

Gestational Diabetes

By Henci Goer

What is gestational diabetes?

Gestational diabetes (GD) simply means elevated blood sugar during pregnancy. To understand it, you must first understand the normal changes in pregnancy metabolism^{3,4}. When you are pregnant, certain hormones make your insulin less effective at transporting glucose, the body's fuel, out of your bloodstream into your cells. This increases the amount of circulating glucose, making it available to your baby for growth and development. This "insulin resistance" increases as pregnancy advances. As a result, your blood glucose levels after eating rise linearly throughout pregnancy. By the third trimester, you will tend to have higher blood glucose levels after eating than nonpregnant women (hyperglycemia), despite secreting normal and above normal amounts of insulin. During overnight sleep, the excess insulin has a chance to mop up, which causes morning glucose levels to be lower on average than in nonpregnant women (hypoglycemia).

In the 1950s, some researchers wondered whether sugar values at the high end of the range for pregnancy would predict the development of diabetes later in life. They tracked a population of women and in 1964, they reported that, yes, it did⁴⁰. The extra stress of pregnancy revealed a woman's "prediabetic" status. This shouldn't have surprised anyone, because high-weight women are much more likely to have higher glucose values in pregnancy than average-weight women and to eventually develop diabetes. However, doctors knew diabetes posed grave threats to the unborn baby, so they worried that glucose levels that were high, but not in the diabetic range, might also do harm. This concern launched what eventually became an avalanche of studies that ended by defining a whole new category of pregnancy complication called "gestational diabetes," although "glucose intolerance of pregnancy" would be a more accurate description. Those studies, and their premise, were fundamentally flawed.

Is gestational diabetes a health risk?

The theory that GD could have the same adverse effects of diabetes was faulty on its face, because GD does not share the risk factors of either type of true diabetes. In Type I diabetes, extremes of low and high blood sugar early in pregnancy can cause malformations or miscarriage. GD women make normal or above-normal amounts of insulin and have normal blood-sugar metabolism in the first trimester²². Either Type I or II, long-standing diabetes can damage maternal blood vessels and kidneys, causing hypertension or kidney complications. These can in turn jeopardize the fetus. Gestational diabetics do not have long-standing diabetes. The one problem GD shares with both types is that chronic hyperglycemia can overfeed the fetus, resulting in a big baby. This is generally defined as a birth weight of more than 8 lbs. 13 oz. (4,000 grams) or a birth weight in the upper ten percent for length of pregnancy (large for gestational age).

Theory aside, the studies designed to test it had significant weaknesses. They included women who were known diabetics prior to pregnancy. They selected women for glucose testing based on such risk factors as prior stillbirth, current hypertension, or extreme overweight, indications that alone could explain poorer outcomes^{1,2}. They failed to account for compounding factors, such as that glucose intolerance associates with increasing maternal weight and age, which themselves strongly predict large babies and maternal hypertension. Finally, they used management protocols that increased risks such as starvation diets, early induction and withholding nourishment from the newborn^{1,8}. Despite these flaws, researchers concluded that mildly deviant glucose values in pregnancy caused serious harm.

We now know that GD doesn't increase the risk of stillbirth or congenital malformations⁴. A couple of modern studies have concluded otherwise, but they didn't take into account that women with high blood sugar are more likely to have other risk factors for poor outcome, or that some women had undiagnosed diabetes prior to pregnancy^{17,24}. Indeed, the fact that these studies were of women whose blood sugar had been normalized by

treatment proves that GD is not the culprit. Besides, GD testing and treatment could not affect the incidence of congenital malformations under any circumstances, because testing isn't done until the third trimester. By that time, the baby is long since fully formed.

We also know that maternal glucose level correlates poorly with birth weight. While GD somewhat increases the odds of having a baby weighing in the upper ten percent^{16,36}, most of this results from GD's association with other factors, in particular, maternal weight^{10,13,21,28,43,57}.

Other supposed risks of GD are preeclampsia, glucose intolerance in the child and childhood obesity. As before, GD is only found in company with these complications; it doesn't cause them. For example, studies show that blood glucose level plays little if any role in high-weight children compared with maternal weight before pregnancy^{8,25}. Also, as before, normalizing blood sugar fails to prevent these problems, which absolves GD^{42,44-45,53}.

All this being said, there is a needle in the haystack. About one in a thousand pregnant women tested will have sugar values in the range of true diabetes². These women may have been diabetic before pregnancy and not known it, or pregnancy may have been enough of a metabolic stress to tip them into diabetes. These women may benefit from being identified and treated.

How do practitioners test for gestational diabetes?

Testing for GD is a two-stage process. The first step is a screening test, which is generally administered to all pregnant women. The screening test is usually given somewhere between week 24 and 28. For this test, you may be asked to drink a glucose solution and have a blood sample drawn an hour later, or you may simply be asked to give a blood sample. If your blood glucose value exceeds a threshold amount, you will be asked to return for an Oral Glucose Tolerance Test (OGTT). The various protocols disagree on the amount of glucose and the threshold value²⁹.

For the OGTT, you will be asked to come in after fasting overnight. Blood will be drawn, you will be given a glucose solution to drink, and blood will be drawn one, two and three hours later. The glucose solution may make you nauseous. As with the screening test, the recommended amount of glucose and the diagnostic thresholds vary from protocol to protocol²⁹. Some guidelines only stipulate a fasting glucose and a two hour value²⁹.

What are the problems with gestational diabetes testing?

A diagnostic test should be reproducible, meaning you get the same results when you repeat the test. Thresholds should be values at which complications either first appear or incidence greatly increases; and normal ranges should apply to the population being tested. The OGTT is none of the above.

Obstetricians adopted data from the original 1950s studies as the normative curve for all pregnant women, but they shouldn't have. For one thing, those researchers tested women without regard to length of gestation, whereas today, doctors typically test women at the beginning of the third trimester. Glucose values rise linearly throughout pregnancy, but no corrections have been made for this¹⁵. For another, they studied a population that was sixty percent white and forty percent black. Hispanics, Native Americans and Asian women average higher blood sugars than black or white women^{10,57}. This means values for that 1950s population have been established as norms for all women, which in turn means that some women are being identified as diseased simply because of race.

The OGTT also isn't reliable. When pregnant women undergo two OGTTs a week or so apart, individual test results disagree twenty to twenty-five percent of the time^{5,23}. A person's blood sugar values after ingesting glucose (or food) vary widely depending on many factors. For this reason, the OGTT has been abandoned as a diagnostic test for true diabetes in favor of high fasting glucose values, which show much greater consistency, or values after eating of 200 mg/dl or more, which are rare^{46,52}. Moreover, pregnancy compounds problems with reproducibility. Because glucose levels rise linearly throughout pregnancy, a woman could "pass" a test in gestational week 24

and “fail” it in week 28⁵⁵. These same reproducibility problems hold true for the glucose screening test that precedes the OGTT^{47,55}.

More importantly, no threshold has ever been demonstrated for onset or marked increase in fetal complications below levels diagnostic of true diabetes. The original researchers chose their cutoffs for convenience in follow-up, but all studies since have used their criteria or some modification thereof as a threshold for pathology in the current pregnancy. Numerous studies since have documented that birth weights and other outcomes fail to correlate with the 1950s or anybody else’s thresholds. Today’s researchers acknowledge that the risks of glucose intolerance almost certainly form a continuum and that screening and diagnostic thresholds are arbitrary^{7,29-30,48,51}.

Several organizational bodies that have looked critically at the GD research have come out against GD testing. A Guide to Effective Care in Pregnancy and Childbirth, the bible of evidence-based care, relegates screening for gestational diabetes to “Forms of Care Unlikely to be Beneficial¹².” The American College of Obstetricians and Gynecologists says no data support the benefits of screening¹. The U.S. Preventative Services Task Force and the Canadian Task Force on the Periodic Health Examination both conclude that there is insufficient evidence to justify universal GD screening^{4,11}.

How is gestational diabetes treated?

The main elements of GD treatment are:

- Normalizing blood sugar: The first step is a diet low in sugars and carbohydrates. Some diets also limit calories. If diet fails to control blood glucose levels, insulin injections are prescribed.
- Monitoring blood sugar: In most cases this will mean pricking your finger and testing your blood once, and more commonly, several times a day.

Many protocols include:

- Monitoring fetal well-being: Many practitioners order repeated fetal surveillance tests beginning at or before the due date. The most common is the nonstress test, which looks at the fetal heart rate changes in response to fetal movements or Braxton-Hicks contractions (normal, nonlabor tightening of the uterus).
- Ultrasound scan to estimate fetal weight
- Planned delivery: This may be either induction of labor or elective cesarean section. Induction is often at, or sometimes before, the due date.
- Monitoring newborn blood sugar: Some protocols call for checking the baby’s blood sugar, which involves a heel stick.

What are the problems with gestational diabetes treatment?

The two questions asked of any therapy are: “Is it safe?” and “Is it effective?” GD management is neither.

GD treatment per se has never been shown to have benefits. In fact, it is virtually untested. The first and only random assignment trial, the standard for determining care because this design eliminates many sources of bias and ensures similar groups, was published in 1997. It concluded that intensive treatment offered no advantages over advising women to eat healthy¹⁶. Meanwhile, several studies have found that identification as a gestational diabetic in and of itself substantially increases the odds of cesarean section^{3,19,38,50}.

Individual components of GD protocols also fail the safety/effectiveness test:

- Diet or diet plus insulin therapy: The standard GD diet is a healthy diet. However, while it reduces blood glucose to normal range in most women, it has little or no effect on birth weight⁵⁴. Many women, though, are prescribed limited calorie diets. Reducing calorie intake by more than one-third causes the body to switch to a starvation metabolism (ketosis) that produces byproducts known to be harmful to the baby³¹. Limiting food intake can also lead to malnutrition²⁷. Aggressive insulin use can cause underweight babies and symptomatic episodes of low blood sugar (hypoglycemia)^{3,32}. A Guide to Effective Care in Pregnancy

and Childbirth lists both diet treatment and diet plus insulin treatment under “Forms of Care Unlikely to be Beneficial^{1,2}.”

- Tests of fetal well-being: Of the four random assignment trials of nonstress testing, the most commonly used fetal surveillance test, none found any benefit for testing, although they were in populations of women at moderate to high risk⁴¹. All tests of well-being have high false-positive rates, meaning the test says there is a problem when there isn't. This leads to unnecessary inductions and cesareans with all their attendant risks.
- Fetal weight estimates: Ultrasound predictions that the baby will weigh over 4,000 grams are wrong one-third to one-half of the time^{6,9,14,20,33,56}. As with fetal well-being tests, the belief that the baby is big leads to unnecessary inductions and cesareans. Two studies showed that when obstetricians believed, based on ultrasound, that women were carrying babies weighing over 4,000 grams, half had cesareans, versus less than one-third of women not thought to have babies this big, but who actually did^{35,56}.
- Induction of labor or planned cesarean: Many doctors induce labor in the belief it averts cesareans due to big babies. Some think induction or planned cesarean prevents shoulder dystocia (the head is born, but the shoulders hang up). Studies of induction and planned cesarean for suspected big baby show no benefits for either practice^{6,9,14,20,33,49,56}.
- Monitoring newborn blood sugar: The reasoning behind this is that if the mother has high blood-sugar levels, the baby will produce extra insulin. After birth, this excess insulin can cause low blood sugar. No studies have tested whether checking the blood sugar of a baby who shows no symptoms of low blood sugar has any value. However, test results can lead to the baby being given a bottle of sugar water or formula, which interferes with establishing breastfeeding, separation from the mother for observation in the nursery, or both.
- Finally, treatment also fails to prevent increased incidence of preeclampsia, impaired glucose tolerance in children, and childhood overweight^{42,44-45,53}.

Another rationale given for diagnosing and treating gestational diabetics is identifying women at risk for developing Type II diabetes. However, predicting who is likely to develop diabetes can be done equally well on the basis of race, ethnicity, and weight.

Curiously, while several prominent GD researchers and experts acknowledge the lack of sound data supporting their recommendations, none have backed off^{1,26,37,39}. These experts devise GD guidelines for practicing doctors and midwives, most of whom have no idea how shaky the GD edifice is. Even those who doubt the value of screening all or most women for GD may have little choice if testing and treatment is the community standard of care.

How does diagnosis as a gestational diabetic affect your pregnancy and birth?

The standard GD diet is a good one; adequate calories, limit simple sugars, moderate fat intake, eat whole grains and plenty of fruits and vegetables and eat smaller meals more frequently. Also beneficial is the advice to engage in moderate, regular exercise. If that was all that happened, identification as a gestational diabetic would be a good thing. Some tracking of blood sugars to make sure they aren't drifting into the true diabetic range is probably also a good thing, as is identifying the one in a thousand women who has or will develop glucose values in that range. However, most women will find themselves caught up in frequent doctor visits, multiple daily blood tests, restrictive diets, possibly insulin injections, repeated fetal surveillance tests and a considerable chance of a labor induction or cesarean section.

REFERENCES

1. ACOG. Diabetes and pregnancy. Technical Bulletin No. 200, 1994.
2. Beischer NA, Wein P, and Sheedy MT. Studies of postnatal diabetes mellitus in women who had gestational diabetes. Part 1. Estimates of the prevalence of unrecognized prepregnancy diabetes mellitus. *Aust N Z J Obstet Gynaecol* 1997;37(4):412-9.
3. Buchanan TA et al. Use of fetal ultrasound to select metabolic therapy for pregnancies complicated by mild gestational diabetes. *Diabetes Care* 1994;17(4):275-283.
4. Canadian Task Force on the Periodic Health Examination. Periodic health examination, 1992 update: 1. Screening for gestational diabetes mellitus. *Can Med Assoc J* 1992;147(4):435-43.
5. Catalano PM et al. Reproducibility of the oral glucose tolerance test in pregnant women. *Am J Obstet Gynecol* 1993;169(4):874-881.
6. Combs CA, Singh NB, and Khoury JC. Elective induction versus spontaneous labor after sonographic diagnosis of fetal macrosomia. *Obstet Gynecol* 1993;81(4):492-6.
7. Coustan DR and Carpenter MW. The diagnosis of gestational diabetes. *Diabetes Care* 1998;21 (Suppl 2):B5-8.
8. Dang K, Homko C, and Reece EA. Factors associated with fetal macrosomia in offspring of gestational diabetic women. *J Matern Fetal Med* 2000;9(2):114-7.
9. Delpapa EH and Mueller-Heubach E. Pregnancy outcome following ultrasound diagnosis of macrosomia. *Obstet Gynecol* 1991;78(3):340-3.
10. Dye TD et al. Physical activity, obesity, and diabetes in pregnancy. *Am J Epidemiol* 1997;146(11):961-5.
11. Ecker JL, Mascola MA, and Riley LE. Gestational diabetes. *N Engl J Med* 2000;342(12):896-7.
12. Enkin M et al. *A Guide to Effective Care in Pregnancy and Childbirth*, 3rd ed. Oxford: Oxford University Press, 2000.
13. Farmer G et al. The influence of maternal glucose metabolism on fetal growth, development and morbidity in 917 singleton pregnancies in nondiabetic women. *Diabetologia* 1988;31:134-141.
14. Friesen CD, Miller AM, and Rayburn WF. Influence of spontaneous or induced labor on delivering the macrosomic fetus. *Am J Perinatol* 1995;12(1):63-6.
15. Forest JM et al. Reference values for the oral glucose tolerance test at each trimester of pregnancy. *Amer J Clin Pathol* 1983;80:828-31.
16. Garner P et al. A randomized controlled trial of strict glycemic control and tertiary level obstetric care versus routine obstetric care in the management of gestational diabetes: a pilot study. *Am J Obstet Gynecol* 1997;177(1):190-5.
17. Girz BA, Divon MY, and Merkatz IR. Sudden fetal death in women with well-controlled, intensively monitored gestational diabetes. *J Perinatol* 1992;12(3):229-33.
18. Goer H. Gestational diabetes. *International Journal of Childbirth Education*. 1991;6(4):1991.
19. Goldman M et al. Obstetric complications with GDM. Effects of maternal weight. *Diabetes* 1991;40(Suppl 2):79-82.
20. Gonen O et al. Induction of labor versus expectant management in macrosomia: a randomized study. *Obstet Gynecol* 1997;89(6):913-7.
21. Green JR et al. Influence of maternal body habitus and glucose tolerance on birth weight. *Obstet Gynecol* 1991;78(2):235-240.
22. Hadden D. Approaches to screening. In *Carbohydrate Metabolism in Pregnancy and the Newborn*. Stowers JB and Sutherland HW, eds. Edinburgh: Churchill Livingstone, 1984.
23. Harlass FE, Brady K, Read JA. Reproducibility of the oral glucose tolerance test in pregnancy. *Am J Obstet Gynecol* 1991;164(2):564-568.
24. Hawthorne G, Snodgrass A, Tunbridge M. Outcome of diabetic pregnancy and glucose intolerance in pregnancy: an audit of fetal loss in Newcastle General Hospital 1977-1990. *Diabetes Res Clin Pract* 1994;25(3):183-90.
25. Hiramatsu Y et al. Heavy-for-date infants: their backgrounds and relationship with gestational diabetes. *J Obstet Gynaecol Res* 2000;26(3):193-8.
26. Hoffman L et al. Gestational diabetes mellitus--management guidelines. The Australasian Diabetes in Pregnancy Society. *Med J Aust* 1998;169(2):93-7.
27. Jovanovic L. American Diabetes Association's Fourth International Workshop-Conference on Gestational Diabetes Mellitus: summary and discussion. *Diabetes Care* 1998;21 (Suppl 2):B131-7.
28. Keen H. Gestational diabetes. Can epidemiology help? *Diabetes* 1991;40(Suppl 2):3-7.
29. Khandelwal M, Homko C, and Reece EA. Gestational diabetes mellitus: controversies and current opinions. *Curr Opin Obstet Gynecol* 1999;11(2):157-65.
30. Kjos SL and Buchanan TA. Gestational diabetes mellitus. *N Engl J Med* 1999;341(23):1749-56.
31. Knopp RH et al. Metabolic effects of hypocaloric diets in management of gestational diabetes. *Diabetes* 1991;40(Suppl 2):165-171.
32. Langer O et al. Intensified versus conventional management of gestational diabetes. *Am J Obstet Gynecol* 1994;170(4):1036-1047.
33. Larsen JS, Pedersen OD, and Ipsen L. Induction of labor when a large fetus is suspected. *Ugeskr Laeger* 1991;153(3):181-3.
34. Lesser KB and Carpenter MW. Metabolic changes associated with normal pregnancy and pregnancy complicated by diabetes mellitus. *Seminars Perinatol* 1994;18(5):399-406.
35. Levine AB et al. Sonographic diagnosis of the large for gestational age fetus at term: does it make a difference? *Obstet Gynecol* 1992;79(1):55-8.
36. Li DFH et al. Is treatment needed for mild impairment of glucose intolerance in pregnancy? A randomized controlled trial. *Br J Obstet Gynaecol* 1987;94:851-4.

37. Metzger BE and Coustan DR. Summary and recommendations of the Fourth International Workshop-Conference on Gestational Diabetes Mellitus. The Organizing Committee. *Diabetes Care* 1998;21(Suppl 2):B161-7.
38. Naylor CD et al. Cesarean delivery in relation to birth weight and gestational glucose tolerance: pathophysiology or practice style? *JAMA* 1996;275(15):1165-70.
39. Okun N, Verma A, and Demianczuk N. Gestational diabetes mellitus. Unresolved issues and future research directions. *Can Fam Physician* 1997;43(1):88-93.
40. O'Sullivan JB and Mahan CM. Criteria for the oral glucose tolerance test in pregnancy. *Diabetes* 1964;13(4):278-285.
41. Pattison N and McCowan I. Cardiotocography for antepartum fetal assessment (Cochrane Review). In: *The Cochrane Library*, Issue 4, 2000. Oxford: Update Software.
42. Persson B and Hanson U. Neonatal morbidities in gestational diabetes mellitus. *Diabetes Care* 1998;21(Suppl 2):B79-84.
43. Phillipou G. Relationship between normal oral glucose tolerance test in women at risk for gestational diabetes and large for gestational age infants. *Diabetes Care* 1991;14(11):1092-1094.
44. Plagemann A et al. Glucose intolerance and insulin secretion in children of mothers with pregestational IDDM or gestational diabetes. *Diabetologia* 1997;40(9):1094-100.
45. Plagemann A et al. Overweight and obesity in infants of mothers with long-term insulin-dependent diabetes or gestational diabetes. *Int J Obes Relat Metab Disord* 1997;21(6):451-6.
46. Rubenstein E and Federman D, eds. *Scientific American Medicine* 9 VI:8-10. New York: Scientific American, 1986.
47. Sacks DA et al. How reliable is the fifty-gram, one-hour glucose screening test? *Am J Obstet Gynecol* 1989;161(3):642-645.
48. Sacks DA et al. Toward universal criteria for gestational diabetes: the 75-gram glucose tolerance test in pregnancy. *Am J Obstet Gynecol* 1995;172:607-14.
49. Sandmire HF and Woolley RJ. Macrosomia: can we prevent big problems with big babies? *Birth* 1998;25(4):263-7.
50. Santini DL and Ales KL. The impact of universal screening for gestational glucose intolerance on outcome of pregnancy. *Surg Gynecol Obstet* 1990;170(5):427-436.
51. Schwartz ML, Ray WN, and Lubarsky SL. The diagnosis and classification of gestational diabetes mellitus: is it time to change our tune? *Am J Obstet Gynecol* 1999;180:1560-71.
52. Unger R and Foster D. Diabetes mellitus. In *Williams Textbook of Endocrinology*. Wilson J and Foster D, eds. Philadelphia: W B Saunders, 1985.
53. Vohr BR and McGarvey ST. Growth patterns of large-for-gestational age and appropriate-for-gestational-age infants of gestational diabetic mothers and control mothers at age 1 year. *Diabetes Care* 1997;20(7):1066-72.
54. Walkinshaw SA. Dietary regulation for 'gestational diabetes' (Cochrane Review). In: *The Cochrane Library*, Issue 3, 2000. Oxford: Update Software.
55. Watson WJ. Serial changes in the 50-g oral glucose test in pregnancy: implications for screening. *Obstet Gynecol* 1989;74(1):40-43.
56. Weeks JW, Pitman T, and Spinnato JA 2nd. Fetal macrosomia: does antenatal prediction affect delivery route and birth outcome? *Am J Obstet Gynecol* 1995;173(4):1215-9.
57. Yue DK et al. Why does ethnicity affect prevalence of gestational diabetes? The underwater volcano theory. *Diabet Med* 1996;13(8):748-52.

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WE NEED NEW CLASSIFICATIONS... THERE IS NOW BIRTH WITH LOVE HORMONES AND BIRTH WITHOUT LOVE HORMONES. THE QUESTION IS, CAN HUMANITY SURVIVE WITHOUT LOVE HORMONES?

DUDE, HE JUST BLEW MY MIND.

WE ARE TALKING ABOUT THE FUTURE OF HUMANITY... CAN HUMANITY SURVIVE THE SAFE CAESAREAN?

MICHEL ODENT
MARCH 9, 2008
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