Management of Obesity in Pregnancy

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Maternal pregravid obesity is a significant risk factor for adverse outcomes during pregnancy. In early pregnancy there is an increased risk of spontaneous abortion and congenital anomalies. In later gestation maternal metabolic manifestations of the metabolic syndrome, such as gestational hypertensive disorders and diabetes, become clinically recognized because of the increased insulin resistance in obese compared with nonobese women. In women with pregestational glucose intolerance, hypertension, central obesity, and lipid disorders, the physiologic changes in pregnancy increase the risk of problems previously not routinely encountered during pregnancy. These include chronic cardiac dysfunction, proteinuria, sleep apnea, and nonalcoholic fatty liver disease. At parturition the obese patient is at an increased risk of cesarean delivery and associated complications of anesthesia, wound disruption, infection, and deep venous thrombophlebitis. For the fetus there are short-term risks of fetal macrosomia, more specifically obesity, and long-term risks of adolescent components of the metabolic syndrome. Although preliminary results of bariatric surgery are encouraging, the procedure is expensive and not for all obese women, and we recognize that long-term follow-up data on offspring of obese women who have undergone bariatric surgery before pregnancy are lacking. In the interim, we need to encourage obese women to lose weight before conception, using lifestyle changes if possible. During pregnancy, weight gain should be limited to Institute of Medicine guidelines (currently under review) and encouragement given for physical activity.

(Obstet Gynecol 2007;109:419–33)

Obesity is an epidemic not only in the United States and developed countries but also in the developing world. “Indeed, it is now so common that it (obesity) is replacing the more traditional public healthcare concerns including under-nutrition and infectious disease as one of the most significant contributors to ill health.”1 The World Health Organization and the National Institutes of Health define normal weight as a body mass index (BMI, weight [kg]/height [m]²) of 18.5–24.9, overweight as a BMI of 25–29.9, and obesity as a BMI of 30 or greater.1 Obesity is further characterized by BMI into class I (30–34.9), class II (35–39.9), and class III (greater than 40).1 In women of reproductive age in the United States, the prevalence of obesity was 30.2%, while the prevalence of overweight was 56.7% in the latest Centers for Disease Control and Prevention (CDC) reports. The problem of obesity is greatest among non-Hispanic black women (48.8%), as compared with Mexican-American (38.9%) and non-Hispanic white women (31.3%). Potentially more important, the prevalence of obesity in children as young as 2 years old and adolescents has increased by 11.3% between 1994 and 2000.2 Again, the increased prevalence has been especially notable among Mexican-American and non-Hispanic black adolescents.

The problems relating to the management of obesity in pregnancy are many. There are both short- and long-term complications and implications for both mother and fetus. This issue has recently been addressed in the American College of Obstetricians and Gynecologists (ACOG) in Committee Opinion No. 315, “Obesity in Pregnancy.”3 I will attempt to first comment on the Committee Opinion, and secondarily discuss the potential implications of obesity for the woman and her offspring.
Obesity is a risk factor for a number of pregnancy complications. Therefore, as recommended by the ACOG committee opinion, obese women should be encouraged to decrease weight before considering pregnancy. Also recently, ACOG Committee Opinion No. 319, “The Role of the Obstetrician-Gynecologist in the Assessment and Management of Obesity,” offers the obstetrician-gynecologist practical guidelines on how to assess and manage obesity in the nonpregnant woman. Included are primers on the assessment of a patient’s readiness to make behavioral changes. Lifestyle measures of calorie-restricted diets and exercise, when employed together, are potentially more beneficial than either modality alone. As noted in Committee Opinion No. 319, fad diets, even ones with a potential physiological basis such as low-glycemic diets, are controversial at best with respect to long-term efficacy. Also included is information regarding approved weight-loss medications and guidelines for referral for bariatric surgery evaluation, which will be discussed later.

Although preconceptual weight loss is certainly a laudable goal for the obese woman, many, if not most, pregnancies are unfortunately not planned. The limited success in normalization of pregravid glucose control in women with pregestational diabetes to decrease the risk of congenital malformations is an analogous example of the limitation of preconceptual management of lifestyle issues. Long-term public health programs addressing awareness of the problems of obesity, like those programs in recent decades that promoted smoking cessation programs and legislation, hold promise for success in the future. In the meantime we need not give up hope, because some obese patients with the proper counseling can achieve meaningful weight loss before conception.

PREGRAVIS OBESITY VERSUS WEIGHT GAIN IN PREGNANCY

The obstetric complications of maternal obesity are generally related to issues of maternal pregravid obesity rather than excessive weight gain during gestation that results in a nonobese women becoming obese. Weight gain in pregnancy is generally considered to be the difference between a woman’s weight at the last antenatal visit and her pregravid weight or her weight at first antenatal visit. However, the concept of “net maternal weight gain” has gathered more interest, because this takes into account the fact that, on average, 4–5 kg of weight at term represents the fetus (3.5 kg), the placenta (0.5 kg), and amniotic fluid (0.5–1.0 kg). Therefore, one could easily express “net maternal weight gain” as the difference between a woman’s weight at her last antenatal visit minus the combination of her pregravid weight and fetal weight.

The recommendations for weight gain in pregnancy have been based on the Institute of Medicine (IOM) guidelines that were published in 1990. The suggested weight gains are a weight gain of 11.2–15.9 kg (25–35 lb) for women with a normal BMI, 6.8–11.2 kg (15–25 lb) for overweight women, and more than 6.8 kg (15 lb) for obese women. These guidelines were initially intended to help decrease the risk of fetal growth restriction. In a report by Schiefe et al. from the CDC Pregnancy Nutrition Surveillance System, in over 266,000 women, the mean maternal weight gain and net weight gain, even when adjusted for week of gestation, both decrease with increasing BMI. Moreover, overweight and obese women had mean weight gains greater than the IOM guidelines. Based on these data and other data, including the increasing prevalence of obesity in the population, the IOM currently is reviewing the recommendations for weight gain in pregnancy.

The components of weight gain have been previously estimated by Hytnen to be approximately 1 kg of protein and 4 kg of fat, with the remainder being water. In prospective studies initiated before pregnancy in women with a wide range of BMIs, Butte et al. reported a wide range of incremental accretion of fat, depending on a subject’s pregravid BMI. The net accrual of fat mass was 5.3 kg women with low BMI, 4.6 kg in those with normal BMI, and 8.4 kg in the high BMI group. However, there was a wide range in increased fat mass within each group. In our own prospective studies, we found no significant difference in gain of fat mass in lean compared with obese women (4.7±3.2 kg versus 4.2±3.5 kg, \( P=.58 \)), nor lean body mass (7.6±3.9 kg versus 8.8±2.6 kg, \( P=.18 \)) although the increase in percentage of body fat was significantly greater in the lean compared with the obese women (3.3±3.8% versus 0.1±3.3%, \( P=.004 \)). The increase in subcutaneous fat was in a central distribution, i.e., between the midthorax through the upper thigh. Interestingly, there was no significant difference in accretion of fat mass in women with normal glucose tolerance compared with women with gestational diabetes mellitus (GDM) matched for pregravid body composition. There are limited data about the relative changes in visceral fat, which potentially may be of more metabolic significance. Kinoshita and Itoh reported an increase in both preperitoneal and subcutaneous fat layers by the third trimester of pregnancy, as well as an increase in the ratio of peritoneal to subcutaneous fat, suggesting intra-abdominal fat increases during pregnancy.
Hence, the accrual of fat mass in pregnancy is variable and may depend on a woman’s pregravid metabolic status and other lifestyle variables such as diet and physical activity.

OBSERVATIONAL STUDIES AND CORRELATION WITH MATERNAL OBESITY: EARLY GESTATION

The obese woman is at increased risk of a myriad of obstetric problems in early pregnancy. There is an increased risk of early miscarriage (odds ratio [OR] 1.2, 95% confidence interval [CI] 1.01–1.46, *P* = .04) and recurrent miscarriage (OR 3.5, 95% CI 1.03–12.01, *P* = .04) in obese women compared with normal weight controls after natural conception. In overweight women conceiving after in vitro fertilization or intracytoplasmic sperm injection, there was an increase in the abortion rate over the first 6 weeks (22% versus 12%) compared with lean or average weight women. The relative risk of spontaneous abortion was 1.77 (95% CI 1.05–2.97). Therefore, particularly in obese women considering assisted reproductive therapy, weight loss before conception should be strongly considered and measures initiated by the patient’s health care provider.

As early as 1994, Waller et al suggested that offspring of obese women were at increased risk of neural tube defects (OR 1.8, 95% CI 1.1–3.0), especially spina bifida (OR 2.6, 95% CI 1.5–4.5). These results have been confirmed in subsequent studies and have also implicated maternal obesity with increased risks of heart defects (OR 1.18, 95% CI 1.09–1.27) and omphalocele (OR 3.3, 95% CI 1.0–10.3). Because these types of congenital anomalies are often seen with pregestational diabetes, some investigators have suggested that many of these obese women may have had undiagnosed type 2 diabetes. Additionally, because neural tube defects are associated with folic acid deficiencies, Mojtabai reported that, after controlling for intake of folate in food and nutritional supplements, increased BMI was associated with a lower serum folate concentrations (*P* < .001) and suggested that women with a BMI greater than 30 would need to increase their folate consumption by 350 mcg/d to achieve the same folate levels as women with BMI less than 20. In contrast, Ray et al in a Canadian population estimated whether the risk of neural tube defects was lower after flour was fortified with folic acid. Before fortification of flour, increased maternal weight was associated with a modest increased risk of neural tube defects (OR 1.4, 95% CI 1.0–1.8); after flour fortification the risk actually increased (OR 2.8, 95% CI 1.2–6.6). Therefore, although the evidence implicated maternal obesity with an increased risk of congenital anomalies, particularly neural tube defects, the mechanisms are not well understood. Practically, short of preconceptual weight loss in the management of obese pregnant women, one should consider glucose screening of obese women in early pregnancy to rule out undiagnosed pregestational diabetes. Folate supplementation of cereal products was initiated in this country in 1998, and whether additional folate supplementation should be offered to obese women before conception or in early pregnancy is speculative at this point. Obviously, more research must be done, but recognition of obesity as a risk factor for congenital anomalies remains an important factor for the clinician to consider in the management of the obese pregnant woman.

If, indeed, obese women are at increased risk of neural tube defects and other congenital anomalies, how then does maternal obesity affect our diagnostic abilities in this population? There is a significant correlation between maternal serum alpha fetoprotein (AFP) and maternal weight (r = 0.24, *P* < .001), with lighter women having greater maternal serum AFP than heavier women. This is generally believed to be a result of the proportionally greater plasma volume in obese compared with nonobese women. Therefore, standard adjustments of maternal AFP values for maternal weights up to 200 lb have been implemented. Additionally, the use of cell-free DNA in the diagnosis of chromosomal abnormalities may be affected by a woman’s degree of obesity. Watagana et al reported that in the first trimester there was no significant association between maternal weight and plasma-free DNA levels. However, in the second trimester there was a significant inverse correlation between maternal weight and serum-free DNA (r = −0.26, *P* < .007), particularly if the woman weighed more than 170 lb.

The other modality commonly used in early pregnancy to identify congenital anomalies is ultrasonography. In 1990, Wolfe et al reported that there was a significant impairment of adequate ultrasound visualization of fetal anatomy when BMI was greater than 36; visualization fell by 14.5%. A decrease in visualization was most marked for the fetal heart and spine. In 2004, Hendler et al reported that, in over 11,000 pregnancies in which 38.6% of the patients were obese, the rate of suboptimal visualization of fetal anatomy was 37.3% in obese compared with 18.7% in nonobese women (P < .001). Increased severity of obesity was again noted for both cardiac and craniospinal structures. The use of advanced ultrasound equipment may be able to improve suboptimal visualization.
visualization of the outflow tracts in obese women after 18 weeks of gestation but not of the four-chamber view.\textsuperscript{23}

In summary, although obese women may be at increased risk of neural tube defects, interpretation of serum markers is more difficult because of the changes in the volume of distribution of these markers, and care needs to be taken so as not to increase the number of false-negative results. The use of population-specific values may aid in the interpretation of results. Similarly, the ability of ultrasonography to detect fetal cardiac and craniospinal abnormalities is significantly limited in obese women compared with nonobese women. The use of advanced ultrasound equipment and delaying evaluation until after 18 weeks may be of some value, although overall maternal obesity still limits visibility of fetal structures.

**OBSTETRIC RISKS ASSOCIATED WITH MATERNAL OBESITY IN LATE GESTATION: MATERNAL PREGNANCY METABOLIC DYSFUNCTION**

The obese nonpregnant woman is at significant risk for what has variously been termed the metabolic syndrome or insulin resistance syndrome. This syndrome has as its metabolic core obesity, ie, central obesity as estimated by an elevated waist-to-hip ratio and insulin resistance. The clinical manifestations of the metabolic syndrome include hypertension, glucose intolerance, and elevated cholesterol and triglycerides. Maternal obesity in pregnancy is also associated with an increased risk of “metabolic syndrome-like complications” in late pregnancy, for example, gestational hypertension and preeclampsia. In retrospective studies, Sibai et al\textsuperscript{24} have previously reported a significant increase in the risk of preeclampsia in women with increased BMI. Based on a prospective multicenter study of 16,102 women, initially evaluated at 10–14 weeks, 85% were controls (BMI less than 30), 9% were obese (BMI 30–34.9), and 6% were morbidly obese (BMI 35 or greater). Obese women and morbidly obese women were 2.5 and 3.2 times, respectively, more likely to develop gestational hypertension than the control group. Similarly, preeclampsia was 1.6 and 3.3 times more likely to develop in obese and morbidly obese women, respectively.\textsuperscript{25} The increase in preeclampsia in obese compared with average weight women extends to women with GDM as well. Yogev et al\textsuperscript{26} reported that, in women with well-controlled GDM, there is a significant increased risk of preeclampsia in obese (10.8%) compared with average BMI women (8.2%). The risk of preeclampsia is also increased in obese women with GDM with poor control (14.9%). Furthermore, Crowther et al,\textsuperscript{27} in the Australian Carbohydrate Intolerance Study in Pregnant Women (ACHOIS), showed that the risk of preeclampsia in the intervention group for GDM was 12%, whereas in the routine care group the risk was 18% ($P<.02$). In summary, although currently there are no known proven therapies to prevent the development of preeclampsia in obese women, for example, aspirin or calcium supplementation, there are data suggesting that tight glucose control in obese women with GDM may decrease the risk. Additionally, the use of antioxidants for the prevention of preeclampsia in obese women has a theoretical benefit because of the increase in oxidative stress generated from maternal adipose tissue. Two recent randomized controlled trials, however, have reported no benefit of antioxidants (1,000 mg of vitamin C and 400 International Units of vitamin E) in the reduction of preeclampsia in treatment compared with placebo-controlled groups.\textsuperscript{28,29} In the Vitamins in Preeclampsia trial, the investigators examined the risk of preeclampsia in primiparous women with a BMI greater than 30 at enrollment. In the subgroup, there was no benefit of antioxidants in decreasing the risk of preeclampsia: risk ratio 0.87 (95% CI 0.59–1.30). The Vitamins in Preeclampsia trial also reported an increase in low birth weight babies in the supplemented compared with the placebo-control groups.\textsuperscript{29} Despite these data, after review by the data safety monitoring committee, the Maternal–Fetal Medicine Units network has decided to continue ongoing recruitment. To date, the results of clinical trials of the use of antioxidants to prevent preeclampsia are not definitive as to their efficacy, and the Maternal–Fetal Medicine Units network study is currently in progress.

Gestational diabetes mellitus is the clinical manifestation of glucose intolerance in pregnancy. The pathophysiology of GDM involves both decreased insulin sensitivity and inadequate insulin response, resulting in hyperglycemia. In general, obese women are more insulin resistant (or have decreased insulin sensitivity) compared with nonobese women, whether pregravid or during pregnancy, when there are already significant 50–60% decreases in maternal insulin sensitivity by the end of the third trimester. Decreased insulin sensitivity is the limited ability of insulin to transport glucose from the intravascular into the peripheral tissues, primarily skeletal muscle. Therefore, it is not surprising that obese women are also at significantly greater risk for the development of GDM, because they have decreased insulin sensi-
tivity compared with nonobese women (Figs. 1A and B). Weiss et al\textsuperscript{25} in the FASTER trial, after adjusting for potential covariables, reported that the adjusted odds ratio for the risk of GDM was 2.6 (95\% CI 2.1–3.4, \( P < .001 \)) for obese and 4.0 (95\% CI 3.1–5.2, \( P < .01 \)) for morbidly obese women. Therefore, in the management of obese pregnant women, consideration should be given to early glucose screening rather than waiting until the 24–28 week standard screening period. This would be helpful in women with other risk factors for GDM, such as a previous history of GDM, a family history of type 2 diabetes (particularly maternal), or a history of a macrosomic fetus.

Increasing non–insulin-mediated glucose use is of theoretical benefit in the prevention of GDM. Therefore, exercise with increased use of large skeletal muscles, such as walking or swimming, may be beneficial. Although the use of insulin sensitizers such as metformin and thiazolidinediones may be theoretically useful to increase insulin sensitivity, these agents cross the placenta and their fetal safety has not been documented. High fiber and complex carbohydrate/low glycemic diets may decrease the need for a large insulin response to a meal and theoretically decrease \( \beta \) cell failure, but the data on efficacy are again controversial. Glyburide, which has recently been introduced into the armamentarium of the treatment of GDM, helps restore euglycemia by enhancing insulin response. Therefore, because obese women may have decreased insulin sensitivity relative to nonobese women even in early gestation, in addition to early testing for GDM, lifestyle measures such as moderate physical activity and nutritional counseling may be beneficial in the obese women with normal glucose tolerance.

Because obese women have an increased risk of developing or having preexisting manifestations of the metabolic syndrome in, for example, hypertension, proteinuria, dyslipidemia, and diabetes, we are also experiencing an increase in medical problems previously assumed to be diagnosed primarily in an older nonpregnant population, for example, sleep apnea, nonalcoholic fatty liver disease, and chronic renal and cardiac dysfunction. As such, consideration should be given to obtaining data, for example, an electrocardiogram to evaluate cardiac function and further cardiac evaluation, depending on clinical history and physical and laboratory evaluation of obese women with chronic hypertension. Because these women are also at increased risk for the development of preeclampsia, obtaining an early evaluation of renal function and degree of proteinuria in early gestation may assist in distinguishing the chronic renal dysfunction secondary to maternal chronic hypertension and/or diabetes from pregnancy-associated hypertension, preeclampsia. Similarly, elevated liver function tests may be an

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\textbf{Fig. 1.} \textbf{A.} The longitudinal changes in insulin sensitivity in women whose pregravid body mass index (BMI) was less than 25 (n=6) and in those with BMI of 25 or more (n=9) as estimated using the hyperinsulinemic-euglycemic clamp. There is a significant decrease in insulin sensitivity over time (\( P < .001 \)) and between groups (\( P = .007 \)). FFM, fat-free mass. \textbf{B.} The women with a BMI of 25 or more before conception were separated into BMI overweight (BMI 25–30, n=6) and obese (BMI 30 or more, n=3). There is a significant difference among groups (\( P < .001 \)) and a group/time interaction (\( P = .002 \)). Reprinted from Catalano PM, Ehrenberg HM. The short- and long-term implications of maternal obesity on the mother and her offspring. BJOG 2006;113:1126–33. Copyright © 2006, with permission from Blackwell Publishing Ltd.\textsuperscript{30}

indication of nonalcoholic fatty liver disease rather than a manifestation of severe preeclampsia. To date, there are no published studies evaluating the prevalence of nonalcoholic fatty liver disease in pregnancy (PubMed research of the literature, English language, 1990–2006, key words: “non-alcoholic steatohepatitis,” “NASH,” “pregnancy”). We have seen an increasing number of obese women in pregnancy after extensive workup in the past 5 years. This is not surprising, given that obesity is recognized as the most common factor associated with nonalcoholic fatty liver disease in the nonpregnant population. Other common risk factors, such as increased estrogen concentrations, elevated lipids, and increased insulin resistance (all present in obese pregnant women), have been recognized as contributing factors. Therefore, as we have observed in our own population, we anticipate that the diagnosis of nonalcoholic fatty liver disease will become a more common diagnosis in obese pregnant women with abnormal liver function studies. Unfortunately, there are no simple laboratory tests to screen for maternal sleep apnea. However, a history of daytime somnolence and a partner’s complaint of loud snoring should increase suspicion of sleep apnea. Consideration of referral of these patients to the appropriate pulmonary specialist to make the diagnosis of obstructive sleep apnea and initiate treatment, such as nighttime continuous positive airway pressure, may be helpful because these women will have an increased risk of cesarean delivery.

PRETERM DELIVERY

In reviewing the literature relating preterm birth to maternal pregravid BMI or weight gain during gestation, most investigators have reported both low pregravid weight and poor weight gain in low BMI women as risk factors for preterm birth. Schieve et al. reported that the greatest risk of a preterm delivery was in women with a low pregravid BMI and weight gain less than 0.10 kg/wk. In a follow-up study, the same investigators reported that, compared with women of average BMI and average pregnancy weight gain, the risk of preterm delivery increased progressively with decreasing pregravid BMI and weight gain of less than 0.5 lb/wk; BMI greater than 26 (OR 1.6, 95% CI 0.7–3.5), BMI 19.8–26 (OR 3.6, 95% CI 1.6–8.0), and BMI less than 19.8 (OR, 6.7, 95% CI 1.1–40.6). Because as many as 25% of preterm births are indicated because of maternal medical or obstetric problems rather than the spontaneous nature of these events, many of the preterm births in the obese women may relate to indicated preterm delivery because of underlying medical and or obstetric issues. Weiss et al. reported that morbidly obese women had a significantly increased risk (OR 1.5, 95% CI 1.1–2.1) of preterm delivery in comparison with a normal weight control group. Relative to management, because the IOM guidelines for weight gain in pregnant women were initially aimed at decreasing the risk of preterm and growth-restricted neonates, the issue of weight gain among pregnant women relative to these issues is being reconsidered.

INTRAUTERINE FETAL DEATH

There has been an increasing awareness in the past decade of the role of maternal obesity in the risk of unexplained antepartum fetal death. Huang et al. in a Canadian population, examined the factors related to 196 unexplained fetal deaths, 25% of the fetal deaths in their population. The factor most strongly associated with unexplained fetal death was increased prepregnancy weight. Maternal prepregnant weight greater than 68 kg increased the risk of unexplained fetal death (OR 2.77, 95% CI 1.85–4.68), even after adjusting for maternal age and excluding maternal diabetes and hypertensive disorders. Factitious postdate pregnancies were eliminated because of ultrasound dating. Not unexpectedly, there was an increase in unexplained fetal deaths when the birth weight ratios were greater than average, ie, more than 1.15 (OR 2.36, 95% CI 1.26–4.44). These results were confirmed in a recent Danish National Birth Cohort study among 54,000 births from 1998 to 2001.

Compared with normal weight women, the fetal death rate among obese women increased with increasing gestational age: from 28 to 36 weeks the hazard ratio (HR) was 2.1 (95% CI 1.0–4.4), 37–39 weeks HR was 3.5 (95% CI 1.9–6.4), and at 40 weeks or more HR was 4.6 (95% CI 1.6–13.4). A similar trend was observed in overweight women. In contrast to the Canadian study, the birth weights of the unexplained fetal deaths among obese women were lower than the median birth weights of the live births, suggesting intrauterine growth restriction. Consistent with this finding was the fact that obesity was associated with a five-fold increase in the rate of stillbirth with histological placental dysfunction.

At this time, we can only speculate as to the pathophysiology of unexplained intrauterine fetal death in obese women. However, because these women are at increased risk for gestational hypertensive disorders and glucose intolerance, increased vigilance for these problems, known to be associated with increased perinatal mortality, is warranted. Given that maternal obesity may be associated with
an increased risk of stillbirth, how should we manage these pregnancies? Women with obesity-related problems such as hypertension and diabetes need to be monitored closely, as would nonobese women, including assessment of fetal well-being and growth, which is all the more difficult, however, as noted previously in obese women. In women without medical or obstetric complications, the increase in stillbirths in overweight women was twice that of normal weight women, whereas the increase in stillbirth in obese women was 240% greater than that in normal weight women.\(^3\) Certainly, close fetal monitoring with assessments such as fetal kick counts in these women is prudent, but the cost/potential benefit of more extensive evaluation is again speculative at this time.

**OBSTETRIC RISKS ASSOCIATED WITH MATERNAL OBESITY: PERIPARTUM**

In addition to the increased risk of antenatal obstetrical problems in obese women, there is an increased risk of cesarean delivery and associated morbidities. Regional and general anesthesia both are concerns in this population. There can be difficulty with placement of epidural or spinal anesthesia in obese women, requiring multiple attempts. Additionally, general anesthesia carries the risk of difficult intubation, and the increased incidence of sleep apnea post partum. Therefore obtaining an anesthesiology consult before the onset of labor should be encouraged. In the multicenter study of Weiss et al,\(^25\) the cesarean delivery rate for nulliparous women was 20.7% for women with a BMI of 29.9 or less, 33.8% for women with a BMI of 30–34.9 and 47.7% for women with a BMI of 35–39.9. Similar data were reported by Durnwald et al,\(^26\) in women attempting vaginal birth after 1 prior cesarean delivery (VBAC), of 510 women attempting a trial of labor, 337 (66%) were successful and 173 (34%) required repeat cesarean delivery. The greatest success rate for VBAC was in underweight (BMI < 19.8) women (84.7%) as compared with normal weight (\(P = .04\)). Decreased VBAC success was observed in obese women (54.6%), but not in overweight women (65.5%) compared with normal weight women (70.5%), \(P = .003\) and \(P = .36\), respectively. Additionally, normal weight women who gained weight between pregnancies to become overweight during their attempted VBAC had decreased success rates compared to those women whose BMI remained average, (56.6% versus 74.2%, \(P = .006\)). Unfortunately, the converse was not true, in that weight loss resulting in a status change from overweight to average did not significantly improve their VBAC success (64.0% versus 58.4%, \(P = .67\)). The increased cesarean rate in overweight and obese women is also associated with an increase in post operative complications such as wound infection/breakdown, excessive blood loss deep venous thrombophlebitis and postpartum endometritis. Therefore, if an obese patient requires cesarean delivery she should receive preoperative antibiotics even if the surgery is elective.

In obese women, there are no prospective trials to determine the optimal type of skin incision (ie, vertical or horizontal) to decrease the risk of wound disruption or infection. The vertical incision may afford greater exposure and room to deliver a macrosomic fetus and avoid an incision in a thick pannus, but this incision may result in increased postoperative pain and risk of evisceration because of lateral tension. In contrast, the Pfannenstiel incision may offer more postoperative comfort. But, if there is a wound breakdown, management may be difficult because of exposure. Therefore, the type of incision at the time of cesarean in obese patients will be made at the time of surgery based on maternal anthropometry and the experience of the individual surgeon. Because obese women are at increased risk for wound breakdown attempts to obviate these complications have included closure of the subcutaneous layers and/or placement of subcutaneous drains. As noted in the Committee Opinion No. 315, suture closure of the subcutaneous layer after cesarean delivery may lead to a significant decrease in wound disruption.\(^3\) However, the efficacy of subcutaneous drains to prevent morbidity of wound breakdown is less clear. Last, obese women are also at increased risk for postoperative deep venous thrombosis (DVT). The use of early ambulation and compression stockings may be of benefit if used properly. The use of postoperative heparin therapy is of value in the obese high risk patient, for e.g. with a previous history of DVT. The data on the use of postoperative heparin in all obese women to prevent DVT are insufficient to make any general recommendations regarding risk benefit. In summary, overweight and obese women are at risk of increased medical and obstetrical problems in pregnancy, which then in turn increase their risk of preterm delivery, cesarean delivery, and attendant operative morbidities.

**BARIATRIC SURGERY FOR OBESE WOMEN: GESTATIONAL CONSIDERATIONS**

As discussed previously, the best way to decrease the risk of medical and obstetric problems in obese women planning pregnancy is weight loss before conception. Given that lifestyle measures and medical treatments have had limited long-term success to date, more obese women of reproductive age are seeking...
Examining the impacts of bariatric surgery as an alternative. It is estimated that there are 150,000 bariatric surgical procedures performed in the United States each year. Bariatric surgery may be considered in patients with class III obesity, i.e., BMI greater than 40 or BMI greater than 35 with comorbid conditions, if nonsurgical modalities have failed. Bariatric surgical procedures can be categorized into two primary types: 1) malabsorptive procedures such as Roux-en-Y gastric bypass, and 2) restrictive procedures such as laparoscopic adjustable gastric banding. The previously performed malabsorptive procedures were associated with complications during pregnancy, such as small bowel ischemia, as well as nutrient deficiencies, such as iron, folic acid, and vitamin B12 deficiencies. There were also reports of fetal abnormalities, small for gestational age infants, and premature births. In a report by Sheiner et al, 298 patients became pregnant after bariatric surgery including both malabsorptive or restrictive procedures. Compared with the general population, there was an increase in premature rupture of membranes (OR 1.4, 95% CI 1.3–2.7, \(P=0.001\)), labor induction (OR 2.1, 95% CI 1.6–2.7, \(P=0.001\)), birth weight more than 4 kg (OR 2.1, 95% CI 1.4–3.0, \(P=0.001\)), and cesarean delivery 25.2% versus 12.2% (OR 2.4, 95% CI 1.9–3.1, \(P<0.001\)). The increased risk of cesarean delivery in women with previous bariatric surgery remained significant after adjusting for possible confounders. Of note, there were no significant differences between groups regarding other morbidities such as placental abruption, placenta previa, labor dystocia, or perinatal complications.

Although restrictive procedures including laparoscopic adjustable gastric banding are becoming more common, they are not without potential morbidity, including gastric ulcer perforation, intragastric band migration and balloon defect, and gastrointestinal hemorrhage resulting from erosion of a synthetic graft from a vertical banded gastroplasty. However, the early reported results from laparoscopic adjustable gastric banding studies are encouraging. Dixon et al and Skull et al in two separate Australian studies reported that maternal weight gain during pregnancy was significantly reduced in women who underwent laparoscopic adjustable gastric banding in comparison with control groups, without significant differences in birth weights. The incidence of gestational diabetes and pregnancy hypertensive disorders were also significantly reduced in women after laparoscopic adjustable gastric banding (Table 1). In the report of Dixon et al, there were no decreased folic acid or vitamin B12 levels in the women with laparoscopic adjustable gastric banding, but some women who were not taking multivitamins regularly had elevated homocysteine concentrations.

Based on the available data, which does not include any randomized prospective trials or long-term follow-up of offspring of obese women who underwent bariatric surgery before pregnancy (PubMed search, English language, 1990–2006, key words: “bariatric surgery,” “pregnancy”), the following recommendations of ACOG Committee Opinion No. 315 are endorsed and expanded: 1) Patients with laparoscopic adjustable gastric banding should be advised that they are at risk of becoming pregnant unexpectedly after weight loss following surgery and

### Table 1. Changes in Maternal Weight Gain, Birth Weight, and Obesity-Related Complications in Obese Women With Laparoscopic Adjustable Gastric Banding and Historical Controls

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<td>Weight gain (kg)</td>
<td>3.7 (0.6–6.9)</td>
<td>15.6 (12.4–18.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Neonatal birth weight (kg)</td>
<td>3.31 (3.14–3.49)</td>
<td>3.53 (3.35–3.72)</td>
<td>.19</td>
</tr>
<tr>
<td>Diabetes mellitus (%)</td>
<td>8.2</td>
<td>25.8</td>
<td>.048</td>
</tr>
<tr>
<td>Hypertension (%)</td>
<td>8</td>
<td>22.5</td>
<td>.06</td>
</tr>
</tbody>
</table>

LAGP: laparoscopic adjustable gastric banding; NS, not significant.

Data for the Dixon et al study (LAGP: n=79; Control: n=79) are expressed as mean±standard deviation, and data for Skull et al study (LAGP: n=49; Control: n=31) are expressed as mean (95% confidence interval), except where otherwise indicated.


should use an appropriate contraceptive method. 2) All patients undergoing laparoscopic adjustable gastric banding should delay pregnancy for 12–18 months to avoid the rapid weight loss phase of the procedure until their weight stabilizes and they are no longer catabolic. This will allow for a greater weight loss before conception and possibly decrease the risk of pregnancy-related complications such as hypertension and GDM. 3) Women with laparoscopic adjustable gastric banding should be monitored by their obstetrician and bariatric surgeon during pregnancy. Adjustments of gastric bands may reduce nausea and vomiting in pregnancy, but prophylactic removal or elimination of all the gastric band fluid may decrease the effectiveness of the treatment and result in excessive weight gain. 4) All women should have adequate supplementation of folic acid, calcium, and B12 after any bariatric surgical procedure because this may decrease the risk of subclinical nutritional deficiencies. Additional nutritional supplementation and close monitoring of fetal growth is necessary in women who have undergone diversionary or malabsorptive procedures.

In women with pregestational glucose intolerance, hypertension, central obesity, and lipid disorders, the physiologic changes in pregnancy increase the risk of problems previously not routinely encountered during pregnancy.

FETAL GROWTH

Maternal obesity is a well-recognized risk factor for fetal macrosomia, more specifically, obesity and long-term risks of adolescent components of the metabolic syndrome. Weiss et al. reported that the incidence of fetal macrosomia, defined as birth weight greater than 4,000 g, was 8.3% in the nonobese group, 13.3% in the obese women, and 14.6% in the infants of the morbidly obese women. However, just as there has been an increase in obesity in the adult and adolescent population in the past decade, neonatal birth weights have increased significantly. Recent studies from both North America and Europe have reported an increase in mean birth weights, particularly those infants either greater than the 90th percentile in weight for gestational age (large for gestational age, LGA) or macrosomic (birth weight more than 4 kg). In Denmark, the percentage of macrosomic newborns increased from 16.7% in 1990 to 20.0% in 1999. Factors such as decreased maternal smoking, an increased incidence of diabetes, and increasing maternal BMI have all been implicated.

In our own population, we have observed a mean increase of 116 g in term singleton birth weight over the last 30 years (Fig. 2). There was a mean 116-g increase in birth weight at term (37–41 weeks) from 1975 through 2003 (3,204±477 versus 3,320±818 g). The increase in birth weight was significant at the 5th, 10th, 50th, 90th, and 95th percentiles (range 85–173), with no significant difference among the percentiles (Fig. 3). From 1987 through 2003, the percentage of women who weighed less than 150 lb at delivery decreased from 33% to 16%, and the percentage of women who weighed more than 200 lb increased from 17% to 34%, with no change in the group between 150 and 200 lb (50%). Mean maternal age increased from 23.8 to 25.3 years. The percentage of white women decreased from 49.4% to 39.1%, while the percentage of Hispanic (6.8–16.2%) and Asian (0.6–2.3%) women increased. The percentage of women with diabetes increased from 4.0 to 5.7%, while the percent of women who smoked decreased from 34.4% to 18.4%. There was no significant change in parity or gestational age at delivery. Using a stepwise regression analysis, 8% of the variance in the increase in birth weight was related to the increase in maternal weight, while African-American race and female gender of the neonate accounted for an additional negative 4% of the variance in birth weight. None of the remaining demographic variables that were considered were found to be significant contributors to the change in birth weight.

Maternal anthropometric variables are important factors relating to fetal growth. Maternal pregravid weight has a very strong correlation with birth weight. Although maternal height is also associated

---

**Fig. 2.** Term singleton birth weight in grams from 37 to 41 weeks from 1975 to 2003 at MetroHealth Medical Center, P<.001.

with an increase in birth weight, when adjusted for weight, there was no longer a significant correlation between maternal height and birth weight. Maternal weight gain during gestation is positively correlated with birth weight. The correlation is stronger in nulliparous women ($r=0.26$) compared with parous women ($r=0.16$). The interaction of maternal pregravid weight and weight gain was examined by Abrams and Laros. There was a progressively stronger correlation between maternal weight gain and birth weight in moderately overweight, ideal body weight, and underweight women. In women weighing 135% of ideal weight for height before conception, there was no correlation between weight gain during pregnancy and birth weight. Lastly, maternal age and parity have independently been reported to have a positive correlation with birth weight. However, McKeown and Gibson reported that when maternal age was adjusted for parity, there was no longer a significant correlation between maternal age and birth weight. Parity has been shown by Thompson et al to be associated with a mean 100–150 g increase in birth weight in subsequent pregnancies. However, the additional effect of parity on birth weight is diminished with increasing parity.

Relative to maternal factors, paternal anthropometric factors have limited impact on fetal growth. Morton reported that, in half-siblings with the mother as the common parent, the correlation between birth weight and the half siblings was $r=0.58$. In contrast, the correlation of birth weight in half-siblings with the father as the common parent was only $r=0.10$. Animal crossbreeding studies support these findings. Walton and Hammond crossbred Shetland ponies with Shire horses. The size of the foals was roughly the same as the foals of the maternal pure breed. Thus, maternal regulation was more important in determining intrauterine growth than were paternal factors. Klebanoff et al using a Danish population registry, reported that paternal birth weight, adult height, and adult weight together explained approximately 3% of the variance in birth weight, compared with 9% for the corresponding maternal factors. In summary, maternal factors, most importantly maternal pregravid weight, have the strongest correlations with birth weight.

In our studies of fetal overgrowth and macrosomia, we have elected to concentrate on measures of body composition, ie, fat and fat free or lean body mass. The rationale for this approach stems from work done in the previous century. As early as 1923, research by Moulton described that the variability in weight within mammalian species was explained by the amount of adipose tissue, whereas the amount of lean body mass was relatively constant and changed in a consistent manner over time. In the human fetus, Sparks used autopsy data and chemical analysis in 169 stillborns, and described a relatively comparable rate of accretion of lean body mass in small for gestational age (SGA), average for gestational age (AGA), and LGA fetuses, but considerable variation in the accretion of fetal fat. Fat accretion in the SGA fetus was considerably less than in the AGA fetus, which in turn was less than that of the LGA fetus. Last, the term human fetus at birth has the greatest percentage of body fat (approximately 12%) compared with other mammals. For these reasons we have elected to assess fetal growth in our studies using estimates of body composition. The methodologies we have employed include anthropometric, stable isotope, and total body electrical conductivity. These methods have been previously described.

![Fig. 3. Mean term singleton (37–41 weeks) birth weight in grams for the 5th, 10th, 50th, 90th, and 95th percentiles from 1975 to 2003 at Metro-Health Medical Center.](image-url)
The utility of using body composition in understanding fetal growth is exemplified by a previous study evaluating the proportion of the variance in birth weight explained by body composition analysis of the fetus, and particularly fat and fat-free mass. The mean birth weight of the population was 3,553±462 g and the mean percentage of body fat was 13.7±4.2%. Fat-free mass, which accounted for approximately 86% of mean birth weight, accounted for 83% of the variance in birth weight. In contrast, body fat, which accounted for only approximately 14% of birth weight, explained 46% of the variance in birth weight.

In an effort to better understand the potential independent effect of maternal obesity on fetal growth in infants of women with normal glucose tolerance, we performed a stepwise logistic regression analysis of body composition on 220 infants of women with normal glucose tolerance (Table 2) previously published. Although maternal weight gain and height had the strongest correlations with birth weight and lean body mass, respectively, maternal prepregnancy weight had the strongest correlation with estimates of neonatal fat and percentage of body fat. Similarly, Sewell et al., using the same data set, reported that the increase in birth weights between infants of women with a BMI of 25 or greater, compared with those having a BMI of less than 25, was explained by an increase in fat mass rather than lean body mass (Table 3). This was the case despite significantly less weight gain (13.8±7.5 versus 15.2±5.3 lb, P<.001) in the overweight/obese compared with lean/average weight women.

| Table 2. Stepwise Regression Analysis of Factors Related to Body Composition of Neonates (n=220) of Women With Normal Glucose Tolerance |
|-----|-----|-----|
| Birth weight | r² | Δr² | P |
| Maternal pregravid weight | 0.029 | — | .1 |
| Maternal weight gain | 0.07 | 0.041 | <.001 |
| Maternal age | 0.098 | 0.028 | <.001 |
| Lean body mass | — | — | — |
| Maternal height | 0.025 | — | .02 |
| Fat mass | — | — | — |
| Maternal weight gain | 0.044 | — | .002 |
| Pregravid weight | 0.10 | 0.056 | <.001 |
| Percentage of body fat | — | — | — |
| Maternal weight gain | 0.031 | — | .012 |
| Maternal pregravid weight | 0.073 | 0.042 | <.001 |

What is the relative contribution of maternal obesity and GDM to the risk of fetal overgrowth in the population? Ehrenberg et al reported that the risk of having a LGA neonate was greatest for women with a history of diabetes (OR 4.4) when compared with maternal obesity (OR 1.6). However, there was four-fold greater number of LGA infants born of obese women than women with diabetes because the relative prevalence of overweight and obesity to diabetes was 47% and 5%, respectively. Therefore, at least in our population, maternal obesity and not diabetes appears to be the more important factor contributing to the population’s increase in mean birth weight.

Relative to obstetric management, again a decrease in maternal pregravid weight appears to be the most important factor relating to fetal overgrowth, defined as an increase in adipose tissue rather than lean body mass. However, limiting weight gain in obese women may prove beneficial in the decreased accretion of fat mass in the infant of the overweight and obese women. In the study of Sewell et al., weight gain in overweight and obese women (BMI of 25 or greater) had the strongest correlation with percentage of body fat (r²=0.13, P=.002), whereas weight gain was not significantly related to fat mass in the lean and average weight women. However, optimal weight gain in average weight, let alone obese, women has yet to be defined. Obviously, total caloric intake is importantly related to fetal growth, but so is the composition of the diet, ie, the percentage and types of fat as well as carbohydrate and protein. The other end of the energy equation, of course, is energy expenditure, ie, physical activity. A sedentary lifestyle only increases the tendency for weight gain. There are not enough evidence-based data, and it is beyond the scope of this review to speculate about the optimal diet in pregnancy for obese women. However, the problems related to maternal pregravid obesity and weight gain in pregnancy are not improving and will only worsen unless appropriate research studies and trials begin to address these issues because the consequences, as will be discussed, have far-reaching implications.

**LONG-TERM RISKS FOR THE FETUS OF THE OBESE MOTHER**

Although much has been written about the increased risk of the metabolic syndrome (obesity, hypertension, insulin resistance, and dyslipidemia) in infants born small for gestational age (SGA), recent evidence points toward an increase in adolescent and adult obesity in infants born either large for gestational age or macrosomic. There is abundant evidence linking higher birth weights to increased obesity in adolescents as well as adults for at least 25 years. Large cohort studies such
as the Nurses Health Study\(^71\) and the Health Professional Follow-up Study\(^72\) report a J-shaped curve, ie, a slightly greater BMI among subjects born small but a much greater prevalence of overweight and obesity in those born large.\(^73\) The increased prevalence of adolescent obesity is related to an increased risk of the metabolic syndrome. The increased incidence of obesity accounts for much of the 33% increase in type 2 diabetes, particularly among the young. Fifty to ninety percent of adolescents with type 2 diabetes have a BMI greater than 27,\(^74\) and 25% of obese children 4–10 years of age have impaired glucose tolerance.\(^75\) Hence, the epidemic of obesity and subsequent risk of diabetes and components of the metabolic syndrome may begin in utero with fetal overgrowth and adiposity rather than undergrowth.

A recent retrospective cohort study by Whitaker\(^76\) in over 8,400 children in the United States in the early 1990s reported that children who were born to obese mothers (based on BMI in the first trimester) were twice as likely to be obese by 2 years of age. If a woman had a BMI of 30 or more in the first trimester, the prevalence of childhood obesity (BMI greater than the 95th percentile based on CDC criteria) at ages 2, 3, and 4 years was 15.1%, 20.6%, and 24.1%, respectively. This was between 2.4 and 2.7 times the prevalence of obesity observed in children of mothers whose BMI was in the normal range (18.5–24.9). This effect was only slightly modified by birth weight.

There is an independent effect of maternal pregravid weight and diabetes not only on birth weight but also on the adolescent risk of obesity. Langer and colleagues\(^77\) reported that, in obese women with GDM whose glucose was well controlled on diet alone, the odds of fetal macrosomia (birth weight greater than 4,000 g) was significantly increased (OR 2.12) compared with women with a well controlled (diet only) GDM with normal BMI. Similar results, were reported in women with GDM who were poorly controlled on diet or insulin. In well-controlled insulin-requiring GDM, there was no significant increased risk of macrosomia with increasing pregravid BMI. Additionally, Dabelea et al\(^78\) also reported that the mean adolescent BMI was 2.6 kg/m\(^2\) greater in sibling offspring of diabetic pregnancies compared with the index siblings born when the mother had previously had normal glucose tolerance. Hence, both maternal pregravid obesity and the presence of maternal diabetes may independently affect the risk of adolescent obesity in the offspring.

This risk of the developing the metabolic syndrome in adolescents was recently addressed by Boney et al\(^79\) in a longitudinal cohort study of AGA and LGA infants of women with normal glucose tolerant and GDM. The metabolic syndrome was defined as the presence of two or more of the following components: obesity, hypertension, glucose intolerance, and dyslipidemia. Maternal obesity was defined as a pregravid BMI greater than 27.3. Children who were LGA at birth had an increased hazard ratio for metabolic syndrome (2.19, 95% CI 1.25–3.82, \(P<.01\)) by 11 years of age, as did children of obese women (1.81, 95% CI 1.03–3.19, \(P=.04\)). The presence of maternal GDM was not independently significant, but the risk of the development of metabolic syndrome was significantly different between LGA and AGA offspring of women with GDM by age 11 (relative risk 3.6).

**CONCLUSION**

The primary objective in the management of obesity during pregnancy is prevention. Having obese women lose weight with lifestyle changes and achieve a normal BMI before conception would be the ideal goal, but realistically it is quite difficult to achieve. Once an obese woman does conceive, management should be directed at increased surveillance for these risks: 1) in early gestation, the risks of spontaneous

<table>
<thead>
<tr>
<th>Pregravid Body Mass Index</th>
<th>Less Than 25 (n=144)</th>
<th>25 or More (n=76)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birth weight (g)</td>
<td>3,284±534</td>
<td>3,436±567</td>
<td>.051</td>
</tr>
<tr>
<td>Body composition (TOBEC)</td>
<td>2,951±406</td>
<td>3,023±410</td>
<td>.22</td>
</tr>
<tr>
<td>Lean body mass (g)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fat mass (g)</td>
<td>331±179</td>
<td>406±221</td>
<td>.008</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>9.6±4.3</td>
<td>11±4.7</td>
<td>.006</td>
</tr>
</tbody>
</table>

TOBEC, total body electrical conductivity.

Data are expressed as mean±standard deviation.

abortion and congenital anomalies, 2) in later gestation, gestational hypertension and diabetes-related problems, as well as the increased risk of unexplained stillbirths, and 3) at parturition, the increased risk of cesarean delivery and attendant complications of anesthesia, wound disruption, infection, and deep vein thrombosis. Limiting weight gain in pregnancy to IOM guidelines (currently under review) and tight glucose control in women with GDM may improve maternal and neonatal outcomes. Therefore, prevention rather than treatment may offer the best hope of breaking the vicious cycle of obesity during pregnancy. Until we attain a better understanding of the underlying genetic predispositions, physiology, and mechanisms relating to maternal and feto-placental interactions and how these, in turn, relate to fetal growth and development, all treatments must, by necessity, be empiric.

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