Chapter 15
Fetal Macrosomia

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Fast Facts

- The ultrasound estimation of fetal weight is imprecise at best.
- Data is limited to suggest the frequency of ultrasound evaluation to detect macrosomia.

15.1 Definitions and Adverse Outcomes

Fetal macrosomia poses significant risks to mother and fetus in both diabetic and nondiabetic pregnancies. There are different definitions of macrosomia throughout the medical literature, including >90th, 95th, or 97th percentile for gestational age and birth weight >4000 g or >4500 g [1, 2]. In general, large for gestational age refers to weight above the 90th percentile for gestational age, while macrosomia refers to fetal weight above 4000 g [3].

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Several studies have evaluated macrosomia in terms of birth weight sufficient to be associated with adverse pregnancy and neonatal outcomes. For example, in a recent retrospective study of the US Linked Birth-Infant Death Cohort dataset from 1995 to 2004 encompassing 30,831,694 singleton term live births and 38,053 stillbirths, Ye et al. evaluated risk for stillbirth, neonatal death, and 5-min Apgar score >4 according to birth weight subgroups [1]. As the main outcome of the study, the authors created a composite perinatal mortality and morbidity index (PMMI), which included stillbirth, neonatal death, and a 5-min Apgar score less than four. They estimated ideal birth weights according to White, Black, and Hispanic ethnic groups [1]. The analysis was predicated on the assumption that perinatal mortality would form a J-shaped distribution with mortality decreasing up to an ideal birth weight and then increasing above it [1, 4, 5].

For this study, the authors define macrosomia as birth weights that exceeded the nadir of the mortality curve and categorized infants according the following birth weight percentiles: 75th, 90th, 95th, and 97th [1]. The authors found no significant increase in these adverse perinatal outcomes until birth weight reached >97th percentile for gestational age [1]. Based on their study of birth weight relative to their composite PMMI outcome, the authors found the lowest PMMI at birth weights between 3500 and 4000 g. Above this threshold, PMMI increased. Therefore, they suggested a birth weight >4500 g in Whites and >4300 g in Blacks and Hispanics as the optimal threshold for defining macrosomia sufficient to cause increased risk for adverse perinatal outcomes [1]. The authors found that cesarean section rates increased significantly with birth weight, with an overall cesarean delivery rate of 20%. Odds ratios for adverse outcomes were greater among the vaginally delivered subgroup, but this subanalysis did not change the authors postulated cutoff points [1].

Similarly, in a Scottish cohort study encompassing 784,576 births, over the period between 1992 and 2008, birth weight above the 97th percentile was significantly associated with antepartum stillbirth with odds ratio 1.8, 95% confidence interval [CI] 1.5,2.4 [5].

Macrosomia has also been found to increase risk for intrapartum and neonatal morbidities [6–8]. A study of the US National Health Statistics database between 1995 and 1997 evaluated birth and neonatal outcomes in the 4000–4499 g group, a 4500–4999 g group, and a >5000 g group as compared to normal weight controls. The investigators found that risk for cephalopelvic disproportion, cesarean section, and birth injury was increased in a dose-dependent manner among macrosomic infants. Increasing birth weights also increased risk for birth asphyxia, meconium aspiration syndrome, hyaline membrane disease, and low Apgar score. Risk of death was only elevated in the >5000 g group as compared with normal weight controls [7].

Similarly, a retrospective cohort study including 36,241 deliveries at the University of California, San Francisco, evaluated birth outcomes stratified by the presence or absence of diabetes and the presence or absence of macrosomia (i.e., birth weight above 4000 g) [8]. The study found that macrosomia was significantly associated with RDS, hypoglycemia, shoulder dystocia, and brachial plexus injury, even in the absence of diabetes [8]. In pregnancies complicated by gestational diabetes, the risk for RDS, hypoglycemia, shoulder dystocia, and brachial plexus injury was significantly increased in macrosomic diabetic pregnancies compared to GDM pregnancies with normally growth fetuses [8].

Complications of Macrosomia

Cesarean Delivery
Birth Injury (Brachial plexus, Erbs)
Maternal pelvic floor injury
Meconium Aspiration syndrome
Low APGAR
Neonatal Hypoglycemia

15.2 Standard Versus Custom Growth Curves

Formulae for estimating fetal weight from standard biometric measurements have been constructed in a variety of different populations. All have been found to be subject to considerable

imprecision [9]. Ultrasound estimation birth weights at the extremes of size have been found to be the least accurate [10]. A systematic review comparing methods of fetal weight estimation found no one method to be clearly superior to the others [10]. Similarly, a prospective observational study that evaluated methods of estimating fetal weight within 7 days of delivery found that sonographic methods of estimating fetal weight remained relatively inaccurate despite improvements in ultrasound equipment over the decade they studied (1991–2000). They found that the error in estimating fetal weights was attributable in the main to the formulae used and only to a lesser extent to inter-operator variation [9].

Some authors have suggested that the use of customized growth curves that adjust for maternal height, weight, and ethnicity may reduce misclassification errors in diagnoses of suspected macrosomia and suspected IUGR [11, 12]. A recent NICHD cohort study of longitudinal fetal growth among 2334 healthy low-risk women has documented significant differences in fetal growth according to maternal ethnicity, a finding with significant implications for classification of potentially macrosomic fetuses [13]. For example, the 95th percentile at 39 weeks was 4402 g for White women, 4226 g for Hispanic women, 4078 g for Asian women, and 4053 g for Black women [13]. However customized fetal growth curves have not yet been demonstrated to improve pregnancy or neonatal outcomes [14]. Fetal MRI is another modality that may hold promise for more accurate estimation of fetal weight at or near term [15].

15.3 Diagnosis of Macrosomia

15.3.1 Diabetic Pregnancies

There are no standard guidelines and little evidence base for frequency of ultrasound in diabetic pregnancies to screen for macrosomia. In women with pregestational diabetes, ACOG has suggested “periodic” ultrasound examinations to assess fetal growth [16]. Similarly, an ultrasound in late pregnancy to assess for macrosomia risk is suggested [16]. For women

diagnosed with gestational diabetes mellitus, ACOG has recommended fetal growth assessment in the late third trimester in order to assess risk for macrosomia [17]. To our knowledge there are no studies that have ascertained the benefits or harms of this approach.

15.3.2 Nondiabetic Pregnancies

In nondiabetic pregnancies, there are no current recommendations from professional organizations regarding screening for macrosomia [14, 18], given that ACOG does not currently recommend any intervention in the instance of nondiabetic pregnancies with suspected macrosomia and estimated fetal weights below 5000 g. One factor inveighing against fetal weight estimation in nondiabetic pregnancies has been the imprecision of fetal weight estimates near term, with estimates varying from true weight by up to 20% [14].

However a recent cohort study has argued for a universal screening approach [2]. This cohort study of 3866 nulliparous women, the pregnancy outcome prediction study, compared selective, clinically indicated, ultrasound at ≥34 weeks' gestation with universal ultrasound at the same time point. This study enrolled nulliparous women with viable singleton pregnancies and did not exclude women with diabetes and other medical comorbidities. For this study, screen positive for macrosomia was defined as EFW above the 90th percentile for gestational age. The study did not include a clinical protocol for induction of labor or other interventions in the instance of pregnancies that were screen positive for macrosomia. The outcomes for this study included macrosomia >4000 g at birth, severe macrosomia >4500 g, admission to the NICU, and neonatal morbidity, defined as 5-min Apgar <7, and metabolic acidosis at birth (cord pH <7.1, and base deficit >10 mmol/L). The authors defined severe adverse neonatal outcome as live birth with neonatal death, hypoxic-ischemic encephalopathy, need for inotropes, mechanical ventilation, or severe metabolic acidosis at birth defined as cord pH <7.0 and base deficit above 12 mmol/L [2].

The authors found that when LGA fetuses with increased abdominal circumference growth velocity were identified, these fetuses were at significantly increased risk for neonatal morbidity (relative risk 2.0) and severe adverse neonatal outcome (relative risk 6.6). These relationships persisted even after adjustment for maternal diabetes. The authors concluded that universal screening for macrosomia and the use of the abdominal circumference growth velocity would identify pregnancies at risk for adverse neonatal outcomes [2].

Among the individual biometric parameters comprising the estimated fetal weight, the abdominal circumference has been demonstrated to be the most important [19]. A systematic review has compared the predictive accuracy of abdominal circumference with ultrasound EFW [19]. Diagnoses of macrosomia defined as EFW > 90th percentile, EFW > 4000 g, or EFW > 4500 g were compared with abdominal circumference >36 cm alone. The authors identified 36 studies with a total of 19,117 women. The authors constructed summary receiver operator curves and likelihood ratios for each parameter and threshold. They found macrosomia diagnosed by EFW to be equivalent to macrosomia diagnosed by AC >36 cm in the prediction of birth weight above 4000 g or above the 90th percentile for gestational age [19]. They found that positive and negative likelihood ratio for EFW in prediction of birth weight >4000 g were 5.7 (95% CI 4.3–7.6) and 0.48 (95% CI 0.38–0.60). For AC above 36 cm, the positive and negative likelihood ratios were 6.9 (95% CI 5.2, 9.0) and 0.37 (95% CI 0.30, 0.45) [19].

15.4 Causes

In diabetic women, risk for macrosomia has been related to alterations in glucose and insulin homeostasis in both early and late pregnancy. For example, Voldner et al. followed a cohort of 553 nondiabetic White women with Scandinavian heritage throughout pregnancy [20]. The investigators measured fasting glucose twice during pregnancy (14–16 weeks and 30–32 weeks) and fasting plasma insulin and HOMA-IR four times during pregnancy (14–16, 22–24, 30–32, and

36–38 weeks). The primary outcome of interest was birth weight ≥4200 g. This study found that among women with BMI > 27 (top quartile), the increase in fasting plasma glucose between 14 and 16 weeks and between 30 and 32 weeks was predictive of macrosomia. This relationship persisted even when pregnancies complicated by GDM were excluded. Those women in the top quartile who delivered normal weight infants did not show a significant increase in fasting plasma glucose. The investigators found that for the total cohort, fasting plasma glucose at 30–32 weeks' gestation was an independent predictor of macrosomia [20].

Similarly, a case-control study of 37 placentas from macrosomic infants and 37 normal weight infants has determined that insulin-like growth factors and their receptors are important determinants of fetal macrosomia [21]. This study compared placental insulin-like growth factor mRNA levels and their receptors. The authors demonstrated that increased placental IGF-II and IGF-IR mRNA levels were positively correlated with macrosomic birth weights [21].

15.5 Risk Factors

A number of different risk factors are associated with development of fetal macrosomia. For example, Jolly et al. evaluated 350,311 singleton pregnancies in England from 1988 to 1997 [22]. The primary outcomes for this study were birth weight above the 90th percentile for gestational age or >4000 g. Pregestational diabetes was the greatest risk factor for birth weight >90th percentile. Maternal BMI > 30 and parity >4 were the greatest risk factors for birth weight >4000 g. The most important risk factors for birth weight above the 90th percentile for gestational age were pregestational obesity (BMI) > 30 (odds ratio (OR) 2.08; confidence intervals (CI) 1.99, 2.17), pregestational diabetes (OR 6.97; CI 5.36, 8.16), gestational diabetes (OR 2.77; CI 2.51, 3.07), parity > 4 (OR 2.20; CI 2.02, 2.40), and maternal age > 40 (OR 1.22; CI 1.11, 1.35) [22].

A more recent observational study among 178,709 single pregnancies in Chinese women aimed to describe prevalence

and risk factors for macrosomia and to describe associations with adverse outcomes compared with normal birth weight controls [23]. Macrosomia was defined as ≥4000 g at birth. The authors found that maternal obesity and gestational diabetes mellitus were the strongest risk factors for fetal macrosomia in this population [23].

In diabetic women, obesity and excessive weight gain during pregnancy have been associated with large for gestational age, suggesting that lifestyle modification might be important in preventing macrosomia. For example, a cohort study of Florida births over the years 2004–2008 found that prepregnancy obesity and gestational weight gain were independently associated with LGA, defined as ≥90th percentile for gestational age [24]. Similarly, a Chinese cohort study of 1049 women showed that among diabetic women, maternal BMI and pregnancy weight gain had an additive effect on birth weight [25].

A meta-analysis of 33 studies encompassing 88,599 women evaluated the effect of weight gain during pregnancies complicated by GDM on birth weight [26]. This meta-analysis found excessive pregnancy weight gain, in excess of Institute of Medicine guidelines, was associated both with macrosomia and LGA. Conversely, the study demonstrated a reduction in macrosomia among women who gained less than the currently recommended degree of weight during pregnancy [26].

Risk Factors for Macrosomia

Pregestational Diabetes
Maternal BMI > 30
Parity > 4
Excessive weight gain in pregnancy

15.6 Prevention of Macrosomia

Several trials have evaluated the effect of diet and insulin therapy on risk for macrosomia [27–29]. In the Buchanan study, subjects with GDM with abdominal circumference

exceeding the 75th percentile at 29–33 weeks' gestational age were randomly assigned to diet plus insulin therapy versus diet alone. There were 30 subjects assigned to the insulin group and 29 subjects assigned to the diet alone group [27]. This small trial demonstrated that insulin therapy reduced the risk of large for gestational age infants to 13% vs 45%, $P < 0.02$ [27].

A much larger randomized trial of 1000 women who were randomly assigned to receive routine care versus diet therapy plus insulin, if needed, for gestational diabetes mellitus demonstrated that treatment of GDM significantly reduced the risk for macrosomia ≥4 kg from 21 to 10% and reduced large for gestational age, defined as birth weight above the 90th percentile, from 22 to 13% [28]. Likewise, a large multicenter trial of treatment versus usual care for among 958 women with mild gestational diabetes demonstrated that treatment reduced risk for shoulder dystocia (1.5% versus 4.0%), large for gestational age (7.1% versus 14.5%), and macrosomia (5.9% versus 14.3%) [29].

There have also been a number of trials of lifestyle interventions for gestational diabetes mellitus [30]. The Cochrane review of these trials included 15 trials that included 4501 women [30]. The lifestyle interventions that were studied included a combination of education, diet, exercise, and self-monitoring of blood glucose [30]. In six trials, that included 2994 infants, lifestyle interventions reduced risk for large for gestational age births (RR 0.60, 95% CI 0.50, 0.71). Lifestyle interventions were also found to reduce mean birth weight and macrosomia [30].

In nondiabetic women at risk, lifestyle interventions have also been proposed in order to prevent macrosomia. For example, in a randomized controlled trial including 399 nondiabetic women deemed to be at risk for GDM and for macrosomia, dietary and exercise counseling reduced the proportion of newborns who were large for gestational age from 19.7 to 12.1% ($P = 0.042$) [31]. However the intervention had no effect on the proportion of women who developed GDM [31].

Several other studies have evaluated the effect of exercise among overweight women at risk for macrosomic infants [32–34]. In a recent randomized controlled trial, Wang and colleagues randomized 300 overweight and obese pregnant women with BMI ≥ 24 to a stationary cycling exercise intervention three times weekly versus usual activity [33]. The primary outcome measure of this study was gestational diabetes mellitus. Birth weight and macrosomia were pre-specified secondary outcomes. This study demonstrated a reduction in the primary outcome of GDM diagnosis with the exercise intervention (22.0% versus 40.6%, $P < 0.001$). The investigators reported a trend toward a reduction in macrosomia >4000 g (6.3% vs 9.6%; OR, 0.624; 95% CI, 0.233, 1.673, $P = 0.3$) and diagnoses of LGA (14.3% vs 22.8%; OR, 0.564; 95% CI, 0.284, 1.121, $P = 0.1$) that did not reach significance. The study reported a 112 g reduction in mean birth weight for the exercise intervention that was statistically significant. (3345.27 g ± 397.07 g vs 3457.46 g ± 446.00 g; $P = 0.049$) [33].

A Spanish trial that included 765 nondiabetic women tested an intervention that included aerobic exercise, aerobic dance, muscular strength, and flexibility three times weekly for 50–55 min per session [34]. This study was carried out in a low-risk population and did not require obesity or overweight for entry. The primary outcome of the study was pregnancy-induced hypertension. Macrosomia was a pre-specified secondary outcome for the trial. The exercise intervention resulted in a significant reduction in macrosomia, defined as birth weight >4000 g from 4.7 to 1.8%, $P = 0.03$ [34].

However, a recent meta-analysis that evaluated nine trials including 1502 overweight and obese women did not find a reduction in macrosomia with prenatal exercise interventions. (Relative risk 0.92, 95% CI 0.72, 1.18) [32]. The reviewers did however find reductions in gestational diabetes mellitus (RR 0.61, 95% CI 0.41, 0.91) and in preterm delivery <37 weeks (RR 0.62, 95% CI 0.41, 0.95) [32].

15.7 Induction of Labor for Suspected Macrosomia in Nondiabetic Pregnancies

In the instances in which macrosomia is suspected near term, there is controversy regarding whether available inventions of induction of labor or cesarean section would improve outcomes. Some authors have postulated that induction of labor in instances of impending macrosomia might be beneficial to mother and fetus. A recent Cochrane review including four trials with 1190 non-diabetic women found that induction of labor reduced risk for shoulder dystocia (RR 0.60, 95% CI 0.37, 0.98), mean birth weight, and fractures (0.20, 95% CI 0.05, 0.79), but had no effect on the risk for cesarean section or operative vaginal deliveries [35]. This Cochrane review found no differences in other perinatal outcomes of interest; however in one included trial, induction of labor increased risk for maternal third- and fourth-degree perineal lacerations [35]. Another meta-analysis including the same four trials with 1190 participants found that induction of labor reduced the likelihood of birth weights above 4000 and 4500 g as well as fetal fractures but had no significant effect on shoulder dystocia or on mode of delivery [36].

These two meta-analyses were strongly influenced by a large European randomized controlled trial that included 822 women with estimated fetal weight above the 95th percentile for gestational age at 37–38 weeks [37, 38]. Participants were randomized to undergo induction of labor between 37 + 0 and 38 + 6 weeks' gestation versus expectant management until spontaneous onset of labor or other condition necessitating delivery. Potential participants were excluded if they had insulin-requiring diabetes; however women with diet-controlled diabetes were not excluded. The primary composite outcome of this study included shoulder dystocia, fracture of a clavicle or long bone, brachial plexus injury, intracranial hemorrhage, or death. Shoulder dystocia in this study was narrowly defined as difficulty with delivery of the

shoulders that was not relieved by McRoberts maneuver or suprapubic pressure. The definition of clinically significant shoulder dystocia required 60 s or more elapsed time between the delivery of the head and the delivery of the body [37].

Significant findings in this study included a reduction of shoulder dystocia from 4 to 1%, (RR 0.47, 95% CI 0.26, 0.86); the number needed to treat was 25 [37]. Induction of labor significantly reduced the composite primary outcome (relative risk 0.32, 95% CI 0.15, 0.71). Of note, there were no brachial plexus injuries, deaths, or intracranial hemorrhages in either randomized group, although fetal fractures were non-significantly reduced by induction of labor. Induction of labor modestly increased the likelihood of spontaneous vaginal delivery completed to expectant management (RR1.14, 95% CI 1.01, 1.29) [37].

Among secondary outcomes, significant findings included increased antepartum hospital length of stay associated with induction of labor, as well as a higher proportion of infants with neonatal bilirubin concentration ≥250 mm/L in the induction of labor group. The proportion of infants requiring phototherapy after delivery was likewise increased by induction of labor (11% versus 7%, $P = 0.03$). There was no difference in neonatal intensive care unit admissions between the two groups [37].

Because of the early-term gestational age at which induction of labor was carried out in the Boulvain trial [37] and the potential increased neonatal need for phototherapy, ACOG does not currently recommend induction of labor for suspected macrosomia in nondiabetic pregnancies [6]. There is limited evidence as to whether later induction of labor at or beyond 39 weeks might reduce shoulder dystocia or improve neonatal outcomes.

15.8 Induction of Labor for Macrosomia in Diabetic Pregnancies

There is a conflicting body of evidence regarding the utility of induction of labor in diabetic pregnancies in the prevention of shoulder dystocia and macrosomia. Management has

traditionally rested upon a small randomized controlled trial that included 200 women with insulin-requiring diabetes whose fetuses were judged to be appropriate for gestational age in size [39]. One hundred women per group were randomized to either induction of labor at 38 weeks' gestation or expectant management [39]. In this study, induction of labor reduced the prevalence of large for gestational age infants (23% vs 10%) and shoulder dystocia (3% vs 0%) without increasing cesarean section risk [39]. However a smaller trial involving 100 insulin-requiring women with diabetes comparing induction of labor at 38 weeks to induction of labor 40 weeks' gestation did not find any significant difference in the rate of large for gestational age infants in the 38-week induction group compared to the 40-week induction group [40].

Given the paucity of evidence from randomized controlled trials, observational studies and systematic reviews have been also used to address the question of induction of labor for macrosomia in diabetic women [41, 42]. One such cohort study reported 2604 diabetic women and compared usual care with a protocol-based approach for management of macrosomia [41]. The protocol-based approach included a policy of induction of labor for ultrasound EFW of ≥90th percentile at 37–38 weeks' gestation and elective cesarean section for EFW ≥4250 g. Compared with births among diabetic pregnancies before the protocol was instituted, the protocol reduced the shoulder dystocia rate from 2.4 to 1.1% (OR 1.9, 95% CI 1.0, 3.5). Likewise, the likelihood of macrosomia at birth (defined as ≥4000 g) was significantly reduced from 11.6 to 8.9% ($P = 0.04$). The rate of shoulder dystocia among infants delivered vaginally was 7.4% compared with 18.8% among vaginally delivered infants before institution of the labor induction protocol [41].

A 2009 systematic review that compared elective induction or cesarean section with expectant management among women with gestational diabetes evaluated evidence from one randomized controlled trial and four observational studies [2]. The authors reviewed each of the studies separately,

given the heterogeneity of study designs and methods. They concluded that a policy of labor induction at term might reduce macrosomia, defined as birth weight >4000 g, as well as shoulder dystocia, but that the quality of available evidence was low and more trials are needed [42].

15.9 Mode of Delivery

There are no available randomized controlled trials to guide choice of mode of delivery for the fetus with suspected macrosomia. Thus clinical decision-making has rested upon two decision analysis studies by Rouse et al. and by Herbst [43, 44]. The Rouse study constructed a decision analysis model that compared (1) routine care without the use of ultrasound estimation of fetal weight, (2) ultrasound with elective cesarean section for EFW ≥4000 g, and (3) ultrasound and elective cesarean section for EFW ≥4500 g [43]. The main outcome measure for this study was shoulder dystocia with brachial plexus injury. Analyses were carried out separately for diabetic and nondiabetic pregnancies. The study estimated the number of additional cesarean section procedures and costs per permanent brachial plexus injury averted. The authors estimated that 3695 cesarean sections would need to be performed for nondiabetic women with ultrasound EFW ≥4500 g to prevent one brachial plexus injury. For diabetic pregnancies with EFW ≥4500 g, 443 cesarean sections would need to be performed to prevent one brachial plexus injury [43].

The Herbst study constructed a decision model comparing (1) elective cesarean section, (2) labor induction at 38–39 weeks, and (3) expectant management for nondiabetic macrosomic infants with EFW >4500 g [44]. This analysis found that expectant management was the most cost-effective strategy, yielding a cost of $4014.33 per injury-free child, compared to labor induction at $5165.08 per injury-free child or elective cesarean section at $5212.06 per injury-free child [44].

More recently a cohort study encompassed 24 years of births at the Galway University Hospital, Ireland. This study evaluated mode of delivery and neonatal outcomes of 201 births of macrosomic infants with birth weight at or above 5000 g. This study reported a 7.1% incidence of shoulder dystocia among nulliparous women who underwent labor and a 4.3% incidence of shoulder dystocia among parous women who labored [45]. Forty-four percent of the nulliparous women and 12% of the parous women in this study ultimately required intrapartum cesarean section. The overall Erb's palsy rate in the study was 1.3%. The authors concluded that a randomized controlled trial is needed to more fully evaluate the risks and benefits of elective cesarean section for suspected macrosomia [45].

15.10 Summary

Suspected fetal macrosomia remains a controversial area for clinical decision-making. Ultrasound diagnosis of macrosomia remains imprecise despite improvements in technology. There is some intriguing evidence that exercise may reduce risk for gestational diabetes and potentially macrosomia among overweight and obese women who are at risk to give birth to macrosomic infants. In the instances in which macrosomia or impending macrosomia have been diagnosed in nondiabetic women, there is considerable controversy whether the benefits of labor induction outweigh the potential harms. There is very limited evidence suggesting benefit for induction of labor in diabetic women. Recommendations for elective cesarean sections to prevent shoulder dystocia rest upon decision analytic models evaluating costs to avert brachial plexus injuries. Given the rarity of these events, as well as the medicolegal climate, it is unlikely that a definitive trial of elective cesarean section to prevent brachial plexus injury among pregnancies complicated by suspected macrosomia will ever be carried out.

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