatal hypoglycemia.

It is perhaps a tribute to modern obstetric care that we and our patients no longer are content merely with the avoidance of perinatal mortality. Stillbirths and neonatal deaths are, thankfully, uncommon enough events that expectations have shifted from survival to the quality of the offspring. Perinatal morbidity has thus become the focus of efforts to describe the risks of GDM. Although many of the various forms of morbidity characteristic of infants of overt diabetic mothers, including neonatal hypoglycemia and hyperbilirubinemia, have been reported in infants of GDM mothers (11), macrosomia has been the most widely studied of these adverse outcomes. In the previously described work by Pettit et al. (12), macrosomia, as well as perinatal mortality, was directly associated with increasing maternal glycemic response to a 75-g glucose challenge. An excess of macrosomia has been reported even among the offspring of GDM women whose glucose levels were relatively well controlled (15). Animal models of neonatal macrosomia related to mild maternal hyperglycemia support this connection (16,17). More recently, yet another type of morbidity has been described among the offspring of GDM mothers. The group at Northwestern University (Freinkel, 1988; 18) reported childhood obesity at 7 yr of age in children who were the macrosomic infants of GDM mothers, and have found a correlation between increased maternal glycemia during the third trimester and this later obesity. Thus, there is evidence for increased perinatal morbidity as well as mortality in pregnancies complicated by GDM.

Human data suggest an increased likelihood of diabetes in the mothers of women with GDM (19). Data on the pregnancies of female offspring of GDM mothers are not yet available, presumably because the diagnosis of GDM is a relatively recent phenomenon. However, in a painstaking series of experiments, Van Assche et al. (20) have shown that, in rat pregnancies, mild maternal hyperglycemia induced by injection of STZ around the time of conception can cause GDM in pregnancies of the offspring for at least two subsequent generations. Using constant maternal glucose infusion instead of STZ to raise maternal glucose levels by ~2 mM, Gauguier et al. (21) demonstrated similar intergenerational effects in the rat model. There is at least the possibility, then, that GDM may be associated with its own recurrence in the female offspring.

DIAGNOSTIC CRITERIA— As mentioned above, the criteria that form the basis for the diagnosis of GDM in the U.S. were validated by epidemiological studies of the subsequent development of diabetes among affected individuals. The original study of O'Sullivan et al. (6) used venous whole blood glucose samples, whereas current laboratory technology relies almost exclusively on plasma, or in some cases serum, samples. Because glucose measurements in plasma are ~14% higher than simultaneous whole blood determinations, the NDDG (22) proposed increasing the thresholds when plasma or serum is used. The original O'Sullivan values and the derived NDDG values are given in Table 1. The NDDG criteria in Table 1 have been adopted by the ADA (23) and the ACOG (24) and currently constitute the standard diagnostic criteria for GDM in the U.S.

One further change in the measurement of glucose has occurred since the original work of O'Sullivan et al. (6). The Somogyi-Nelson technology, used in O'Sullivan's original work, has been replaced by more specific enzymatic methods. Our group (25) proposed a conversion from the criteria of O'Sullivan et al.

(6), correcting not only for the change from whole blood to plasma, but for the use of glucose oxidase or hexokinase methodology, which yields values ~0.27 mM (5 mg/dl) lower than Somogyi-Nelson. These criteria are listed in Table 1.

It would seem that the most appropriate way to address the question of how best to convert GTT criteria from the original values of O'Sullivan et al. (6) to current methodologies would be to recreate the original methods, and measure parallel samples using both approaches. This, in fact, has been done (26), and it was determined that the current NDDG criteria exceed 95% CIs above the appropriate conversions at the 1-, 2-, and 3-h sampling times. On the other hand, the Carpenter and Coustan criteria (25) were within the appropriate range at all four sampling times. Tallarigo et al. (27) provided further evidence to support the likelihood that the NDDG criteria are higher than necessary. In their study, among patients with normal glucose tolerance, the likelihood of delivering a macrosomic baby was directly proportional to the height of the 2-h glucose response to a 100-g oral glucose challenge (27). Likewise, Langer et al. (28) found a markedly increased risk of neonatal macrosomia among the offspring of gravidas who had one abnormal value on their OGTT (using NDDG criteria), and thus were not diagnosed as having GDM. Thus, it would appear reasonable to use thresholds lower than those currently in vogue, if an appropriate conversion of the criteria of O'Sullivan et al. (6) is desired. Such a change would not, however, address the question of whether the current criteria (6), based on subsequent diabetes in the mother, are the most appropriate for obstetric practice, as the greatest concern during pregnancy is for the prevention of fetal morbidity and mortality.

SCREENING PARADIGMS— The simple and traditional method of screening for GDM is to take a history from the patient, looking for risk factors such as a family history of diabetes in a first-degree relative, previous large baby, previous perinatal loss, or other adverse obstetric outcome. If any such historic risk factors are present, a full diagnostic test (3-h, 100-g OGTT) is performed. Unfortunately, such a screening paradigm is only ~50% sensitive (29–31). In 1973, O'Sullivan et al. (29) reported a study in which a 50-g, 1-h glucose challenge was administered randomly to 752 prenatal patients, predominantly but not exclusively in the second and third trimesters. Most importantly, all subjects underwent the diagnostic 3-h, 100-g OGTT as well. The Boston group found that a venous whole blood glucose level of 7.2 mM (130 mg/dl) or more (Somogyi-Nelson method) identified 79% of the 19 individuals with GDM in the population. This was much more sensitive than the use of historic risk factors, and O'Sullivan recommended that all gravidas ≥25 yr of age undergo this screening test. The first International Workshop Conference on GDM extended this recommendation to all pregnant women, suggesting that the test be done between the 24th and 28th wk of gestation (32). The ADA subsequently adopted this recommendation as its formal position (23). The ACOG recommends screening all pregnant women ≥30 yr of age, with screening of younger women reserved for those with risk factors (24). Although a population-based study has demonstrated >50% of women with GDM <30 yr, this position still stands (31). Because of the change from whole blood to plasma or serum glucose, both the ADA and the ACOG recommend a threshold for further testing of 7.8 mM (140 mg/dl) (Table 1). Two studies have demonstrated that ~10% of women with GDM manifest screening test values between 7.2 and 7.8 mM (130–139 mg/dl) (31,33). Thus, the choice of thresholds depends on whether the increased sensitivity of a 7.2 mM cutoff warrants performance of full OGTTs on a larger proportion of patients. From the viewpoint of cost, lowering the age cutoff for universal screening to 25 years would result in virtually no increase in the cost per case of GDM diagnosed (31). Universal screening with a threshold of 7.2 mM (130 mg/dl) would result in ~30% increase in cost per case diagnosed, compared with following the ACOG recommendations.

When the 100-g, 3-h OGGT manifests only one abnormality among the four plasma glucose measurements, rather than the two abnormal values necessary to diagnose GDM, we repeat the test one month later. We have found that 34% of 106 patients tested in this manner had GDM at the second GTT (34).

MEDICAL MANAGEMENT— The goal of medical management of GDM is the prevention of perinatal mortality and morbidity. If we assume that GDM represents a generally milder end of the spectrum that extends to type I and type II diabetes, and that maternal-fetal hyperglycemia represents a threat to fetal well-being no matter what the cause of the elevated glucose level, then medical management can be viewed as an attempt to assure relatively normal circulating glucose levels in the mother (11). Although recommendations vary, as discussed below, there is no apparent reason to allow more severe hyperglycemia in patients with GDM than in those with pre-existing diabetes who are pregnant. The fetal insulin response to a hyperglycemic environment is related to the severity of hyperglycemia, and not to the nature of the maternal condition, whether attributable to insulinopenia, insulin resistance, or a combination of the two. Thus, it is generally true that the degree of hyperglycemia that would prompt a change in therapy for a mother with pre-existing diabetes should do the same for a mother with GDM.

The cornerstone of diabetes management is dietary intervention. Recommendations for dietary prescriptions in

women with GDM usually are parallel to those for pregnant women in general. In addition to maintenance of euglycemia, goals include the achievement of appropriate maternal weight gain and fetal growth. In our center, we prescribe 30–35 kcal · kg ideal body weight^{-1} · day^{-1}, divided into 3 meals, with 1–3 snacks, depending on whether the patient requires insulin (35). Other approaches have been effective as well (36). A thorough review of dietary approaches to diabetic pregnancy, written from a historical perspective, warrants reading (37).

Although weight reduction is effective in reversing the metabolic disturbance in many patients with type II diabetes, this approach is not practical in pregnant individuals because of the need for fetal nourishment to ensure proper growth and development. There has been some recent interest in moderate, and even severe, caloric restriction for obese patients with GDM; but, thus far, the results are equivocal. An overriding concern has been the production of starvation ketosis. Because epidemiological studies (38,39) suggested that maternal ketonuria may be associated with impairment of intellectual performance in the offspring, the avoidance of ketonuria became a goal for management of diabetic and nondiabetic gravidas. Because the process of starvation ketosis is enhanced during pregnancy (2,40), and because data from the Collaborative Perinatal Project also suggested an adverse effect of weight loss or minimal weight gain (particularly in underweight individuals) on pregnancy outcome (41), attention directed toward assurance of adequate weight gain and caloric restriction generally was considered inadvisable.

In 1985, Algert et al. (42) reported that obese (BMI >27 kg/m^2) GDM subjects, when placed on a moderately restricted diet (1700–1800 kcal/day; but not <25 kcal/kg), gained significantly less weight than lean GDM subjects or control subjects who were prescribed more liberal diets (33 kcal/kg). The authors noted that none of the patients demonstrated ketonuria on weekly clinic visits; however, no information was provided as to timing of urine samples, and circulating ketone body levels were not measured. Interestingly, the obese GDM subjects on hypocaloric diets gave birth to significantly larger infants than did the other two groups. The authors concluded that modest caloric restriction may be advisable for obese GDM women who are not prone to ketosis. Magee et al. (43) randomized obese pregnant women with GDM to control (2400 kcal/day) and calorie-restricted (1200 kcal/day) regimens under metabolic ward conditions. After 1 wk of fasting, plasma glucose was unchanged, but 24-h mean glucose profiles were significantly improved in the calorie-restricted group. Fasting β-hydroxybutyrate levels rose significantly in the calorie-restricted group, raising questions about potential fetal effects of this regimen. A recent study found that mothers with GDM who were prescribed calorie-restricted diets (1200–1800 calories per day) gained less weight and gave birth to similar-sized infants compared with a matched group of nondiabetic gravidas with negative diabetes screening tests (44). Women with normal GTTs but positive 50-g, 1-h screening tests had a significantly higher likelihood of delivering macrosomic (> 4,000 g) babies. Serum or urinary ketone bodies were not measured in this study. The use of caloric restriction for management of GDM should be considered promising but remains questionable.

Upwards of 25% of women with GDM will require insulin to maintain relative euglycemia (45). To detect patients who will require insulin, in addition to dietary therapy, it is necessary to measure circulating glucose levels at frequent intervals. Both the ADA (23) and the ACOG (24) suggest that glucose measurements be performed fasting and postprandially at 1- to 2-week intervals. Daily self-blood glucose monitoring has become routine for pregnant women with pre-existing diabetes mellitus; this same approach is used for GDM in many centers. Although this approach has not been demonstrated to more effectively prevent perinatal mortality than less frequent testing, it may enable earlier detection of hyperglycemia, possibly decreasing the likelihood of the fetal-neonatal complications of hyperinsulinemia. Nevertheless, because 2–3% of gravidas in the U.S. have GDM, it is critical to determine whether the benefits of this technology outweigh the costs (in both money and the human resources required to teach patients to make these measurements with relative reliability). Such data are not available at the present time; so, daily self-glucose monitoring should remain an option, albeit one that seems quite reasonable.

No uniform recommendations exist for the most appropriate time for postprandial glucose measurements. Both ADA (23) and ACOG (24) suggest that fasting and 2-h postprandial values be measured. Many centers recommend 1-h postprandial measurements because the peak glycemic response to a meal occurs by that time (46). Jovanovic-Peterson et al. (47) demonstrated that 1-h postprandial glucose measurements correlated more strongly with birth weight in diabetic pregnancies than did fasting values, but 2-h tests were not evaluated. Combs et al. have found similar results (47a). Although these findings were in pregnancies of women with pre-existing diabetes, it appears reasonable to assume that postprandial hyperglycemia attributable to GDM would have the same effect.

At present, it would seem that either 1- or 2-h postprandial values are appropriate, but different thresholds for intervention would apply to each approach. In centers such as ours, where the routine is to measure a set of blood glucose values weekly (fasting, 2 h after breakfast, 2 h after lunch), we make the assumption that glycemia on the day of testing is probably the most optimal of

the week. It is thus necessary to respond to even a single abnormal value. Thresholds of response for women with GDM, as suggested by both the ADA (23) and the ACOG (24), are the same as for women with pre-existing diabetes in pregnancy. There is no reason for the fetus of a mother with GDM to be subjected to glycemia higher than that of a mother with pre-existing diabetes. According to these guidelines, insulin is recommended when fasting plasma glucose exceeds 105 mg/dl (5.9 mM) and/or 2-h postprandial plasma glucose exceeds 120 mg/dl (6.7 mM) on two or more occasions within a 2-week interval. In our center, the response to a single elevation of glucose may prompt initiation of insulin therapy, the institution of daily self-glucose testing, or the return of the patient the next day for more measurements. Because the time available for therapeutic intervention is limited (usually to 8–10 weeks between diagnosis of GDM and delivery), the response should not be to return a week later for repeat testing.

If insulin therapy is to be instituted, several successful schemes can be used. Currently, there is widespread agreement that the insulin administered to women with GDM should be of human origin. In our center, women with GDM who require insulin because of hyperglycemia are started on a mixture of 20 U of intermediate-acting (NPH) and 10 U of short-acting (regular) human insulin each morning before breakfast. Although this starting dose may appear large, the marked insulin resistance of pregnant women make it unlikely to precipitate hypoglycemia, particularly if initiated in the third trimester. When GDM is diagnosed before the third trimester, and insulin is required, we generally reduce the starting dose described above by 50%. Most women with GDM will not require further insulin doses during the day, as their pancreatic reserve is capable of taking over once the morning insulin resistance has been overcome. However, some women require predinner or prelunch regular insulin and, sometimes, bedtime intermediate-acting insulin. Another successful approach is the use of a single dose of intermediate-acting insulin at bedtime for GDM subjects with elevated fasting plasma glucose. If the fasting value is controlled, many of these women will then be able to regulate glycemia throughout the rest of the day without further exogenous insulin. The most important aspect of insulin management in GDM is the frequent measurement of circulating glucose levels to ascertain the success of the regimen.

The above discussion of diet and insulin management of GDM is based on therapeutic goals similar to those prescribed for pregnant women with preexisting diabetes. These goals were derived from studies and series' concerned with reducing perinatal mortality rates. Various forms of morbidity are also more common in pregnancies complicated by GDM. Macrosomia, in particular, has received much attention, probably because it is relatively easy to measure and has certain fairly predictable consequences. In 1966, O'Sullivan et al. (48) reported the results of a randomized prospective trial in which macrosomia (defined as birthweight of ≥9 lbs) was reduced from 13% of the infants of untreated GDM women to 4% of those treated with an arbitrary 10 U of NPH insulin before breakfast each day. Although maternal obesity is a strong risk factor for fetal macrosomia, the above difference was present for the offspring of both overweight and normal weight mothers. Because of the concern that the temporary use of prophylactic insulin might predispose the women to antibody formation and, later, development of diabetes, O'Sullivan et al. (49) subsequently followed the women who had participated in the randomized trial. At 16 yr after their index pregnancies, similar 40% rates of diabetes had appeared in the insulin-treated and untreated groups and, in fact, decompensated diabetes was more common in untreated women who had borne large babies. Although this might suggest some sort of protective effect of prophylactic insulin, such a phenomenon has not been evaluated in other published studies to date. At least three other randomized trials of prophylactic insulin to prevent macrosomia have been conducted. Although one (50) found no advantage, two others (51,52) demonstrated a reduction in the rate of macrosomia. In a retrospective review of 445 GDM pregnancies, prophylactic insulin was associated with a 7% incidence of babies weighing >4000 g, whereas dietary management and no treatment were both associated with ~18% likelihood of such large babies (53). In addition, operative deliveries, defined as primary cesarean sections, mid-forceps, and mid-vacuum extractions, were 50% less common in the GDM pregnancies treated with prophylactic insulin.

Prophylactic insulin is probably effective in reducing the likelihood of macrosomia. Unfortunately, a large number of patients must be treated to benefit only a few. Ideally, a method to detect those individuals with GDM who are destined to deliver macrosomic children would allow the selection of a subset who would benefit from insulin. The use of frequent self-glucose monitoring with institution of insulin therapy for even mild degrees of maternal hyperglycemia has had effects similar to prophylactic insulin in preventing macrosomia (54).

Recently a national health consciousness has emerged, leading to an interest in vigorous, regular exercise to improve fitness. Because exercise exerts an insulinlike effect on glucose metabolism it is reasonable to consider physical activity as a potential therapeutic intervention in GDM pregnancy. Jovanovic-Peterson et al. (55) have demonstrated a significant improvement in fasting plasma glucose, the response to a 50-g glucose challenge, and HbA_{1c} measurements in a randomized trial of regular arm ergometry exercise versus diet only in 19 women with GDM. Widespread implementation of exercise as therapy for GDM awaits further study.

OBSTETRIC MANAGEMENT— The obstetric management of GDM centers around surveillance to detect potential fetal compromise and the establishment of the proper time for delivery. Other goals are the diagnosis of fetal macrosomia and the avoidance of shoulder dystocia.

Although the risk of sudden, unexplained fetal death is well documented in pregnancies complicated by preexisting maternal diabetes, this complication is considerably less likely in GDM pregnancy. The problem most likely is related to suboptimal metabolic control. Landon and Gabbe (56) have suggested counting daily fetal movement starting at 28 wk gestation, with biophysical fetal surveillance (nonstress testing, biophysical profile, or contraction stress testing) instituted at 40 wk gestation, provided no complications exist and metabolic control is optimal. Biophysical testing is started at earlier stages of pregnancy in patients with hypertensive disorders and adverse obstetric histories, and in those requiring insulin for metabolic management. Others have delayed the onset of surveillance to 32 wk gestation, and then instituted biophysical testing on a weekly basis (57). Outcomes were excellent in both studies; and, currently, there is no consensus as to the ideal time to commence surveillance in patients with GDM. Each of the three methods of biophysical surveillance noted above has its adherents; none is convincingly superior to the others. In our center, we begin performing weekly nonstress tests in otherwise uncomplicated GDM pregnancies at ~36 wk. The most important factors, which would precipitate earlier and/or more frequent testing, would be hypertensive disorders or suboptimal metabolic control.

The pregnancies of most women with GDM can be allowed to proceed to term and spontaneous labor without intervention. However, there are certain circumstances when obstetricians consider elective or indicated early delivery, either by induction of labor or by cesarean section, and in which special care should be taken in decision making. Because carbohydrate disturbances may delay fetal pulmonic maturation (58) by a mechanism mediated by fetal hyperinsulinism (11), thus slowing the production of surfactant, it is important to ascertain the presence of surfactant before elective delivery of the preterm pregnancy of a GDM mother. Currently, amniocentesis to detect the presence of biochemical markers such as PG or saturated phosphatidylcholine is the most common way to ensure fetal lung maturity (59). Whether to perform amniocentesis before elective delivery at term is somewhat controversial. We reported that 20% of 29 GDM women undergoing amniocentesis at 38 wk of gestation had absent PG, whereas all 33 such patients undergoing amniocentesis at 39 wk had PG present (60). Because no patient was delivered immediately after noting the absence of PG, it is impossible to be certain how many would have developed respiratory distress syndrome if delivered at 38 wk gestation without PG. In our center, we continue to document pulmonic maturity before elective delivery at any gestational age. However, we do not attempt apparently difficult amniocenteses at 39 wk or beyond, and consider diminished amniotic fluid at this point an indication for delivery.

GDM is not an indication for cesarean section. However, complications associated with GDM such as fetal macrosomia or maternal hypertensive disorders may increase the likelihood of abdominal delivery. Because macrosomia is associated with shoulder dystocia in diabetic pregnancy, it is particularly important to diagnose it before labor (61). Unfortunately, current ultrasound technology is not particularly efficient at diagnosing macrosomia (62) or predicting shoulder dystocia. Because ultrasound estimates of fetal weight are often in error by ≥10%, it is difficult to determine a particular estimated weight at which to recommend performing a cesarean section without labor based on data derived from actual birth weights. Nevertheless, at our center we would give strong consideration to primary cesarean section without labor when we believe the fetal weight to be ≥4500 g. With fetal weight between 4000 and 4499 g, we would also consider factors such as the patient's past obstetric history, the length of her second stage, and our clinical impression of her pelvic architecture.

LONG-TERM FOLLOW-UP— As mentioned earlier, the diagnostic criteria currently in use during pregnancy were validated by the subsequent development of diabetes. O'Sullivan (63) has found that ~40% of women diagnosed as having GDM during an index pregnancy develop overt diabetes within 20 yr. For this reason, ADA (23) recommends that women with GDM be followed postpartum to detect diabetes early in its course. This organization suggests an initial evaluation at the first postpartum visit, using a standard 75-g, 2-h OGTT. The high prevalence of subsequent diabetes may serve to make women with previous GDM an ideal group on which to attempt interventions designed to prevent the development of diabetes in the future.

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