Update on Gestational Diabetes

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Gestational diabetes mellitus (GDM), diabetes first diagnosed in pregnancy, complicates about 5% to 10% of pregnancies, which is an expected wide range of prevalence, given the variation in populations studied, the current variability in screening and diagnosis,¹ and a recent disproportionate increase in younger, obese women.²

GLUCOSE METABOLISM AND GESTATIONAL DIABETES

In normal pregnancy, directly or indirectly, the growth of the fetal-placental unit increases cortisol, growth hormone, human placental lactogen, estrogen, progesterone, and prolactin, which in concert lead to hyperinsulinemia, insulin resistance, fasting hypoglycemia, and postprandial hyperglycemia.³–⁵ A progressive transition of fuel sources occurs so that by the third trimester, the metabolic fuel to meet the demands of the fetus changes from predominately maternal carbohydrate to fat.

Pregnancy is characterized by increased and adaptive pancreatic beta-cell function to compensate for decreased insulin sensitivity and increased requirements.⁶ Morphologically, maternal pancreatic hypertrophy and hyperplasia occur.⁷ In response to elevated insulin levels, peripheral muscle glucose use and tissue glycogen storage increase in an effort to maintain normal insulin sensitivity in the first trimester of pregnancy.⁸–¹⁰ As gestation advances, these responses become inadequate to meet the energy requirements of the fetus, and insulin resistance develops.¹¹,¹²

Insulin resistance in normal pregnancy is estimated to increase by 40% to 70%, predominately in the third trimester. In a longitudinal study of healthy pregnant women using the hyperinsulinemic-euglycemic clamp, Catalano and colleagues¹¹ found a 56% decrease in insulin sensitivity in nonobese women by late pregnancy. Using the euglycemic-hyperinsulinemic clamp, Sivan and colleagues¹³ demonstrated that healthy women developed insulin resistance mostly in the third trimester and showed a 40% reduction in peripheral glucose uptake by muscle in the third trimester compared with the nonpregnant state. Using a minimal model technique, Buchanan

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and colleagues found that insulin sensitivity in normal pregnant women was reduced to about one-third of that of nonpregnant women of similar weight and age. Furthermore, the reduction in insulin sensitivity was compensated by reciprocal increase of the first and second phase insulin response.

There seems to be no significant change in insulin receptor binding in pregnancy; thus insulin resistance in normal pregnancy is likely related to postreceptor handling of glucose. Postreceptor mechanisms contributing to insulin resistance include (1) impaired tyrosine kinase activity, which is normally responsible for the phosphorylation of cellular substrates; (2) decreased expression of insulin receptor substrate-1, a cytosolic protein that binds phosphorylated intracellular substrates and transmits signals downstream; and (3) decreased expression of the GLUT4 glucose transport protein in adipose tissue, which promotes glucose uptake. The cytokine tumor necrosis factor-α and leptin may also be involved in insulin resistance seen in normal pregnancy.

Compared with normal pregnant women, women with GDM have impaired beta-cell function and reduced beta-cell adaptation resulting in insufficient insulin secretion to maintain normal glycemia. Women with GDM, and more so obese women with GDM, have greater insulin resistance and less endogenous hepatic glucose production than non-GDM women. Catalano and colleagues used the hyperinsulinemic-euglycemic clamp in a longitudinal study to assess insulin sensitivity and endogenous glucose metabolism in obese and nonobese pregnant women with and without GDM. These investigators found that obese women who develop GDM have a decreased insulin sensitivity along with suppression of hepatic glucose production during insulin infusion. Shao and colleagues noted a more profound drop in tyrosine kinase activity in women with gestational diabetes when compared with healthy normal women, suggesting a postreceptor mechanism abnormality as at least one cause of the increased insulin resistance in GDM.

Pregnancy-induced insulin resistance unmasks the beta-cell defects, which underlie GDM. These defects range from beta-cell dysfunction secondary to autoimmune factors or chronic insulin resistance or highly penetrant genetic abnormalities of insulin secretion.

SCREENING AND DIAGNOSIS

There has been a question regarding the need to diagnose and treat mild hyperglycemia in pregnancy; however, recent evidence has quieted the debate. Crowther and colleagues showed that treatment of hyperglycemia in pregnancy improved neonatal outcomes. The long-awaited Hyperglycemia and Adverse Pregnancy Outcomes (HAPO) study made unambiguous the linear positive association between maternal glycemia and adverse pregnancy outcomes. The study was a prospective, blinded, international, and multicentered 10-year study, with 25,505 pregnant women enrolled. The objective of the study was to clarify risks of adverse pregnancy outcomes associated with degrees of maternal glucose intolerance less severe than overt diabetes. Women were excluded from the study and their clinicians informed of test results if fasting plasma glucose was more than 105 mg/dL (5.8 mmol/L), or the 2-hour value greater than or equal to 200 mg/dL (11.2 mmol/L), or a random plasma glucose greater than or equal to 160 mg/dL (8.9 mmol/L). An additional random plasma glucose was collected between 34 and 37 weeks to identify possible late development of GDM; hypoglycemic women were also excluded. Routine prenatal and neonatal care was delivered in each of the 15 centers. The study investigators reported a significant association between maternal hyperglycemia and their
primary outcomes, birth weight greater than the 90th percentile, cesarean section, and cord plasma C-peptide level, reflective of fetal hyperinsulinemia. The degree of association for each outcome was graded across the spectrum of maternal hyperglycemia, with even mild maternal hyperglycemia of one standard deviation more than the mean associated with their primary outcomes (Table 1). Landon and colleagues recently reported for the Maternal-Fetal Medicine Network a multicenter study of 958 women, in which mild GDM was compared with normal glycemia. Women with mild GDM had more overgrown babies, shoulder dystocia, cesarean delivery, preeclampsia, and gestational hypertension. The network investigators concluded that treatment of mild GDM is beneficial.

There continues to be no resolution regarding the best method for screening or diagnosis of GDM. The current practice in the United States is 2-step testing, screening, and diagnosis. Universal screening (screening every pregnant woman) is practiced by most obstetricians in the United States because this method is associated with fewer errors of omission that might occur in a busy obstetric practice. However, now the American College of Obstetricians and Gynecologists and the American Diabetes Association recognize that there are low-risk women who do not need screening. The US Preventive Services Task Force suggests that physicians do not need to screen routinely for GDM but do need to discuss screening with each woman and make a case-by-case decision.

In the United States alone, an abnormal screening test has variability with the O’Sullivan 50-g glucose, 1-hour screening test cutoff ranging from 130 to 140 mg/dL (7.2–7.8 mmol/L). Even the next diagnostic step, the 3-hour, 100-g glucose tolerance test has at least 2 different glucose algorithms that are used for diagnosis of GDM: the National Diabetes Data Group and Carpenter-Coustan criteria (Table 2). Most clinicians outside the United States use a 1-step, 2-hour, 75-g glucose tolerance test for detection. A fasting plasma glucose level is obtained; then, after a 75-gm glucose load, 1-hour and 2-hour plasma glucose levels are measured. Variability in screening and detection has been difficult to resolve because of lack of consensus on the level of glycemia associated with adverse pregnancy outcomes until the recent HAPO study.

### Table 1

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Adjusted Odds Ratio(^a)</th>
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<tr>
<td></td>
<td>Birth Weight (&gt;90th Percentile)</td>
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<tr>
<td>Fasting Plasma Glucose&gt;1 SD</td>
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<tr>
<td>(6.9 mg/dL, 0.4 mmol/L)</td>
<td>1.38 (1.32–1.44)</td>
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<tr>
<td>1-h Plasma Glucose&gt;1 SD</td>
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<tr>
<td>(30.9 mg/dL, 1.7 mmol/L)</td>
<td>1.46 (1.39–1.53)</td>
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<tr>
<td>2-h Plasma Glucose&gt;1 SD</td>
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<tr>
<td>(23.5 mg/dL, 1.3 mmol/L)</td>
<td>1.28 (1.32–1.44)</td>
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</table>

Women with blood sugar levels in the gestational diabetes range were excluded from the study. All 3 primary outcomes were significantly increased.

*Abbreviation: SD, standard deviation.

\(^a\) Odds ratio (95% confidence interval).

In addition to providing definitive confirmation of the association of hyperglycemia with adverse pregnancy outcome, the HAPO study\(^{27}\) showed a positive, near-linear correlation between hyperglycemia and adverse pregnancy outcome. The HAPO study information should allow experts and stakeholders to reach a consensus on glycemic levels for diagnosis of GDM, which should be forthcoming.

Risk factors for GDM provide the basis for targeted screening. GDM is most frequent in women with prior GDM, severe obesity, or a sibling with diabetes. Many other risk factors are described (Table 3).\(^{37–46}\) More recently published associations with GDM include periodontal disease,\(^{47,48}\) low maternal birth weight,\(^{49}\) and high consumption of sugar-sweetened colas (see Table 3).\(^{50}\)

| Table 2 | Diagnostic parameters for the 3-hour, 100-g glucose tolerance test |
|-----------------|-----------------|-----------------|
| Time of Value   | National Diabetes Data Group Criteria | Carpenter-Coustan Criteria |
|                 | mg/dL           | mmol/L          | mg/dL           | mmol/L          |
| Fasting         | 105             | 5.8             | 95             | 5.3             |
| 1 h             | 190             | 10.6            | 180            | 10.0            |
| 2 h             | 165             | 9.2             | 155            | 8.6             |
| 3 h             | 145             | 8.0             | 140            | 7.8             |

Two or more values met or exceeded required to make diagnosis.


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| Table 3 | Risk factors for gestational diabetes |
|-----------------|-----------------|-----------------|
| Risk Factor      | Odds Ratio      | References      |
| Overweight       | 2               | Torloni et al; Chu et al |
| Obesity          | 3.7             | Torloni et al; Chu et al |
| Severe Obesity   | 7               | Torloni et al; Chu et al |
| Prior Gestational Diabetes | 23          | McGuire et al |
| Prior Macrosomic Infant | 3.3           | McGuire et al |
| Maternal Age Greater than 25 y | 1.4       | Cypryk et al |
| Maternal Age Greater than 35 y | 2.3       | Xiong et al |
| Multiple Gestation | 2.2\(^a\)     | Rauh-Hain et al |
| South East Asian  | 7.6\(^a\)       | Dornhorst et al |
| Hispanic         | 2.4\(^a\)       | Dooley et al |
| African American  | 1.8\(^a\)       | Dooley et al |
| Polycystic Ovarian Syndrome | 2.9     | Toulis et al |
| Parent with Diabetes | 3.2         | Kim et al |
| Sibling with Diabetes | 7.1        | Kim et al |
| Periodontal Disease | 2.6         | Xiong et al |
| Low Maternal Birth Weight | 1.9     | Seghieri et al |

\(^a\) Relative risk compared with white race.

Data from Refs.\(^{47,49}\)
Women at very high risk of development of GDM, such as those with obesity or prior GDM, may benefit from early screening in the first trimester. If early screening is normal, screening is repeated at 24 to 26 weeks of gestation.

**MATERNAL AND FETAL RISKS**

Glucose travels freely from the mother to the fetus, but maternal insulin does not. Thus, untreated hyperglycemia exposes the fetus to higher concentrations of glucose than normal, forcing the fetus to increase its own insulin production. Unfortunately, excess insulin produced by the fetus results in macrosomia, either from excessive fat deposition or as a direct growth effect of insulin. Mean maternal plasma glucose levels\(^{51}\) and fetal blood insulin levels\(^{52}\) are strongly associated with neonatal birth weight. Maternal glycemia during third trimester and prepregnancy body mass index are independent predictors of birth weight in pregnancies complicated by GDM.\(^{53}\)

The occurrence of GDM imparts significant and long-lasting health risks on mother and baby (Table 4). Fetal programming in utero increases the risk of obesity and obesity-related complications in children of mothers with diabetes.\(^{54}\)

**MONITORING**

Close monitoring and treatment of GDM are important to the long-term health of a pregnant woman and her baby. The fifth International Workshop-Conference on Gestational Diabetes recommended the following blood glucose concentrations: fasting plasma glucose of 90 to 99 mg/dL (5.0–5.5 mmol/L), 1-hour postprandial plasma glucose less than 140 mg/dL (<7.8 mmol/L), and 2-hour postprandial plasma glucose less than 120 to 127 mg/dL (<6.7–7.1 mmol/L).\(^{1}\) Baseline and interval hemoglobin A\(_{1c}\) levels during treatment are helpful, particularly in women who have fasting hyperglycemia. Most women with GDM on diet treatment alone monitor capillary blood glucose levels 4 times a day (fasting blood glucose once a day and postprandial blood glucose thrice a day); women on pharmaceutical therapy often monitor 4 to 6 times a day and include preprandial values. Weekly in-office monitoring and daily self-monitoring seem

<table>
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<th>Mother</th>
<th>Fetus</th>
<th>Newborn</th>
<th>Child/Adult</th>
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<tr>
<td>Birth trauma</td>
<td>Hyperinsulinemia</td>
<td>Respiratory distress syndrome</td>
<td>Obesity</td>
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<tr>
<td>Increased cesarean delivery</td>
<td>Cardiomyopathy</td>
<td>Hypoglycemia</td>
<td>Type 2 diabetes</td>
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<tr>
<td>Preeclampsia/ Gestational hypertension</td>
<td>Stillbirth</td>
<td>Hypocalcemia</td>
<td>Metabolic syndrome</td>
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<tr>
<td>Type 2 diabetes</td>
<td>Large for gestational age/ macrosomia</td>
<td>Hypomagnesemia</td>
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<tr>
<td>Metabolic syndrome</td>
<td>Birth trauma</td>
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<td>Hyperbilirubinemia</td>
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<td>Cardiomyopathy</td>
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to have comparable outcomes in perinatal mortality and morbidity. However, Hawkins and colleagues\textsuperscript{55} suggested that women with daily, more frequent monitoring may have less macrosomia.

Women on dietary and exercise therapy alone with normal self-monitored blood glucose levels can decrease the frequency of monitoring to twice a day. Our center prefers a fasting blood glucose and 1 other postprandial level per day, alternated throughout the week.

**DIET AND EXERCISE**

The initial treatment for GDM continues to be diet and exercise. Generally, a 1900- to 2400-kcal/d diet with carbohydrate restriction to 35% to 40% of calories is prescribed, calculated on ideal prepregnancy body weight and using complex and high-fiber carbohydrates.\textsuperscript{56} The assistance of a trained dietician is ideal for tailoring dietary needs for each woman.

Dietary therapy delays pharmacologic therapy. Moses and colleagues\textsuperscript{57} used a low-glycemic diet as treatment for GDM and in a prospective fashion showed that a low-glycemic diet decreased the need and timing for insulin. Most women lose weight during the initial weeks of dietary therapy but then resume modest weight gain. Insufficient dietary calories can be judged by excessive hunger, excessive weight loss, or persistent ketonuria.

If exercise is not contraindicated for other obstetric complications of pregnancy, it can improve glycemic control in any type of diabetes. Women with GDM should be asked to walk 1 to 2 miles at least 3 times a week, if possible.

**PHARMACOLOGIC THERAPY**

Pharmacologic therapy is most commonly instituted once diet and exercise have failed as evidenced by abnormality in more than half of self-monitored glucose values or an abnormal value in those women tested weekly. Traditionally, insulin has been the drug of choice because of its safety in pregnancy, lack of significant transplacental passage, and history of use. Most women can be treated as outpatients. The recommended initial insulin dose for pregnancy is based on maternal weight and can be calculated by the following guidelines to determine total daily insulin needs: 0.8 U/kg actual body weight in the first trimester, 1.0 U/kg actual body weight in the second trimester, and 1.2 U/kg actual body weight in the third trimester. However, because women with GDM have varying degrees of severity, in practice, insulin is started at 0.7 U/kg actual body weight to prevent hypoglycemia at home. Clinical judgment and experience assist in the selection of the starting dose of insulin. Once the total daily insulin dose is calculated, two-thirds of the daily dose is given before breakfast, divided into two-thirds neutral protamine hagedorn (NPH) insulin and one-third regular insulin, and the remaining one-third of the daily dose is divided into half regular insulin before dinner and half NPH insulin at bedtime. Very-short-acting insulin can also be used, but is best dosed with each meal in place of the twice-daily regular insulin. Further discussion of insulin types and regimens (see the article by Gabriella Pridjian elsewhere in this issue for further exploration of this topic) and in other published reviews.\textsuperscript{57,58}

In the twenty-first century, oral hypoglycemic agents have been included in the armamentarium of treatment modalities for GDM (Table 5). Earlier concerns with use of these agents in pregnancy were the unknown risk of teratogenicity and neonatal hypoglycemia caused by transplacental passage. In 2000, Langer and colleagues\textsuperscript{59} published a small but landmark study describing the use of glyburide for treatment of GDM. Women from 11 to 33 weeks of gestation with GDM were randomized to treatment
with glyburide or insulin. There were no significant differences between the insulin treated group (201 women) and the glyburide group (203 women) in demographics and other characteristics, blood glucose concentrations, or neonatal outcomes. Glyburide was started at 2.5 mg in the morning and increased weekly to a maximum of 10 mg twice a day. The investigators concluded that glyburide is a clinically effective alternative to insulin therapy. In a retrospective study, Jacobson and colleagues\(^6\) compared women with GDM treated with glyburide with those treated with insulin and noted that women in the glyburide group were more likely to achieve mean fasting and postprandial glucose goals and had newborns with similar weights and that the newborns were less likely to be admitted to the neonatal intensive care unit. The glyburide group had a higher rate of preeclampsia and need for phototherapy treatment of their newborns. In a different report, these investigators noted a somewhat higher risk of neonatal hypoglycemia with glyburide therapy,\(^6\) but neonatal hypoglycemia may have been related to the higher rate of macrosomic infants in the group studied.

Glyburide does not seem to cross the placenta when studied in an in vitro isolated, perfused cotelydon model\(^6\) and may actually be actively transported from fetal to maternal circulations. However, other investigators\(^6\) have noted that the umbilical cord/maternal plasma ratio of glyburide is 0.7 ± 0.4, suggesting transfer across the placenta and no active transport back.

Failures of glyburide treatment can be predicted. Kahn and colleagues\(^6\) reviewed 95 women with GDM in their diabetes clinic who were treated with glyburide. Of the 95 women, 19% failed glyburide treatment. Failures were more likely in women

<table>
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<th>Table 5</th>
<th>Pharmacologic agents for gestational diabetes</th>
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<tr>
<td></td>
<td>Insulin</td>
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<tr>
<td>Mechanism of Action</td>
<td>Receptor-mediated glucose uptake; other actions</td>
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<tr>
<td>Onset of Action</td>
<td>Varies</td>
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<tr>
<td>Peak</td>
<td>Varies</td>
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<tr>
<td>Dosing</td>
<td>Varies</td>
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<tr>
<td>Placental Transport</td>
<td>Minimal (only antibody-bound fraction)</td>
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<tr>
<td>FDA Pregnancy Category</td>
<td>B(^a)</td>
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<tr>
<td>Experience with Use in Pregnancy</td>
<td>Substantial</td>
</tr>
<tr>
<td>Failure Rate Requiring Insulin</td>
<td>20%</td>
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Abbreviation: FDA, Food and Drug Administration.

\(^{a}\) Certain newer insulin analogs category C.

\(^{b}\) Insufficient evidence at present to recommend use in pregnancy.

\(^{c}\) Minimal experience with use at less than 11 weeks of gestation; insufficient number of large studies addressing neonatal risk.
diagnosed early in pregnancy, of older age and higher parity, and with higher fasting glucose levels, reflecting reduced beta-cell function and reduced capacity to respond to insulin secretagogues. These factors should be considered with counseling or initiating glyburide therapy. Glyburide therapy alone is not likely to achieve optimal blood sugar control if the fasting glucose level is greater than 140 mg/dL and may not even achieve optimal control if fasting glucose level is between 120 to 140 mg/dL.

Use of glyburide is not without pitfalls. Some practitioners and women have begun to believe that diabetes is not a critical complication of pregnancy because it can be taken care of with a pill. Thus, laxity in diet and compliance may occur more often. Experience with glyburide use in the first trimester, during embryogenesis, is limited, and safety in later trimesters should not automatically be extended to the early first trimester. Furthermore, glyburide may not be the ideal oral hypoglycemic agent for pregnancy. Its absorption and steady state and associated insulin secretion do not mimic the in vivo state. The ideal oral hypoglycemic agent for use in pregnancy is one that is not teratogenic, does not cross the placenta, and exerts its peak effect quickly after ingestion, mimicking in vivo insulin secretion and designed to be taken before each meal.

Metformin has been studied recently for treatment of GDM, because women often present to the obstetrician already pregnant and on metformin for treatment of polycystic ovarian syndrome, infertility, or metabolic syndrome. Rowan and colleagues performed a randomized controlled trial of metformin versus insulin for treatment of GDM. A total of 363 women were assigned to metformin; 92.6% continued metformin until delivery, but 46.3% required supplemental insulin to achieve euglycemia. Neonatal outcomes were similar in each group, and women preferred metformin treatment even if insulin was added. In a randomized, controlled study, Moore and colleagues compared the use of metformin with that of glyburide for the treatment of women with GDM. If glycemic control was achieved, women treated with metformin were comparable with women treated with glyburide in outcomes studied. However, failure of metformin therapy was 2.1 times higher than failure of glyburide therapy. Of the metformin group, 34.7% of women eventually required insulin, but only 16% of the glyburide group required insulin. The investigators speculated that ethnic differences may influence success of metformin.

Until more information is obtained regarding safety and efficacy of metformin use in pregnancy, the best approach is to not use metformin for treatment of GDM. If a woman is already on metformin for other reasons, it is best to discontinue its use and perform diabetes screening at the appropriate time as indicated by risk factors or universal screening. Women on metformin for treatment of type 2 diabetes are best changed to insulin if unexpected pregnancy occurs.

ANTENATAL AND INTRAPARTUM MANAGEMENT

Once GDM is diagnosed, the pregnant woman should be seen at least every 1 to 2 weeks, more frequently if other complications ensue. Frequency and timing of antenatal testing in women with GDM is controversial. Generally, women on diet control who do not have macrosomic infants can wait until 40 weeks for antenatal testing; their risk of stillbirth is not substantially higher than the general population. It is prudent to manage women who are noncompliant, require pharmacologic therapy, have macrosomic or growth-restricted fetuses, or have other pregnancy complications similar to those women with preexisting diabetes and initiate antenatal testing. Close assessment of symptoms, blood pressure, and proteinuria to diagnose preeclampsia is paramount.

The timing and mode of delivery of women with GDM is also controversial given the lack of sufficient data to support a specific recommendation. There is no evidence to
support delivery before 40 weeks of gestation. However, some investigators have found a higher incidence of shoulder dystocia by waiting for delivery until after 40 gestational weeks.\textsuperscript{67} Induction of labor at 39 gestational weeks in women with good metabolic control should not require documentation of fetal lung maturity by amniocentesis.\textsuperscript{68} Documentation of fetal lung maturity is prudent if delivery is electively planned earlier without other obstetric indications.

Women with GDM requiring pharmacologic therapy are best managed with intravenous insulin drips and glucose monitoring protocols during labor similar to women with pregestational diabetes.\textsuperscript{57} Women with very mild GDM may not require insulin therapy but should have blood glucose assessment during labor.

In light of the somewhat poor prediction of macrosomia by ultrasonography and the higher rate of shoulder dystocia in GDM infants when compared with non-GDM infants of comparable size,\textsuperscript{69} a fetal weight cutoff for vaginal delivery has not been easy to establish. The current recommendation is to offer women with GDM whose estimated fetal weight is 4500 g or greater elective cesarean to prevent shoulder dystocia. In those women whose fetal weight ranges from 4000 to 4500 g, clinical pelvimetry and other obstetric factors should assist in the decision to offer cesarean section.\textsuperscript{31}

**POSTPARTUM MANAGEMENT**

Many women who are diagnosed with type 2 diabetes are classified first as having GDM, even though they really have undiagnosed pregestational diabetes; these women continue to be diabetic in the postpartum period. Women with GDM should have a fasting or random blood sugar level test in the immediate postpartum period to identify undiagnosed type 2 diabetes. There is epidemiologic evidence that about 15\% to 50\% of women with GDM develop diabetes or impaired glucose tolerance well after pregnancy. A 75-g glucose, 2-hour glucose tolerance test should be performed at or around the time of the routine postpartum visit. The frequency of subsequent testing for detection of glucose intolerance or type 2 diabetes ranges from annually to triannually. The American Diabetes Association recommends glucose tolerance testing at least once every 3 years,\textsuperscript{70} even though more frequent testing might be appropriate if further pregnancies are contemplated.

It is not surprising that there is marked variability in the proportion of women with GDM who are screened postpartum as well as in the type of screening used.\textsuperscript{71–73} Ferrara and colleagues\textsuperscript{73} showed that between 1995 and 2006, the proportion of women in their study who were screened postpartum increased from 20.7\% (95\% confidence interval [CI], 17.8–23.5) to 53.8\% (95\% CI, 51.3–56.3). Independent predictors of successful postpartum screening in their study were women who were older, of Asian or Hispanic ethnicity, better educated, and diagnosed with GDM earlier in gestation. Obese women and women of low parity were less likely to have postpartum screening.

There are considerable data to support that weight loss and use of metformin or thiazolidinediones can prevent or delay progression of glucose intolerance and type 2 diabetes.\textsuperscript{74,75} Dietary modifications and treatment of periodontal disease may also prevent glucose intolerance.\textsuperscript{47} Additional research and specific clinical guidelines for women with history of GDM will allow interventional strategies to prevent or delay the onset of type 2 diabetes.

**REFERENCES**


