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Review article

Obesity in pregnancy

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Overweight and obesity are common findings in women of reproductive age in the UK; as 32% of 35- to 64-year-old women are overweight and 21% obese. Obesity causes major changes in many features of maternal intermediary metabolism. Insulin resistance appears to be central to these changes and may also be involved in increased energy accumulation by the fetus. Maternal obesity is associated with many risks to the pregnancy, with increased risk of miscarriage (three-fold) and operative delivery (20.7 versus 33.8% in the obese and 47.4% in the morbidly obese group). Other risks to the mother include an increased risk of pre-eclampsia (3.9 versus 13.5% in the obese group) and thromboembolism (0.05 versus 0.12% in the obese group). There are risks to the fetus with increased perinatal mortality (1.4 per 1000 versus 5.7 per 1000 in the obese group) and macrosomia (>90th centile; 9 versus 17.5% in the obese group). Maternal obesity is associated with an increased risk of obesity in the long term. Obese woman should try to lose weight before pregnancy but probably not during pregnancy. There is no real evidence base for the management of maternal obesity but some practical suggestions are made.

Keywords Complications, management, obesity, pregnancy.

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Introduction and epidemiology of maternal obesity

Overweight and obesity are prevalent and increasing in the UK. Thirty two percent of 35- to 64-year-old women are overweight (body mass index [BMI] 25–30 kg/m^2) and 21% obese (BMI > 30 kg/m^2).[1] These proportions are increasing and the percentage of adults who are obese has roughly doubled since the mid-1980s.

Therefore, overweight and obesity are common in antenatal clinics in the UK. A retrospective analysis of data obtained from a validated maternity database system in the North West Thames Region was used to compare pregnancy outcomes based on maternal BMI measured at booking (Figure 1).[2] Forty-two percent of women were nulliparous and 72.3% were white women. Of the whole cohort, 27.5% of women were overweight and 10.9% were obese, defined at the booking BMI. Obese women were more likely to book late, but this did not account for their increased BMI. Over the last decade, there has been a two-fold increase in women being recognised as obese at booking visits.[3] The number of obese women at booking has increased to 18.9 from 9.4%. When adjusted for maternal age, parity, smoking status and deprivation status, the mean BMI was 1.37 kg/m^2 higher.

There are considerable risks in terms of maternal overweight and obesity in terms of maternal and fetal morbidity and mortality. In the most recent Confidential Enquires into Maternal and Child Health report (2000–2002), 35% of all women (n = 78) who died were obese compared with 23% of the general maternal population, a dramatic rise from the 16% reported in 1993.[4]

Energy expenditure

Body weight and weight gain are a result of energy intake against total energy expenditure. Energy intake is difficult to measure and there are few good measures in normal pregnancy. The increment in energy intake is between 0.3 and 0.5 MJ per day representing 84–140 MJ through pregnancy. This increased intake does not alone meet the increased energy needs of pregnancy and therefore the gain in energy must result from alterations in energy expenditure.

Total energy expenditure is considered to have three main contributors. Seventy-five percent of total energy expenditure comes from resting energy expenditure (REE), 10–15% may come from exercise and 10–15% from thermogenic activities. REE is determined by lean body mass;[5] the larger a person, the greater their REE. Although fat mass, age and sex may account

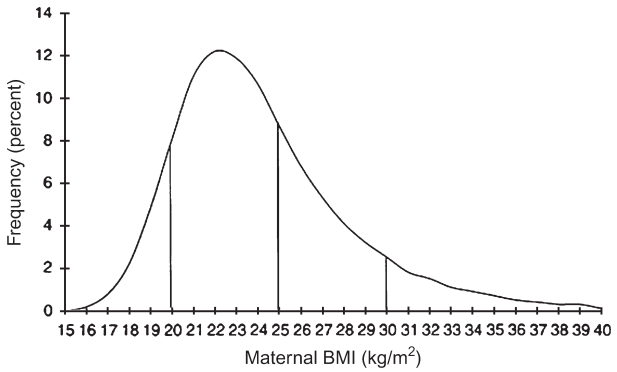

Figure 1. Maternal obesity and pregnancy outcome; a study of 287 213 pregnancies in London. The distribution of BMI is shown.

for variation in REE between individuals, fat mass has very little, if any, effect on REE.[6] Postprandial thermogenesis or diet-induced thermogenesis, the increment in energy expenditure after a meal, can be considered to have a facultative portion which is variable in some physiological and pathological states and an obligate portion which is determined by the stoichiochemistry with processes such as protein synthesis. Protein and carbohydrate have the greatest effect on this increment in energy expenditure after meals.

There is an accumulation of energy in the extra weight of the mother and fetus during pregnancy, which represents a combination of altered energy intake together with an alteration in the components of energy expenditure. This includes alterations in REE per unit lean body mass and postprandial thermogenesis per unit food consumed; furthermore, the alterations in REE are brought about by the increase in lean body mass with the extra weight of pregnancy. The best estimate of average total fat gained during pregnancy is 3.0 kg.[7] The total extra energy needs of pregnancy amount to 360 MJ or roughly 1.2 MJ per day.[8] Table 1 shows the theoretical energy cost of pregnancy. There are differences in REE and variation in pregnancy depending on the energy status as well as the adiposity. A serial study using 24-hour calorimetry concluded that there are highly characteristic changes in each subject with large intersubject differences.[9] Lean women tended to decrease their REE early in pregnancy whereas overweight and obese women showed an increased REE from the beginning of pregnancy. The energy maintenance cost of pregnancy was correlated with the degree of fat mass of women before they became pregnant ($r = 0.72$) and the weight gain during pregnancy ($r = 0.79$).[10]

Postprandial thermogenesis has been studied in pregnancy. Two studies suggest that there are no changes in the increase in energy expenditure after a meal,[11,12] while another two studies have demonstrated a decrease in postprandial thermogenesis during pregnancy.[13,14] The latter study also demonstrated that the reduction in postprandial thermogenesis correlated with the degree of insulin resistance during normal pregnancy.[14] This is in accord with a large study demonstrating postprandial thermogenesis to be associated with insulin resistance outside pregnancy.[5] There have been no specific studies of obesity and postprandial thermogenesis in pregnancy, but obese women are more insulin resistant.

Intermediary metabolism in pregnancy

Maternal insulin resistance is central to changes in intermediary metabolism during normal pregnancy. This insulin resistance may have a role in liberating metabolites for fetal growth. Fasting blood glucose levels decrease in normal pregnancy. Excursions of glucose above fasting especially after a glucose load appear increased in normal pregnancy.[15,16]

Fasting plasma insulin levels increase through pregnancy, but these changes do not occur at the same stage as the decrease in glucose concentrations. This would suggest that the glucose and insulin concentrations are not directly linked unless insulin sensitivity is altered or the pancreatic β cell

Table 1. Theoretical energy cost of pregnancy

	0–10 weeks	10–20 weeks	20–30 weeks	30–40 weeks	Cumulative total
	KJ/day (g/day)	KJ/day (g/day)	KJ/day (g/day)	KJ/day (g/day)	KJ/pregnancy (Kg/pregnancy)
Protein	15 (0.64)	43 (1.8)	112 (4.8)	144 (6.1)	21 800 (0.93)
Fat	234 (5.9)	1065 (26.8)	872 (22.0)	132 (3.3)	157 900 (3.96)
REE	188	416	622	954	150 000
Total net additional energy	437	1522	1606	1229	329 500
Total net energy + 10%	481	1677	1767	1352	362 400

Four 10-week time periods are shown, with the whole pregnancy values in the last column. The protein and fat accumulation are shown with the extra energy expenditure to maintain the extra weight; REE is dependent on lean body mass. The total net extra energy per day and per pregnancy is then shown. This then has an obligate postprandial thermogenesis shown in the last row.[8]

glucostat is set at an altered level.[17,18] Insulin response to a meal or glucose challenge is increased in normal pregnancy.[16,19] Insulin concentrations are increased and insulin sensitivity is reduced in obese compared with nonobese pregnant women, although these changes are not statistically significant.[20] Obese women are insulin resistant compared with nonobese women.[21] Noncomplicated pregnancy is characterised by insulin resistance when investigated with a euglycaemic clamp,[22] an intravenous glucose tolerance test with minimal modelling of insulin sensitivity[23] or the short insulin tolerance test.[14] When assessed with stable isotope techniques, hepatic glucose production is not altered in pregnancy.[24] Outside pregnancy, obesity is not associated with a change in hepatic glucose output unless fasting glucose is elevated such as in diabetes.[25] The length of fast has a major effect on nonesterified fatty acid concentrations (NEFA). The fasting NEFA concentrations are probably not altered in pregnancy, although the NEFA concentrations do rise more quickly with length of fast increasing from 12–18 hours compared with those in nonpregnant women.[26]

Triglyceride concentrations increase in pregnancy to two or three times the nonpregnant levels.[27] This is probably a result of increased adipose tissue lipolysis as a consequence of insulin resistance and enhanced NEFA delivery to the liver which is then associated with increased very low density lipoproteins concentrations.[28] Reduced lipoprotein lipase activity leads to a reduced capacity for triglyceride removal from the circulation.[27] Maternal hypertriglyceridaemia is associated with maternal insulin resistance.[14] Maternal BMI is statistically associated with triglyceride concentrations in the second trimester ($r = 0.58$, $P < 0.1$).

Cardiovascular system

Pregnancy is associated with wide-ranging cardiovascular changes through increased oxygen demand. Obesity-induced changes have profound effects on cardiac, endothelial and vascular function which is dependent on the duration of obesity.[29] Every 100 g of fat deposited increases the cardiac output by 30–50 ml/minute; this is also accompanied by an increase in blood volume. Volume load initially brings about left ventricular hypertrophy and then subsequently the myocardium starts to dilate against increased pressure overload. The pressure overload is secondary to increased sympathetic activity due to the potentiating effects of hormones such as leptin, insulin and various inflammatory mediators. The heart rate then increases with the elevated cardiac output, thereby decreasing the diastolic interval and thus the time for myocardial perfusion. Impaired myocardial diastolic relaxation leads to diastolic dysfunction. The conduction and contractility can be further compromised when fat deposition occurs in the myocardial tissue.[29,30]

Maternal complications

Hypertension

The association of hypertension and adiposity outside pregnancy has been described for some time.[31] The separation of pregnancy-associated hypertension and essential hypertension is neither clear nor are the diagnostic criteria uniform through all studies. These factors have led to a variation in the prevalence of hypertension in severely obese pregnant women between 5 and 66%. In a study of 4100 deliveries in California, the prevalence of pregnancy-induced hypertension was 4.2% in normal weight women and increasing to 9.1% in the obese women, the values being 1.2 and 5.3% for what the authors called hypertension.[32] The incidence of gestational hypertension increased from 4.8% in the normal weight group to 10.2% in the obese group ($n = 1473$) and 12.3% in the morbidly obese group ($n = 877$).[33] Women with a BMI greater than 30 kg/m^2 had a significantly increased risk of developing pre-eclampsia ($P = 0.042$), and excessive weight gain was associated with higher rates of pre-eclampsia in overweight women ($P = 0.016$; excessive weight gain in normal group, $n = 498$ [9.7%], $n = 415$ [24.2%] in the overweight group and $n = 604$ [35.4%] in the obese group).[34]

A systematic review of maternal BMI and risk of pre-eclampsia showed that the risk of pre-eclampsia typically doubled with each 5 to 7 kg/m^2 increase in pre-pregnancy BMI.[35] Some of the studies are summarised in Figure 2.

Thromboembolism

Generally, there is a 30% increase in venous stasis by 15 weeks and a 60% increase by 36 weeks of gestation. Vascular damage is also increased at the time of delivery. In the North West Thames study, the prevalence of thromboembolism was 0.04% in the normal weight women. This rose to 0.07% in the overweight and 0.08% in the obese women.[2]

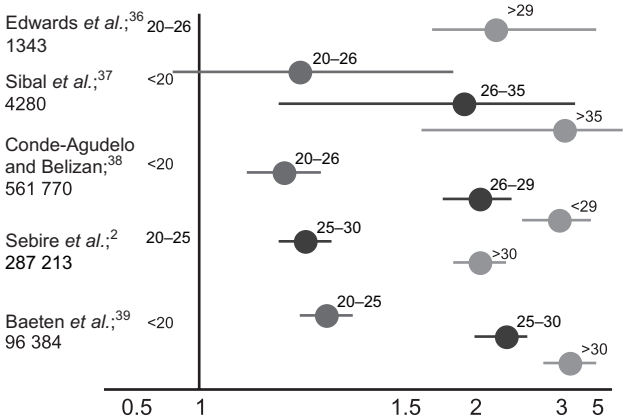

Figure 2. A summary of some of the maternal obesity and pre-eclampsia studies. The *x*-axis represents the odds ratio. The first author, date and number studied are shown in left column. The next column shows the reference population.

Gestational diabetes

Gestational diabetes mellitus (GDM) is glucose intolerance first recognised in pregnancy. Although not integral to the diagnosis, while glucose intolerance normally resolves following pregnancy, it does predict a high risk of type II diabetes mellitus (T2DM) in later life. Therefore, GDM represents an early presentation of T2DM. Within 15 years of pregnancy complicated by GDM, 30% of lean women and 70% of obese women develop T2DM.[40]

In a study of 16 102 women, the incidence of GDM was 2.3% in the control group and increased to 6.3% in the obese group (OR 2.6) and 9.5% in the morbidly obese group (OR 4.0).[33]

In a UK study, women with a BMI greater than 30 kg/m^2 are 3.6 times more likely to develop GDM compared with women with a normal BMI.[2] In a large Danish study consisting of 8092 women, the odds of developing GDM also increases with BMI (BMI < 25 kg/m^2, OR 1; BMI 25–29 kg/m^2, OR 3.4; BMI > 30 kg/m^2, OR 15.3).[41]

Weight change is also important in the development of GDM in obese women. In an observational study, women who lost 10 lb between pregnancies were found to have a decreased risk of GDM with a relative risk (RR) of 0.63 (95% CI 0.38–1.02), after adjusting for maternal age. However, a gain of 10 lb between pregnancies is associated with an increased risk of GDM (RR = 1.47; 95% CI 1.05–2.04).[42] Therefore, pre-pregnancy weight reduction and behavioural education is of paramount importance for these women.

The prevalence of T2DM in younger women is increasing,[43] especially in some ethnic groups.[44] Therefore, diabetes is associated with increasing overweight and obesity. Sixty percent of women have an unplanned pregnancy and may have undiagnosed diabetes. The pregnancy is at increased risk of fetal malformation in addition to fetal macrosomia.

Fetal complications

Congenital malformations

Ultrasonography in obese women is often suboptimal. In a study of 1622 ultrasound scans performed in the second and third trimester, for women with a BMI in the 97th centile, the visualisation decreased to 63% and an overall 14.5% decrease in visualisation of all organs systems in women with a BMI greater than the 90th centile compared with those with a normal BMI was observed.[45] The ideal gestation for a detailed anomaly scan is at 18–20 weeks in order to exclude congenital abnormalities. The role of serum biochemistry, particularly alfafetoprotein may be useful for increasing the detection rate of neural tube defects (NTD).

There is conflicting evidence regarding the association between obesity and congenital malformations. One case–control study found that women with a BMI greater than 31 kg/m^2 had a significantly increased risk of delivering infants with NTD and defects of the central nervous system, great vessels in the heart, ventral wall and other intestinal defects.[46] The association between spina bifida and obesity was also confirmed in a study which concluded that for every incremental unit increase (kg/m^2) in BMI, the risk of NTD increased by 7%. There is also an increase in other malformations such as omphalocele (three-fold), cardiac anomalies (especially septal defects two-fold) and multiple defects among infants of the overweight and obese group.[47] However, other studies have not found an association of an increased risk of congenital malformation and obesity.[48,49]

The mechanism for the observed association between obesity and birth defects is unknown but several possible explanations have been put forward. Increased serum insulin, triglycerides, uric acid and endogenous estrogens in addition to increased insulin resistance, chronic hypoxia and hypercapnia have been suggested as possible mechanisms. It is possible that the increased malformation found with maternal obesity is an extension of the fuel-mediated teratogenesis suggested in diabetes mellitus.[50] Obesity is often associated with pre-existing diabetes, a known risk factor for birth defects. The increased risk of NTD in infants of obese women was thought to be related to the lower levels of folic acid that reach the embryo due to poor absorption and higher metabolic demands. In a case–control surveillance program of birth defects, a daily intake of 400 microgram of folate was protective against NTD in infants of women with body weight less than 70 kg but not in infants of women weighing more than 70 kg.[51] The authors concluded that the risk of NTD increases with maternal weight independent of folic acid intake. Another possible explanation is that an increase in anomalies may be related to the failed detection due to the difficulty in sonographic assessment as previously discussed.

Macrosomia

Several studies have shown that maternal obesity and excessive weight gain during pregnancy are associated with macrosomic babies.[2,33,52] Obesity and pre-GDM are independently associated with an increased risk of large-for-gestational-age infants, and this impact of abnormal body habitus on birthweight increases with increasing BMI and is associated with significant obstetric morbidity.[53,54] The original Pedersen hypothesis suggested that increased glucose concentrations in the mother with diabetes led to increased fetal growth.[55] Obesity is associated with increased maternal insulin resistance and fetal hyperinsulinaemia even in the absence of maternal diabetes.[56] Insulin-resistant individuals have higher fasting plasma triglyceride levels and greater leucine turnover.[57,58] Amino acids are insulin secretagogues and an increased flux in amino acids could stimulate fetal hyperinsulinaemia. Triglycerides are energy rich, and placental lipases can cleave triglyceride and transfer free fatty acids to the fetus across a haemochorial placenta.[59] The combination of an

increased energy flux to the fetus and fetal hyperinsulinaemia may explain the increased frequency of large-for-gestational-age infants seen in obese women without diabetes.

Antepartum stillbirth

The combination of rapid fetal growth induced by the endogenous hyperinsulinaemia in obese women and the functional limitations of the placenta to transfer sufficient oxygen to meet the requirements of the fetus may lead to hypoxia and death.

Studies have suggested that obesity is associated with an increased risk of antepartum stillbirth. In a prospective population-based cohort study (n = 3,480), a three-fold increase in antepartum stillbirth was found in morbidly obese women compared with women with a normal BMI.[52] In a large Swedish population-based cohort study (n = 167 750), the risk of late fetal death increased consistently with increasing pre-pregnancy BMI.[60] Among nulliparous women, the risk of late fetal death was doubled among women with a normal BMI as compared with lean women, tripled among those who were overweight and quadrupled among those who were obese. Among the parous women, the risk of late fetal death was significantly increased only among obese women. For early neonatal death, the risk was doubled in nulliparous women with a higher BMI, but this was not true in parous women.

The Swedish Medical Birth Register was used to investigate the relationship between weight gain during pregnancy and antepartum stillbirth.[61] After controlling for multiple variables, overweight (BMI 25–29.9 kg/m^2) and obese (BMI ≥ 30 kg/m^2) women had a two-fold increase in the risk of term antepartum stillbirth. However, weight gain during pregnancy was not associated with an increased risk of antepartum stillbirth.

In a Danish study involving 24 505 singleton pregnancies, the overall rate of stillbirth was 4.6 per 1000 deliveries and of neonatal death was 3.1 per 1000 live births. Maternal obesity was associated with more than double the risk of stillbirth (OR 2.8) and neonatal death (OR 2.6) compared with women of normal weight. No single cause of death explained the higher risk of stillbirth in children of obese women. However, higher proportions of stillbirths caused by unexplained intrauterine death and fetoplacental dysfunction were found in children of obese women compared with children of non-obese women (BMI < 30 kg/m^2). There was no apparent cause of neonatal death.[62]

Long term for the fetus

Infants who are at the highest end of the distribution for weight or BMI or who grow rapidly during infancy are at increased risk of subsequent obesity.[63] Obese babies were nine times more likely than normal weight babies to grow into obese adults, and infants who grew rapidly were five times more likely to become obese.

Obstetric complications

Infertility/miscarriage

Obese women have a higher prevalence of amenorrhorea and infertility. Obesity is common, occurring in 35–40% of women with polycystic ovary syndrome.[64] Fifty percent of overweight women have polycystic ovaries or polycystic ovary syndrome compared with 30% of lean women.[65] The risk of miscarriage before the first liveborn child is 25–37% higher in obese women.[66] Obesity has a negative impact on infertility treatment and if conception occurs, there is an increased risk of pregnancy loss. Three cohort studies have suggested that obesity is an independent risk factor for spontaneous miscarriage in women who undergo fertility treatment.[67–69] With ovulation induction using gonadotrophin-releasing hormone, there is a three-fold increase in the risk of pregnancy resulting in miscarriage, and with egg donation in women with a BMI greater than 30 kg/m^2, there is a four-fold increased risk of miscarriage. Therefore, obese women should be encouraged to lose weight prior to their fertility treatment as this can result in significant improvement in reproductive outcome for all fertility treatment.[70]

Delivery and surgical complications

The inability to obtain interpretable external fetal heart rate and uterine contraction patterns in obese women is frequent. Women with a BMI of 35 kg/m^2 or greater are likely to have pre-existing medical conditions such as hypertension or diabetes, and this may further increase their anaesthetic risks. Specific resources such as additional blood products, a large operating table and extra personnel in the delivery room are essential prior to the delivery. Other intrapartum complications include failure of epidural insertion, increased risk of aspiration during anaesthesia, difficult intubation, poor peripheral access and difficulty in monitoring of maternal blood pressures. Increased retention of lipid-soluble agents, increased drug distribution and more rapid desaturation have also been reported.[71] The significant difficulty in administering epidural analgesia should not preclude their use in labour. Prophylactic placement of an epidural catheter when not contraindicated in labouring morbidly obese women would potentially decrease anaesthetic and perinatal complications associated with attempts at emergency provision of regional or general anaesthesia.[72]

Obese women had a higher rate of induction of labour (25.5%, BMI 20–30 kg/m^2; 36%, BMI > 30 kg/m^2; OR 1.6, 95% CI 1.3–1.9)[73] and a higher rate of failed induction (7.9 versus 10.3 versus 14.6% with increasing BMI)[34] and caesarean section rates in nulliparous women (20.7% in the control group versus 33.8% in obese group and 47.4% in morbidly obese group; P > 0.01).[33] There was also a higher rate of obstetric complications in women who were overweight at their first antenatal visit such as operative vaginal delivery

(8.4 versus 11.4 and 17.3% with increasing BMI; $P < 0.001$), shoulder dystocia (1 versus 1.8 and 1.9% with increasing BMI; $P < 0.021$) and third/fourth degree lacerations (26.3 versus 27.5 and 30.8% with increasing BMI; $P < 0.001$) when compared with the normal BMI group.[34] The frequency of both elective (8.5 versus 4%) and emergency caesarean section (13.4 versus 7.8%) were almost twice as high for the very obese women compared with the normal BMI group.[2] Maternal obesity was found to influence the route of delivery, independent of co-morbid conditions such as macrosomia, nulliparity, induction or diabetes, and obese and overweight women had a higher risk of caesarean section delivery compared with normal weight women (13.8 and 10.4 versus 7.7%, $P < 0.0001$).[74] In another study of 126 080 deliveries, after excluding women with diabetes and hypertensive disease, there was a three-fold increased risk in failure to progress in the first stage and higher caesarean section rate of 27.8 versus 10.8% (OR 3.2) in the obese group compared with the normal weight group.[75] The increase in emergency caesarean sections in these obese women may be related to an increased number of large-for-gestational-age infants, suboptimal uterine contractions and increased fat disposition in the soft tissues of the pelvis leading to dystocia during labour.

Common operative complications include the loss of landmarks, making vascular access difficult. A hospital-based perinatal database was used to identify women with a BMI of greater than 35 undergoing their first caesarean delivery. These authors reported an overall wound complication rate of 12.1%; those with a vertical skin incision were at greatest risk (34.6 versus 9.4%).[76] The proposed benefits of a transverse incision are reported to have a more secure closure, less fat dissection and less postoperative pain. Earlier ambulation and deep breathing can further decrease the risk of atelectasis and hypoxaemia. The disadvantage of a low transverse incision is that it potentially increases infection rates due to the warm and moist area underneath the pannus. Retraction of the large pannus, in order to gain good access, can compromise the maternal cardiopulmonary system.[71] A vertical skin incision allows a better visualisation of the operative field with less physical exertion on the operator, decreased operative time and decreased blood loss. However, it should be closed by a mass closure technique with either a permanent or delayed absorbable monofilament suture.

In a randomised study ($n = 76$), the use of a subcutaneous drain or suture closure was assessed as an effective means of decreasing wound complications. These authors concluded that a subcutaneous suture or drainage in women with at least 2 cm of subcutaneous fat at the time of caesarean delivery can reduce the incidence of postoperative wound complications.[77]

Postoperative respiratory complications such as pneumonitis are more common. Early mobilisation, aggressive chest physiotherapy and adequate pain control are essential components of effective postoperative care.[72,78]

In the puerperium, endometritis, postpartum haemorrhage, prolonged hospitalisation and wound infections appear more frequent in obese women (Table 2). The risk of postpartum haemorrhage rises with increasing BMI and is about 30% more frequent for women with moderately raised BMI and about 70% more frequent for women with highly raised BMI compared with the normal BMI group.[2]

Vaginal birth after caesarean section

A study of 510 women attempting a trial of labour investigated the impact of maternal obesity and weight gain on the success of vaginal birth after caesarean section.[79] After adjusting for confounding factors such as ethnicity, labour induction, gestational age at delivery and infant birthweight, the high pre-pregnancy BMI and weight gain between pregnancies reduce vaginal birth after a single low transverse caesarean delivery (54.6 versus 70.5%; $P = 0.04$). In a study of 1213 women, obese women were 50% less successful when attempting a trial of vaginal delivery after a caesarean section when compared with underweight women ($P = 0.043$).[80]

Breastfeeding

The fall in progesterone that occurs immediately postpartum is the trigger for the onset of copious milk secretion, and the maintenance of prolactin and cortisol concentrations is necessary for this trigger to be effective. Maternal obesity is

Table 2. Maternal complications according to each BMI category

	BMI (kg/m^2) group	Proportion (%)	OR (99% CI)
Chest infection	20–25	0.13	
	25–30	0.16	1.07 (0.81–1.41)
	>30	0.28	1.34 (0.99–1.92)
Genital infection	20–25	0.66	
	25–30	0.73	1.24 (1.09–1.41)
	>30	0.76	1.30 (1.07–1.56)
Wound infection	20–25	0.39	
	25–30	0.59	1.27 (1.09–1.48)
	>30	1.34	2.24 (1.91–2.64)
Urinary tract infection	20–25	0.69	
	25–30	0.84	1.17 (1.04–1.33)
	>30	1.10	1.39 (1.18–1.63)
Pyrexia of unknown origin	20–25	1.00	
	25–30	1.29	1.19 (1.08–1.32)
	>30	1.54	1.29 (1.13–1.48)
Prolonged postnatal stay	20–25	20.35	
	25–30	21.08	1.00 (0.97–1.04)
	>30	22.86	1.48 (0.82–2.69)

Box 1. Suggested management

Pre-pregnancy
Advise that weight loss will reduce risks of pregnancy and increase success with infertility treatment
Ensure folate supplementation for all overweight and obese women and consider higher dose of 5 mg a day if BMI > 40 kg/m^2
Booking
Weigh all mothers and calculate BMI
Advise on risks
Give dietary advice to all obese women (BMI > 30 kg/m^2) and see dietician if BMI > 40 kg/m^2
Suggest diet but not weight loss
Provision of specific information concerning maternal and fetal risks of obesity in pregnancy
Early ultrasound to confirm dating and detailed anomaly scan
Diabetes
Screen for GDM in all overweight and obese mothers. At St Mary's Hospital, we screen all women for GDM using 50 g of loading glucose at 27 weeks
Thromboembolism
Graduated compression stockings, hydration and early mobilisation be used after any operative delivery
Thromboprophylaxis and adequate dose of anticoagulant for an appropriate duration is recommended
Hypertension
Consider screening for pre-eclampsia by uterine artery Doppler if facilities permit
Regular antenatal visits with blood pressure checks
Anaesthetic review if BMI > 40 kg/m^2
Regional anaesthesia unless contraindicated
Anticipation of problems and effective preparation in terms of equipment, monitoring and personnel
General anaesthesia if required should be delivered with tracheal intubation and controlled ventilation
Postoperative care that includes close monitoring, early mobilisation and physiotherapy; a high-dependency setting may be appropriate
Judicious use of neuraxial, oral and intravenous opioids for postoperative pain
Surgery
Experienced operators
If BMI > 40 kg/m^2, assess the ability for hip abduction to allow vaginal delivery and allow McRoberts' manoeuvre for shoulder dystocia
If BMI > 40 kg/m^2, occupational health and home assessment after delivery
Infection
Beware of increased infection risks
Education
Advise on long-term risks of obesity, hypertension and diabetes
Suggest weight loss prior to next pregnancy

associated with a reduction in breastfeeding frequency.[2,81,82] Although it is likely to be multifactorial in origin, the simple mechanical difficulties of latching on and proper positioning of infant when the mother is obese can pose a problem for establishing breastfeeding. From an endocrine perspective, obesity is associated with a reduced prolactin response to suckling.[83]

Long-term consequences for the mother

Overall, in pregnant women, a weight gain of 10 kg is statistically associated with the best obstetric outcome. A weight gain in pregnancy of over 9 kg is more likely to be retained when not pregnant.[84] Gestational weight gain and postpartum behaviours associated with weight change from early pregnancy to 1-year postpartum have been investigated in 540 New York women.[85] One-year postpartum, the women were a mean 1.5 ± 5.9 kg heavier, while 25% experienced a weight gain of 4.6 kg or more. Weight gain in excess of guidelines was three times more likely in low-income groups. Gestational weight gain, a lack of postpartum exercise and food intake were all associated with weight gain to 1-year postpartum.

In a randomised controlled trial of 120 normal weight women, healthy eating and exercise were used to prevent excessive weight gain in pregnancy. In the intervention group, weight gain exceeded 15.9 kg in 33% women compared with 58% in the untreated group. The postpartum retention of weight was proportional to weight gain in pregnancy.[86]

Conclusions

Obesity is recognised as a frequent condition and a major risk factor in the western world. Twenty-eight percent of pregnant women are overweight and 10.9% are obese. Obesity in pregnancy is a major predictor of obesity later in life, which is

commonly associated with the development of chronic hypertension, dyslipidaemia and T2DM.[87] Therefore, from a public health perspective, obesity represents an important modifiable risk factor for adverse pregnancy outcome. There is no real evidence base for the management of maternal obesity but some practical suggestions are made. ■

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