

# The effects of overweight and obesity on pregnancy-related morbidity

Amanda M. Wang<sup>1, \*</sup>, Alan J. Lee<sup>1</sup>, Shannon M. Clark<sup>1</sup>

 $^1$  Department of Obstetrics and Gynecology, University of Texas Medical Branch, Galveston, TX 77555, USA

\*Correspondence: Amanda.mei.wang@gmail.com (Amanda M. Wang)

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Objective: Overweight and obesity in pregnancy poses many possible complications for both mother and fetus. This article reviews the possible morbidity risks, including but not limited to pregnancy loss, maternal diabetes, hypertensive disorders, and post-partum complications, as well as possible neonatal and fetal complications. This article also provides recommendations regarding suggested antenatal weight gain and management strategies, and additional antenatal and postpartum management considerations. Mechanism: This is a review article summarizing the pertinent most recent data on overweight and obesity in pregnancy. Findings in brief: Obesity has effects on pregnancy loss, maternal diabetes, hypertensive disorders, venous thromboembolism, postpartum hemorrhage, and maternal infectious diseases. It also has an impact on preterm birth, management of prenatal ultrasound and induction of labor. Conclusions: Maternal overweight and obesity has many implications on pregnancyrelated maternal morbidity.

# Keywords

Obesity; Overweight; Pregnancy; Morbidity; Maternal gestational weight gain; Large for gestational age

# **1. Introduction**

Obesity is a known health risk and is frequently categorized using the World Health Organization's (WHO) classification based on body mass index (BMI) [1]. In the United States (U.S.), obesity (BMI > 30 kg/m<sup>2</sup>) is very common with a nationwide prevalence in adults of 42.4% [2]. In reports from the National Center for Health Statistics in 2015-2016 and 2018, approximately 67.4% of patients in the U.S. over the age of 20 were overweight (BMI 25-29.9 kg/m<sup>2</sup>) and 40.7% were obese; among U.S. female adolescents ages 12–19, the prevalence of obesity was 20.9% [3, 4]. With the obesity epidemic in the U.S. clearly affecting patients of reproductive age, this presents particular challenges in the pregnant population. Birth data from 2018 revealed that 54.7% of patients enter pregnancy overweight or obese [5]. Obesity is a major health concern in pregnant patients, particularly due to the obstetric challenges and complications seen in the antenatal, peripartum, and postpartum periods. In this article we will review the effects of overweight and obesity on pregnancyrelated morbidity and mortality as it concerns both mother and fetus. For the purposes of this article, overweight will be defined by the WHO classification of BMI of 25–29.9 kg/m<sup>2</sup>, and a BMI of  $\geq$ 30 kg/m<sup>2</sup> is obese.

# 2. Antenatal considerations

# 2.1 Maternal weight gain

Excessive maternal gestational weight gain (GWG) can further compound the complications of obesity in pregnancy. For patients with pre-existing obesity, excessive maternal weight gain can increase the risk of hypertensive disorders of pregnancy, gestational diabetes mellitus, Cesarean delivery, postpartum weight retention and worsening of obesity, as well as fetal macrosomia [6, 7]. In 2009 the Institute of Medicine (IOM) produced recommendations for weight gain in pregnancy which the American College of Obstetricians and Gynecologists (ACOG) adopted (Table 1) [8]. These guidelines do not recommend attempting weight loss during pregnancy or dieting. Ideally a normal body weight would be achieved pre-conception to minimalize adverse outcomes in pregnancy. Thus, these recommendations serve to mitigate and minimize the risks of obesity in pregnancy. Despite these recommendations, patients who are overweight or obese at the beginning of pregnancy are still likely to gain more weight than is recommended. Furthermore, there are common misunderstandings regarding recommended GWG among patients who are overweight and obese [9]. As a result, counseling and educating patients regarding the recommended GWG as pertains to the weight in which they enter pregnancy, as well as the implications of starting pregnancy at a higher BMI, is crucial.

Further complicating inappropriate GWG in gravidas with overweight or obesity is the fact that many patients may be undereducated and under counseled about the risks of obesity in pregnancy, as well as the added risks with additional excessive weight gain. Lack of such counseling is particularly prevalent in socioeconomically disadvantaged patients [10]. Assessing maternal weight, counseling gravidas with overweight or obesity about recommended GWG, and informing the patient regarding the effects of excessive GWG should be repeated throughout the pregnancy, as patients may not be able to recall information readily after a single session. Finally, consideration for referral to a nutritionist for additional assessment and counseling, especially early in pregnancy, can provide further education and intervention by assessing maternal diet and providing information on healthy eating practices.

## 2.2 Pregnancy loss

Several large retrospective studies have demonstrated that stillbirth, defined as fetal demise after 20 weeks' gestation, is more common in patinets with obesity even after adjusting for confounding factors [11, 12]. The biological cause of this association is not well understood, but several mechanisms have been proposed. One study suggests that malperfusion and impaired stromal decidualizationand infarcts in the placentas of stillborn fetuses is more common in pregnancies resulting in stillbirth [13]. Other studies have suggested that hyperlipidemia reduces prostacyclin production leading to vasoconstriction and platelet aggregration in the maternal circulation, which may also affect the placenta during pregnancy [14]. It is also possible that the inflammatory state caused by pro-inflammatory agents such as leptin, tumor necrosis factor-alpha, and interleukin 6 secreted by adipose tissue contributes to this increase in rates of stillbirth in patients with obesity [15]. Obesity also increases the risk for gestational diabetes and hypertensive disorders (as discussed below), which are known risk factors for stillbirth in pregnancy.

Obesity is also associated with spontaneous abortion (SAB), defined as fetal demise before 20 weeks' gestation, with one meta-analysis estimating an increase in relative risk of 1.21 per 5-unit increase in maternal BMI [16]. The association between obesity and SAB continues with evidence that obesity is associated with recurrent pregancy loss [17-19]. One theory to explain the association is related to adverse endometrial or ovarian functions secondary to obesity, however the precise mechanisms remain unknown [20]. There are currently no recommendations for additional antenatal surveillance or optimal delivery timing solely for obesity to reduce these risks. However, given the evidence of obesity and pregnancy loss, preconception counseling in apatient with overweight or obesity of reproductive age that involves a discussion of the benefits of prepregnancy weight loss should be considered at any health care visit.

# 2.3 Maternal diabetes

Obesity in pregnancy is associated with significant metabolic sequela. Many studies have demonstrated an association between maternal overweight or obese status with gestational diabetes mellitus (GDM), with Hispanics and Asians at highest risk [21–24]. A meta-analysis of 11 observational cohort studies calculated an adjusted odds ratio of 2.28 (95% CI 1.97–2.63) of developing GDM with each 3-unit BMI increase in during pregnancy [25]. Thus, ACOG recommends early GDM screening at initiation of prenatal care in patients who are overweight or obese and have one or more risk factors, including, but not limited to, hyperten-

Table 1. WHO weight classification and ACOG gestational weight gain recommendations.

Classification	BMI (kg/m <sup>2</sup> )	Recommended range of total gestational weight gain (lbs)
Underweight	<18.5	28-40
Normal	18.5-24.99	25-35
Overweight	25-29.99	15-25
Obese	$\geq$ 30	11-20
Class I	30-34.99	
Class II	35-39.99	
Class III	$\geq$ 40	

\*based on Institute of Medicine recommendations.

sion, A1c  $\geq$ 5.7, polycystic ovarian syndrome, and a history of cardiovascular disease and/or gestational diabetes [26]. This screening, if negative early in pregnancy, should still be repeated at 24–28 weeks, with consideration for screening earlier in that window, especially in the presence of excessive GWG.

GDM, especially in the setting of obesity, can increase the risk of fetal macrosomia (as defined under 'Fetal Growth Abnormalities'). Additionally, the pattern of adipose tissue distribution in GDM in macrosomic infants predisposes these infants to shoulder dystocia. Thus, it essential that these patients are appropriately screened for GDM. Pre-existing diabetes mellitus (DM), especially type 1 DM, increases the risk of fetal loss and congenital birth defects, likely due to the cytotoxic effects of hyperglycemia during early pregnancy [27]. This diabetic embryopathy mostly affects the cardiovascular and nervous systems of the fetus. In contrast to GDM however, if a patient has vascular damage secondary to pre-existing DM, there is an increased risk for a small for gestational age infant, defined as birth weight of less than 10th percentile [28].

Finally, it is possible that obesity itself can compound the risk of diabetes during pregnancy even if well-controlled. A study comparing diet-controlled GDM in normal weight versus patients with overweight or obesity demonstrated an increased risk for adverse pregnancy outcomes in the overweight or obese patients, despite adequate glucose control in both groups [29]. However, these outcomes are not evident when glucose control is achieved using insulin treatment [30]. In patients with either GDM or pre-existing DM, consideration for increased antenatal fetal surveillance via fundal heights, serial growth ultrasounds and fetal monitoring is recommended. If impending fetal macrosomia is a concern, a growth ultrasound can be obtained between 34 weeks 0 days to 38 weeks 6 days. Antenatal monitoring is recommended for patients with pre-existing DM and medication- or poorly controlled GDM. This is commonly initiated at 32 weeks and consists of some combination of nonstress test, biophysical profile, or modified biophysical profile [26, 31].

## 2.4 Maternal hypertensive disorders

Hypertensive disorders in pregnancy include gestational hypertension, pre-eclampsia, eclampsia, and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome. Patients who are overweight and obese pre-pregnancy have an increased risk of developing hypertensive disorders in pregnancy, especially those with a 5-year history of obesity [21, 22, 32, 33]. A review of 13 cohort studies estimated a doubling of risk of preeclampsia with each 5-7-unit increase in pre-pregnancy BMI [34]. In obese patients, GWG above the IOM recommendations was associated with increased risk of gestational hypertension and pre-eclampsia, with the incidence of hypertensive disorders increasing to up to 30% in class IV obesity, compared to a baseline incidence of 6-8% in the U.S. [6]. Similar to DM, the added risk of hypertensive disorders of pregnancy in the obese patient poses additional challenges for management, as well as increased risk of both maternal, fetal and neonatal morbidity and mortality.

The pathophysiology linking obesity and preeclampsia is not well understood. Inflammation is seen in both obesity and preeclampsia, with increases in inflammatory mediators such as C-reactive protein, interleukin 6, and tumor necrosis factor alpha [35]. Increased oxidative stress has been implicated in preeclampsia, and obesity is associated with increased free fatty acids and reactive oxygen species. It has also been suggested that patients with obesity have impaired cytotrophoblast-mediated remodeling of spiral arteries, which is seen in preeclampsia. Leptin, which has been shown to mediate hypertension, is seen in higher concentrations in patients with obesity and pre-eclampsia [36].

Pre-existing chronic hypertension (HTN), as defined as HTN preceeding pregnancy or diagnosed before 20 weeks' gestation, poses additional challenges. Obesity itself is predictably associated with increased rates of chronic HTN in pregnant patients [37]. In one study, 7% of patients with class III obesity entering pregnancy had chronic HTN, compared to 0.5% of normal weight patients [38]. While chronic HTN alone is a risk factor for pre-eclampsia, there is further risk of potential vascular damage during pregnancy in patients with chronic HTN, especially if poorly controlled when entering pregnancy. Patients with chronic HTN have an increased risk for worsening high blood pressures, pulmonary edema, hypertensive encephalopathy, and acute renal failure in pregnancy [39]. Compromise of the vasculature in the uteroplacental unit can cause intrauterine fetal growth restriction, oligohydramnios, placental abruption, and even perinatal death [40].

ACOG recommends that in patients with pre-existing hypertension, antenatal surveillance is recommended in the event of complications such as need for medications, intrauterine fetal growth restriciton, or superimposed preeclampsia [34]. While there is no consensus on when to initiate such surveillance or what this would consist of, however, many institutions will initiate such testing at 32 weeks. Antenatal testing for hypertensive disorders of pregnancy varies widely depending on the severity of the disorder [41].

The United States Preventive Services Taskforce (USP-STF) has recommended low dose aspirin be initiated between 12–28 weeks' gestation, but ideally before 16 weeks, to reduce the risk of development of pre-eclampsia in patients with one or more high risk factors or more than one moderate risk factors [42]. High risk factors include chronic HTN, history of preeclampsia, or Type 1 or Type 2 DM, and moderate risk factors includes maternal BMI of >30. While doses of aspirin have varied between studies, ACOG recommends 81 mg daily due to its availability in the United States [43].

#### 2.5 Maternal venous thromboembolism

Obesity independently increases the risk of venous thromboembolism [44]. The mechanisms include chronic inflammation, impaired fibrinolysis, endothelial dysfunction, plaque rupture, and platelet activation [45]. Pregnancy itself adds additional risk with increased venous stasis and decreased venous return due to an enlarging uterus, hypercoagulability, and decreased mobility, as well as a general increase in procoagulants. Additionally, a Danish study found that the odds of a pulmonary embolism are higher than venous thrombus embolism (VTE) in pregnancy, adding further morbidity to the risk of thromboembolism in pregnancy [46]. While this risk is present throughout pregnancy, it is highest during the first week postpartum [47].

Currently, there is no consensus on prophylactic anticoagulation to decrease the risk for VTE inpregnant patients with obesity based on obesity alone. The post-Cesarean period is especially associated with increased risk of VTE, and the American Association of Chest Physicians considers obesity as a minor risk factor when estimating the risk of post-Cesarean thromboembolism [48]. ACOG recommends the use of mechanical prophylaxis prior to, during, and following cesearean section, with individualization of antepartum and postpartum pharmacological management based on risk factors and VTE history [49]. These recommendations are similarly expressed in the Society of Obstetrician and Gynaecologists of Canada's guidelines [50]. However, other organizations, such as the British Royal College of Obstetricians and Gynaecologists suggests that for patients with preconception obesity and additional risk factors, which include pre-eclampsia, Cesarean section, and hospitalization for 3 or more days, antenatal pharmocologic thromboprophylaxis is indicated as early as possible [50]. Recently, The Society for Maternal-Fetal Medicine (SMFM) released recommendations stating that patients with class III obesity (BMI >40) should receive intermediate enoxaparin dosing after Cesarean section [51]. In the absence of a clear consensus the routine use of compression device prophylaxis and an individualized approach to pharmacologic prophylaxis for obese patients is reasonable.

# 2.6 Preterm labor and delivery

The rate of preterm delivery is higher in patients with obesity. However, this is thought to be primarily due to medically indicated deliveries secondary to maternal co-morbid conditions [52, 53]. Studies attempting to determine the rate of spontaneous preterm birth linked solely to obesity have been mixed [54–56]. Increased rates of preterm premature rupture of membranes (PPROM), defined as rupture of membranes before the onset of labor before 37 weeks gestation, may contribute to this trend as PPROM has been shown to be more frequent in obese patients although adverse neonatal outcomes have not been well defined [57]. There is no current evidence to recommend enhanced or additional screening for preterm labor or PPROM in obese patients.

## 2.7 Fetal aneuploidy screening

ACOG recommends that all patients regardless of age or risk factors be offered genetic screening for fetal aneuploidy [58]. However, the pregnant patient with obesity can present a unique challenge for this screening. Screening can start as early 9-10 weeks using cell free DNA (cfDNA), a noninvasive genetic screen for fetal aneuploidy that can be done from 9-10 weeks of gestation until term gestation. cfDNA analyzes fetal DNA fragments in maternal serum to screen for aneuploidy and therefore requires a sufficient fraction of fetal DNA in order to successfully complete the assay and deliver an assessment of risk. With increasing maternal weight, the fetal cell-free DNA fraction decreases leading to higher rates of inconclusive test results [58-61]. The fraction of cfDNA increases with gestation and in obese patients after 21 weeks' gestation there is no longer a statistically significant difference in inconclusive tests between obese and normal weight patients [62]. A patient with obesity must be counseled regarding the risk of inconclusive cfDNA and possible need for repeated testing or testing later in gestation in order to obtain a valid screen and reportable results, which can ultimately take weeks.

The first trimester screen is typically completed between 10 weeks 0 days and 13 weeks 6 days gestation and includes ultrasound assessment of the nuchal translucency (NT) measurement +/- the maternal serum analytes pregnancy associated plasma protein A (PAPP-A), beta human chorionic gonadotropin (hCG), and/or alpha-fetoprotein (AFP). While obesity seems to increase the amount of time needed to obtain the NT measurement via ultrasonography, studies conflict regarding the effect of obesity on failure to complete the NT measurement [63–65]. In some instances, the patient may be asked to come back at a later date to reattempt the NT measurement.

As mentioned above, maternal serum analyte analysis to assess the risk for fetal aneuploidy is utilized in first trimester screening with an NT measurement and in various forms of first and second trimester screening (quadruple, integrated, sequential, and contingent), as well as in the second trimester quadruple screen. These analytes are obtained from maternal serum and will utilize some combination of measurements of

free beta hCG, AFP, estrogen, inhibin, and PAPP-A to calculate the risk of fetal aneuploidy. The calculated risk is modified by race, maternal age, diabetic status, and weight prior to determining a final risk. The analyte levels are inversely related to maternal weight. Although maternal weight is typically adjusted for by the laboratory, the capability for correction in many laboratories reaches a maximum at a maternal weight of 250 to 270 pounds [60, 61]. Diabetes, which is more common in obesity, also decreases the level of serum analytes and can be adjusted for as well. If, however, patients with obesity do not receive the recommended early diabetes screening at the beginning of pregnancy, diabetes (gestational or undiagnosed pre-existing) may not be diagnosed until the typical 24-28-week gestational diabetes screen, potentially confounding the analyte results [60]. Practitioners should be aware of how obesity can affect the fetal aneuploidy screening tests that utilize maternal serum analytes and be prepared to counsel patients with additional screening or diagnostic tests.

The potential limitations that obesity places on fetal aneuploidy screening modalities can affect not only the accuracy of the screen, but the receipt of timely results. For example, in the case of cfDNA, repeat testing may be required in the case of an inconclusive result due to low fetal fraction which can add weeks to get a result. In the case of maternal serum analytes, suboptimal evaluation and interpretation of the levels of the analytes can lead to suboptimal results. This can result in missed indications to perform detailed ultrasound anatomy scans, fetal echocardiograms, and diagnostic tests, such as amniocentesis, that can inform and direct care. Delays in or missed opportunities for additional evaluation may limit choices regarding pregnancy continuation and delay multidisciplinary planning for the neonate if needed. If there are other factors present that place the pregnancy at increased risk for aneuploidy, and/or receiving a timely screening result is necessary for the patient to make important timely decisions regarding her pregnancy, appropriate counseling on the limitations of screening in these patients is essential.

# 2.8 Fetal ultrasound

Obesity is not reported to strongly affect the performance of first trimester ultrasound. The crown rump length measurement is unchanged with obesity, and transabdominal and transvaginal ultrasound perform similarly. First trimester evaluation of the fetal heart does not appear to be affected by obesity aside from the possibility of needing a transvaginal approach [66–68]. Two European studies noted that patients with obesity are at increased odds of having due dates reassigned to later dates, however, it is unclear if this is an effect of imprecise dating due to irregular menstruation that can be associated with obesity or obesity itself [69, 70].

Second trimester ultrasound has many concerns relating to the obstetric patient with obesity. Even though congenital abnormalities are more frequent in this population, several studies have demonstrated that fetal abnormalities are less likely to be detected in patients with obesity via ultrasound. These abnormalities include neural tube defects, cardiovascular anomalies, cleft lip and palate anomalies, anorectal atresia, hydrocephaly, limb reduction, and trisomy 21, with the notable exception of gastroschisis [56, 71, 72]. Currently there are no recommendations for detailed anatomy ultrasound studies, serial fetal growth assessments, or cardiac evaluation for the indication of obesity alone, though many patients with obesity will undergo these evaluations for maternal medical conditions associated with obesity such as hypertension or diabetes.

The technical aspect of ultrasound is oftentimes more challenging due to maternal body habitus, requiring more time for image acquisition, increased numbers of ultrasounds needed to adequately obtain all necessary images, and resulting in more suboptimal views or incomplete ultrasound studies [65, 73–76].

The sonographic fetal heart evaluation presents a particular challenge in patients with obesity, with decreased cardiac anomaly detection and increased suboptimal cardiac views [77]. The American Institute of Ultrasound Medicine does not currently recommend fetal echocardiography solely for obesity unless a fetal malformation is suspected [78]. Strategies to improve the acquisition of these views, such as advanced ultrasonography equipment, have not improved fetal cardiac evaluation [65, 79]. As a result, the potential for missing major and minor fetal anomalies due to maternal obesity and body habitus remains a diagnostic challenge in this patient population. Finally, failure to detect fetal anomalies on ultrasound can lead to missed opportunities for counseling on additional screening and/or diagnostic testing for fetal aneuploidy.

Currently ACOG recommends that all patients receive an ultrasound to evaluate fetal anatomy between 18-22 weeks' gestation, and a consensus statement recommends performance of a detailed anatomical survey without fetal echocardiography for obese patients with a BMI  $\geq$  35 [80, 81]. To improve ultrasound evaluation for patients with obesity, attempts have been made to determine optimal timing for ultrasound assessment of fetal anatomy. Some studies have found that in patients with obesity delaying the anatomy scan until 20-22 weeks' gestation increases the rate of completion of the study in a single scan, while another study suggested combining an early 15-week ultrasound with the standard 18-22-week ultrasound may improve scan results [82-84]. The improved performance that delaying the anatomy scan may afford must be balanced with the benefits of early detection of fetal anomalies and appropriate counseling and management of the patient and pregnancy. Although there is no recommended optimal timing for performing a fetal anatomy assessment in patients with obesity, a detailed fetal anatomy ultrasound is recommended in patients with a BMI  $\geq$  35.

As with optimal ultrasound timing, there are no recommendations for universal first trimester cardiac or anatomy evaluations. Image quality may be improved by utilizing lower frequencies to achieve better sonographic tissue penetration, considering a transvaginal approach, and repositioning the patient to optimize sonographic windows. Providers may inform patients with obesity of increased duration of exams as well as the possibility for the need for repeat imaging. Importantly, patients should be counseled that ultrasound in the setting of obesity is limited and cannot completely rule out fetal abnormalities, especially of the fetal heart.

# 2.9 Fetal growth abnormalities

There is strong evidence for increased risk for large for gestational age (LGA) and macrosomic fetuses in both patients with pre-pregnancy obesity and those with excessive gestational weight gain [56, 85-88]. ACOG defines LGA as birth weight  $\geq$ 90th percentile for gestational age, while macrosomia is less universally defined, and is generally accepted to be 4000 or 4500 grams regardless of gestational age [89]. A proposed mechanism is that fetal hyperglycemia (secondary to maternal hyperglycemia) leads to increased fetal production of insulin, which acts as a growth factor, insulinlike growth factor, and growth hormone, leading to fetal macrosomia [20, 89]. In addition, GDM and pre-existing DM, especially in the setting of obesity, can increase the risk of fetal macrosomia and is associated with fetal birth weights >5000 grams [89–91]. As such, it is vital to appropriately screen patients for GDM, manage and monitor for appropriate glucose control, and initiate recommended antenatal fetal surveillance.

Fetal macrosomia and LGA impose risks on both mother and fetus. Labor progress can be affected, which in turn can increase the need for a Cesarean section. The mother is at risk for postpartum hemorrhage (PPH), third- and fourthdegree lacerations, and infections. The fetus is at increased risk for shoulder dystocia, birth injuries including brachial plexus injuries, meconium aspiration syndrome, stillbirth, and neonatal mortality, particularly when birth weight is over 5000 grams [92]. There is conflicting evidence linking obesity alone to shoulder dystocia with one study demonstrating that the association of obesity and shoulder dystocia disappears after adjusting for factors such as diabetes [93– 95]. The risk of shoulder dystocia in patients with obesity is likely more attributable to comorbid conditions such as diabetes and macrosomia. Together, these findings support the ACOG statement that excessive maternal weight and GWG are not good predictors of shoulder dystocia [96].

Accurately estimating the weight of these infants is difficult both sonographically and clinically. There is currently no indication for serial ultrasound monitoring of fetal growth for maternal obesity alone [89]. Furthermore, even though there may be an elevated risk of shoulder dystocia in these patients, there is no benefit to delivery before 39 weeks' gestation in the absence of other obstetric or fetal indications. However, scheduled Cesarean for suspected macrosomia of  $\geq$ 5000 grams in a mother without diabetes or  $\geq$ 4500 grams in a mother with diabetes may be beneficial and should be discussed with the patient. Because of the complications that may occur due to macrosomia, fetal growth should be closely monitored using fundal height measurements in any fetus with suspected macrosomia or condition predisposing to macrosomia such as pre-existing diabetes or poorly controlled gestational diabetes, supplemented by ultrasound where fundal height is disparate from gestational age [89].

# 3. Peripartum considerations

# 3.1 Induction of labor

Patients with obesity are more likely to require induction of labor (IOL) for obstetric indications such as chronic HTN, hypertensive disorders of pregnancy, diabetes, preeclampsia, and post-term gestation (>42 weeks) [97]. With increasing parity, the odds of a vaginal delivery increase, however, patients with obesity spend more time in the first stage of labor regardless of parity [94, 98]. Per the ARRIVE trial, induction of labor at 39 weeks' gestation in healthy, nulliparous patients reduced the rate of Cesarean birth, preeclampsia, and gestational hypertension as compared to expectant management among the same population [99]. It is notable that 53.5% and 52% of the participants in the induction and expectant management groups respectively had a BMI  $\geq$  30 indicating that even in those with obesity, IOL at 39 weeks' gestation may be beneficial in avoiding Cesarean delivery [99]. Finally, patients with obesity are at higher risk for medically indicated delivery before 39 weeks' gestation for maternal indications such as hypertensive disorders, DM, and suspected fetal macrosomia. One study of over 3 million singleton deliveries showed a 22.9-27% rate of medically indicated delivery in patients with obesity [100].

#### 3.2 Fetal monitoring

Intrapartum assessment of the fetus can be challenging in patients with obesity. These patients tend to spend more time unmonitored during labor due to limitations with external fetal monitoring [101]. Internal monitoring of the fetal heart rate with the fetal scalp electrode and uterine contractions with the intrauterine pressure catheter (IUPC) can help overcome these difficulties in monitoring fetal status while in labor. However, there are times when these modalities are undesirable or impractical such as in a patient with HIV, HCV, or a closed cervix early in labor. The need for more reliable monitoring may also lead to an early amniotomy, thus predisposing the patient and fetus to prolonged rupture of membranes in labor. This, in addition to prolonged internal monitoring, may place the mother at an increased risk of intraamniotic infection, and amniotomy before engagement of fetal head in latent labor may increase the risk of umbilical cord prolapse [102].

The assessment of uterine contractions by the usual external tocodynamometry can be particularly difficult in patients with obesity. An alternative method for external contraction assessment in obese patients may be with the utilization of electrohysterography—a form of external monitoring that measures uterine electromyography signals to monitor uterine contractions [103]. However, electrohysterography requires specialized equipment that may not be widely available. A study comparing IUPC to external tocodynamom-

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etry and electrohysterography showed that electrohysterography was easily interpreted and was more sensitive in maternal obesity, however, accurate contraction strength could not be determined with electrohysterography [104]. Regardless of the methods used for fetal monitoring during labor, it is important that it be effected as accurately and consistently as possible, especially in the presence of additional maternal and/or fetal comorbidities.

## 3.3 Cesarean delivery

Cesarean delivery (CD) is well established to be more likely in patients with overweight or obesity and is usually due to fetal distress, failure to progress, and/or fetal malpresentation [22, 25, 33, 86]. While many Cesarean deliveries are medically indicated due to conditions such as severe hypertensive disorders, patients with obesity are also at higher risk for preterm labor and premature, prelabor rupture of membranes which may also necessitate Cesarean section [21]. Cesarean delivery in the obese patient is made more challenging because of difficulty in establishing regional or general anesthesia, more technically challenging surgical field, and greater incision to delivery time [105, 106]. Additionally, those patients with a BMI >50 who deliver by Cesarean carry a five-fold risk of ICU admission [107].

Practitioners should keep these surgical considerations in mind when caring for patients with obesity, being mindful of the potential limitations to how quickly an emergency Cesarean delivery may be performed due to maternal body habitus. In addition, early consultation with anesthesia colleagues to prepare for and address any concerns is ideal. Depending on the habitus of the patient, a vertical and possibly supraumbilical surgical approach may be necessary. If postoperative complications requiring ICU admission occur, a multi-disciplinary team involving obstetricians, anesthesia, and critical care providers is recommended. Establishing reliable early regional anesthesia may assist with ensuring adequate anesthesia should an emergent delivery be indicated.

Perioperative antibiotics should be given during a Cesarean delivery, and for patients weighing  $\geq 120$  kg increasing antibiotic dosage may be considered [56]. Closure of any subcutaneous tissue defects  $\geq 2$  cm and utilizing suture rather than staples for skin closure, specifically a synthetic monofilament, is associated with decreased wound morbidity and should be considered [108–111]. Utilization of prophylactic negative pressure wound dressing, as seen with a wound vacuum, in obese patients after Cesarean section has not been shown to improve surgical site infections in the majority of trials and its routine use is not recommended [112–114].

Trial of labor after Cesarean (TOLAC) is not contraindicated in patients with obesity though several studies have noted an inverse relationship between BMI and TOLAC success. This relationship has been shown to persist even after adjusting for birth weight however perceived cephalopelvic disproportion accounts for around half of failed trial of labors [115, 116]. Those who do attempt TOLAC tend to have more neonatal complications of low 5-minute APGAR scores, maternal ICU admission, and increased neonatal intensive care unit (NICU) admission, maternal infection, uterine rupture, and maternal blood transfusion. In the event of uterine rupture, neonates of patients with obesity are more likely to be admitted to the NICU, have low APGAR scores, require prolonged respiratory assistance, and have seizures, while maternal morbidity is similar to that of patients of normal weight [117, 118]. Practitioners should counsel patients regarding TOLAC risks, utilize success rate calculators, and be prepared for the surgical difficulties should emergent Cesarean delivery become indicated. As with TOLAC patients of normal weight, following the ACOG recommendations regarding TOLAC is recommended [119].

# 4. Postpartum considerations

#### 4.1 Postpartum hemorrhage

The link between postpartum hemorrhage (PPH) and obesity is not completely clear. While several studies note an association between PPH and obesity, especially in the setting of uterine atony, other studies find moderate to no association, and still others note that after adjusting for comorbidities, the association between obesity and PPH disappears [120–127]. Although the precise contribution of obesity to the risk of PPH may not be established, known risk factors for PPH such as macrosomia, prolonged labor, infection, and preeclampsia that are associated with obesity should prompt providers to be alert and prepared for the possibility of PPH.

# 4.2 Infection

Obesity is associated with increased infectious complications in pregnancy. Intraamniotic infection increases in obese patients as a function of the increased labor duration and prolonged internal monitoring that can be associated with obesity. Additionally, for the patient with obesity with a history of Cesarean delivery undergoing TOLAC, both successful TOLAC vaginal birth after Cesarean and failed TOLAC requiring repeat Cesarean are both associated with higher levels of infections than those who underwent elective repeat Cesarean delivery [116, 128]. After a Cesarean section patients with obesity have greater rates of wound disruption, further increasing the risk of wound infection [129-131]. When wound infection and disruption do occur, the wound should be examined, opened, drained, and surgically debrided if necessary, and severe infectious morbidity such as systemic infection or necrotizing fasciitis must be ruled out and appropriately treated. Antibiotics should be administered if systemic infection is suspected or cellulitis is noted. After appropriate care, wounds left to heal by secondary intention have been shown to heal more quickly through use of negative pressure wound therapy [132, 133].

#### 4.3 Thromboembolic events

As previously described above, obesity has been shown to increase the risk of VTE, with the postpartum period representing one of the highest risk periods [48, 51, 134]. Al-

though there is no clear consensus on which patients with obesity should get postpartum VTE prophylaxis and for how long, individual assessment of risk by the practitioner should be employed.

#### 5. Conclusions

Reproductive age patients have been affected by increasing obesity rates as has the population at large. For the obstetric patient, obesity brings unique challenges that can significantly affect the morbidity and mortality of both the mother and the fetus. These challenges affect all aspects of pregnancy care, including the antenatal, intrapartum, and postpartum periods, and require special considerations for this group of patients. When caring for obstetric patients with obesity, it is necessary that providers understand the unique risks that these patients face, provide appropriate counseling, and be cognizant of the complications and diagnostic challenges in order to optimize care and outcomes for both patient and baby. Importantly, all practitioners should work with any patient with obesity prior to pregnancy to optimize prepregnancy weight, not only for the benefit of the pregnancy, but also for global health outcomes.

# Abbreviations

BMI, body mass index; CD, cesarean delivery; cfDNA, cell free DNA; DM, diabetes mellitus; GDM, gestational diabetes mellitus; GWG, gestational weight gain; HELLP, hemolysis, elevated liver enzymes, low platelets; HTN, hypertension; IOL, induction of labor; IUPC, intrauterine pressure catheter; LGA, large for gestational age; NT, nuchal translucency; PPH, postpartum hemorrhage; PPROM, preterm premature rupture of membranes; SAB, spontaneous abortion; TOLAC, trial of labor after Cesarean; VTE, venous thromboembolism.

# Author contributions

AMW and AJL contributed to literature review as well as writing and edition of the paper. SMC contributed to paper review. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

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# **Conflict of interest**

The authors declare no conflict of interest.

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