

# **Induction of labour or expectant management for suspected macrosomia in term pregnancies: a randomised controlled trial**

## **Literature review**

Before Caesarean section became reasonably safe, induction of labour for suspected macrosomia was performed because it was thought to prevent severe cephalopelvic disproportion and its associated maternal mortality and severe morbidity<sup>1</sup>. Nowadays, some obstetricians induce labour at term when the fetus is estimated to be either large for gestational age or macrosomic, while others prefer to wait until spontaneous onset of labour<sup>2</sup>. Maternal anxiety may be increased by the delivery of a large infant, but women may be reluctant to opt for labour induction. Large-for-dates fetus refers to an estimated weight above the 90th percentile, or more, in utero, while macrosomia is defined as birthweight above 4000 g or a greater cut-off<sup>3</sup>.

The purpose of labour induction in case of suspected fetal macrosomia is to limit fetal growth during the last weeks. This intervention is believed to reduce the likelihood of caesarean section and of difficult operative delivery, which may possibly result in maternal or perinatal morbidity<sup>4</sup>. Observational studies cast doubts on the effectiveness of such a policy<sup>5, 6</sup>. Two randomised controlled trials compared a policy of labour induction with expectant management in women with suspected large fetus<sup>7-9</sup>. These studies were of too small sample size to have sufficient power to show differences according to the evaluated policies.

For a policy of induction to be effective, large for gestational age fetuses must be reliably identified before they become macrosomic. Estimation of the fetal weight is difficult. Clinical estimation based on manual palpation of the uterus or uterine height measurements, as well as ultrasound scanning are currently used methods to attempt at predicting the fetal weight. The predictive value of such tests, especially for large fetuses, is controversial<sup>10</sup>. This may be one of the limitations of a policy of induction of labour for suspected macrosomia.

Another limitation of labour induction for suspected fetal macrosomia is the potential side effects of labour induction, irrespective of the indication for such an intervention. Few randomised controlled trials comparing induction of labour with spontaneous labour onset were conducted. Some trials evaluated elective induction of labour before 41 weeks of gestation<sup>11</sup>. Labour induction was also evaluated in women with premature rupture of membranes and in post-term (beyond 41 weeks) pregnancies<sup>12, 13</sup>. Beside the above-mentioned trials, several retrospective studies were conducted<sup>14</sup>. These retrospective studies are of limited validity, because potential confounding factors may bias the estimate of the risk associated with labour induction.

### **1 *Definition: Large for Gestational Age and macrosomia***

Macrosomia is usually defined by a birthweight above 4000 g or greater cut-offs.<sup>15</sup> Several limits are proposed, as the incidence of maternal and neonatal morbidity is increasing with increasing weight. Guidelines for obstetric management are usually based on a cut-off of 4000 g and 4500 g.<sup>16, 17</sup>

A large for gestational age fetus is usually defined when the estimated weight is above the 90th percentile, or a greater cut-off, for the gestational age at which the estimation was made. This estimate is made to predict the delivery of a macrosomic infant. Similarly, an infant is large for gestational age when birthweight is above the 90th percentile for the gestational age at birth. Numerous reference charts, based on birthweight, have been proposed. Recently, charts based on a large collaborative study in the French population have been developed.<sup>18</sup> Birthweights at the 90<sup>th</sup> and the 95<sup>th</sup> percentile for the 36<sup>th</sup> to 42<sup>nd</sup> week of pregnancy are reproduced below. Male fetuses estimated at more than the 90<sup>th</sup> percentile and female fetuses estimated at more than the 95<sup>th</sup> percentile are at risk of having a birthweight of more than 4000 g at 40 weeks, which is the average gestational age at delivery when no intervention is performed.

Week	Boys		Girls	
	p90	p95	p90	p95
36	3354	3534	3225	3405
37	3555	3727	3416	3588
38	3731	3896	3584	3748
39	3884	4044	3729	3887
40	4017	4076	3854	4009
41	4131	4296	3958	4117
42	4227	4408	4042	4212

## 2 Etiologic factors of macrosomia

The growth and development of the fetus are regulated by and dependent on numerous factors that includes genetic and hormonal factors, uterine environment, placenta function and the availability of nutrients to mother and fetus.

The initial drive for growth is genetic. By mechanisms that remain poorly defined, there is a genetic control of cell growth and differentiation that is the basic determinant of species size at birth. Male gender contributes approximately 150-200 g of increased birthweight, compared with female infants at term. Macrosomia as part of genetic syndrome accounts for only a small portion of cases. Specific syndromes, including Beckwith-Wiedemann, Sotos and others rare syndromes are associated with fetal macrosomia.

Maternal weight before pregnancy is an important determinant of fetal weight<sup>19</sup>. Parous women are two to three times more likely than nulliparous women to have macrosomic infants<sup>20</sup>. Multiparity and age over 35 years are also significant risk factors for macrosomia<sup>21</sup>. Some authors showed that birthweight has increased over time.

The difference in fetal size becomes apparent in the third trimester. Fetal growth in late gestation can be considered the result of the interrelation between the genetic cause of growth and constraining influences that inhibit growth. The balance between genetic and exogenous influences (maternal nutrition, placental factors) is probably controlled by fetal hormones. Clinical and experimental evidence has indicated that insulin can be considered as the true fetal growth hormone.<sup>22</sup> Nesidioblastosis, an autosomal

recessive genetic disorder, is a diffuse or disseminated proliferation of pancreatic islet cells associated with macrosomia. Chronic fetal hyperglycaemia accelerates the development of insulin secretory mechanisms, predisposing infants of mothers with diabetes to have higher levels of insulin and macrosomia.

### **3 Consequences of macrosomia**

#### **3.1 Cephalopelvic disproportion and maternal morbidity**

Cephalopelvic disproportion is defined as a disparity between the size of the fetal head and the size of the maternal pelvis, precluding vaginal delivery. Clinically, CPD is diagnosed when labour is prolonged.

A large fetal weight is associated with an increased risk of cephalopelvic disproportion, dysfunctional labour, and failure of engagement of the fetal head. As a consequence, the risk of caesarean section and instrumental delivery is increased. Long-term complications, including perineal trauma, urinary and fecal incontinence are more frequent when the fetus is large.<sup>23, 24</sup>

The risk of caesarean section is increased 2 to 3 times when the fetus is macrosomic.<sup>25</sup> Maternal morbidity associated with caesarean section includes thromboembolism, haemorrhage, wound complications, endometritis and urinary tract infection.<sup>2</sup> The risk of complications is higher when the caesarean section is performed as an emergency, compared to elective procedures.<sup>26</sup> Prolonged first and second stage of labour is more frequent when the fetus is macrosomic.<sup>27</sup> Use of oxytocin for labour augmentation may cause uterine hyperstimulation, with the attendant risk of fetal heart rate abnormalities. Prolonged second stage of labour leads to instrumental delivery, which is associated with maternal morbidity (uterine rupture, cervico-vaginal, perineal and anal sphincter tears, and haemorrhage). A large study conducted in Switzerland showed that prolonged first and second stage of labour, caesarean section and operative delivery are significantly more frequent when birthweight is more than 4500 g<sup>28</sup>.

#### **3.2 Shoulder dystocia and neonatal morbidity**

Shoulder dystocia is a dramatic emergency with devastating consequences for both the mother and the neonate. The cause of the dystocia is the impaction of the anterior shoulder on the symphysis pubis.<sup>29</sup> Shoulder dystocia is defined as a delivery of the shoulders requiring manoeuvres in addition to downward traction and episiotomy. A specific duration between the delivery of the fetal head and the completion of the delivery, i.e. more than 60 seconds, was also proposed to define shoulder dystocia.<sup>30, 31</sup> The incidence of shoulder dystocia in the general obstetric population was estimated to be between 1% and 4% of vaginal births in vertex presentation<sup>17</sup>. The estimate depends on the population and the criteria used to define shoulder dystocia. The retrospective nature of most of the studies may also underestimate the incidence of this accident.<sup>31</sup>

The risk of shoulder dystocia is associated with higher fetal weight<sup>32</sup>. Diabetes during pregnancy is a significant risk factor, because of the risk of large-for-dates fetus and of a larger biacromial diameter. Other risk factors include prolonged first and second stage of labour, instrumental delivery and past history of shoulder dystocia.<sup>33</sup> When birthweight is above 4000 g, the risk of shoulder dystocia was estimated to be 10 times higher than

with lower birthweight<sup>34</sup>. The relative risk when birthweight is above 4500 g is about 20, compared to average weight infants.<sup>28</sup>

Maternal and neonatal morbidity following shoulder dystocia are severe. Among 98 women with shoulder dystocia, 19% had severe perineal tears, 14% had postpartum haemorrhage and 1% had uterine rupture, compared to 0.5%, 0.4% and 0.06%, respectively, in the general obstetric population of the same hospital<sup>35</sup>. Shoulder dystocia was found to be significantly associated with perineal morbidity, in a study including 390 women with 4<sup>th</sup> degree perineal tear<sup>36</sup>.

Neonatal complications in case of shoulder dystocia include brachial plexus injury, bone fracture, asphyxia and death. In the case of shoulder dystocia, 12% are complicated by brachial plexus palsy, 4% by severe asphyxia and 0.3% by neonatal death.<sup>37</sup> Erb-Duchenne palsy, the most frequent trauma of the brachial plexus (80%), results from injury of C5-C6 roots<sup>38</sup>. Affected neonates have adducted arm, with internal rotation, extension of the elbow and pronation of the forearm. This palsy improves in 80% of the cases after 3 to 6 months.<sup>39</sup> Klümpke palsy involves the lower roots (C7-T1) of the brachial plexus. The prognosis is poorer, as only 40% recover after 6 months. A combination of both palsies has an even worse prognosis. The clavicle is the bone which is the most frequently fractured during a traumatic delivery. This occurs in 0.4% to 1.1% of all deliveries, and a relative risk of 5 was reported with high birthweight<sup>28</sup>. The incidence may be underestimated, as some fractures are not diagnosed. These fractures usually heal spontaneously without sequelae, but are cause of concern for both the medical staff and the parents. Perinatal death following shoulder dystocia is rare. A total of 56 deaths were reported voluntarily in the UK, which gives an incidence of 2/100 000 births<sup>40</sup>. In Kuwait, a mortality of 7% among 96 cases of shoulder dystocia was observed<sup>35</sup>

Prompt diagnosis and treatment of shoulder dystocia are important to minimise the consequence of this dramatic emergency. Birth asphyxia is a consequence of delay, with cord blood pH decreasing by 0.04 units per minute after the delivery of the head. In macrosomic fetuses, severe neonatal asphyxia is reported in 1.4% after a delivery without shoulder dystocia and in 14.3% with shoulder dystocia. Shoulder dystocia was shown to be a significant and independent risk factor for the occurrence of neonatal seizures<sup>41</sup>.

In a decision analysis model, Rouse and Goldenberg estimated the cost-effectiveness of elective caesarean section for the prevention of morbidity related to the delivery of large-for-dates fetuses<sup>42</sup>. The risk of shoulder dystocia was estimated to be 7% when birthweight is between 4000 to 4499 g in non-diabetic women and 14% in diabetic women. With a birthweight of more than 4500 g, the risk increased to 15% and 50%, respectively. Because of the difficulties in estimating fetal weight and the risks and costs associated with caesarean section, their conclusions were that an elective caesarean section to prevent shoulder dystocia is not recommended when fetal weight is below 4500 g, or 4200 g in diabetic women. Given the low cost-effectiveness ratio of elective caesarean section, induction of labour might be an alternative strategy to prevent the occurrence of shoulder dystocia and its consequences.

## **4 Detection of macrosomia**

For a policy of induction of labour to be effective, large for gestational age fetuses must be reliably identified before they become macrosomic. Clinical estimation based on manual palpation of the uterus or uterine height measurements, as well as ultrasound scanning are currently used methods to attempt at predicting the fetal weight. The predictive value of such tests, especially for large fetuses, is controversial.<sup>10</sup> Maternal estimates of fetal weight, compared to that of a previous pregnancy, are as reliable as the estimation made by clinicians<sup>43</sup>. The limitation of this approach is that it is not applicable to nulliparous women.

### **4.1 Clinical estimates of birthweight**

Clinical estimates of fetal weight are usually based on Leopold manoeuvres or on symphysis fundus height measurement.<sup>44</sup> These estimates were said to be less precise at the extremes of birthweight<sup>45</sup>. Reported sensitivity and specificity of clinical estimates of fetal weight, based on Leopold manoeuvres, vary greatly. When clinical estimates of fetal weight were assessed in comparison with a birthweight 4000 g or more, sensitivities ranged from 24 to 97% with specificities ranging from 98 to 82%. Clinical estimates were analysed with the use of ROC curve. The area under the curve for clinical estimates was 0.84, significantly greater than 0.50, which is the area under the curve of a useless test<sup>46</sup>. The authors of a recent review conclude that clinical estimation of fetal weight is a useful test for predicting birthweight.<sup>47</sup>

Other authors estimated the prediction of birthweight above 4000 g based on symphysis fundus height measurement.<sup>48, 49</sup> A height of more than 38 cm was shown to be accurate in the prediction of birthweight above 4000 g.

### **4.2 Sonographic estimates of birthweight**

Ultrasound is the most commonly used and widely studied method for fetal weight estimation. Sonographic methods for the diagnosis of macrosomia were developed to improve clinical estimates. Most sonographers use fetal biparietal diameter, abdominal circumference and femur length to estimate fetal weight. These parameters are combined using data-derived formulas, which best fit the specific population from which the method was developed. Using any of the formulas with other populations yield errors of 7 to 10%. Benacerraf reported the prediction of birthweight in 1301 fetuses, of which 324 were greater than 4000 g, by sonographic estimated fetal weight based on abdominal diameter and biparietal diameter. The sensitivity was 65% and the specificity was 90% for predicting a birthweight greater than 4000 g. In the same group of women, the sensitivity improved to 82% and the specificity decreased to 79% when a cut-off of 3800 g was used for the estimated fetal weight instead of 4000 g.

### **4.3 Comparison between clinical and sonographic estimates of fetal weight**

A meta-analysis of studies in which both clinical and sonographic estimates were performed showed that 67% of clinical estimates and 66% of sonographic estimates were within 10% of the actual birthweight.<sup>47</sup> The mean absolute error of clinical and sonographic estimates of birthweight was 300 g. Among reports of comparison between clinical and sonographic estimates, three reported the accuracy of clinical estimates

when the birthweight was  $\geq 4000$  g. In these studies, 58% of clinical estimates were within 10% of birthweight, compared with 51% of sonographic estimates<sup>46, 50, 51</sup>.

When clinical estimates of fetal weight were compared with sonographic estimates in parallel samples, clinical estimates performed favourably. A large study compared clinical and sonographic suspicion of macrosomia (fetal weight at or above 4000 g), with actual macrosomia, defined as birthweight at or above 4000 g. Clinical diagnosis had a sensitivity of 54% and a positive predictive value of 60%, while sonographic diagnosis had a slightly higher sensitivity (71%), but lower positive predictive value (55%). ROC curves for both clinical and sonographic predictions of macrosomia subsume areas between 0.81 and 0.95, significantly larger than the area of 0.5 that indicates a useless test. Thus, these tests are defined as useful from a statistical point of view. Prediction of macrosomia by clinical techniques and ultrasound is limited by false positives and false-negatives cases, but, nevertheless, these tests identify a population with high risk of macrosomia.

To prepare this randomised controlled trial, we have also evaluated the reliability of fetal weight estimates performed in our clinic. In a sample of 136 pregnancies with birthweight of 4000 g or more and 153 randomly selected controls. These controls represent a known fraction (1:8) of the total births in our clinic during the same period, and computations were adjusted to take into account the sampling procedure. Sensitivity of the clinical estimate was 61% and predictive value was 49%. With a similar methodology, sonographic estimates had a sensitivity of 73% and a positive predictive value of 71%. Our results were similar to those reported in the literature. We have also evaluated retrospectively the risk of caesarean section for women having induction of labour. As the indication for induction of labour was not recorded in our database, we included women delivered at 37 to 39 weeks with a neonate with birthweight above 3800 g, a group in whom the majority of inductions are performed for suspected large-for-dates fetus. Primiparous women with induction of labour had a higher risk of caesarean section, compared to women who had spontaneous labour. The risk was similar in multiparas. Limitations of this analysis include its retrospective nature and that we were unable to adjust for potential confounders.

## **5 Induction of labour for suspected macrosomia**

Many obstetricians induce labour at term when the fetus is estimated to be either large for gestational age or macrosomic. The purpose of labour induction in case of suspected fetal macrosomia is to reduce the likelihood of Caesarean section and of difficult operative delivery, possibly resulting in maternal or perinatal morbidity. Observational studies cast doubts on the effectiveness of such a policy.<sup>6, 52</sup> For a policy of induction to be effective, large for gestational age fetuses must be reliably identified before they become macrosomic. This may be one of the limitations of a policy of induction of labour for suspected macrosomia.

Two randomised controlled trials comparing induction of labour to expectant management when the fetus is suspected to be macrosomic were included in this review.<sup>7, 8</sup> Women were included when fetal weight, estimated by ultrasound examination, was between 4000 g and 4500 g<sup>7</sup> or between 4000 g and 4750 g<sup>8</sup>. Diabetic women were excluded from both trials. A computer-generated table of random numbers was used in both studies. The method for concealment of the

allocation was by sealed, sequentially numbered, opaque envelopes in one of the trials<sup>8</sup>, while the method was not described in the other report<sup>7</sup>. The method used for labour induction was dependent on the cervical status (prostaglandins for cervical ripening in the case of an unfavourable cervix, otherwise oxytocin infusion). In one of the trials<sup>7</sup>, elective Caesarean section was performed when estimated fetal weight was greater than 4500 g and women in the expectant management group underwent induction of labour upon completion of 42 weeks of gestation.

A total of 313 women were included in the two studies. Compared to expectant management, induction of labour for suspected macrosomia did not reduce the risk of caesarean section (Odds ratio 0.85, 95% confidence interval 0.50 to 1.46) or instrumental delivery (OR 0.98, 95%CI 0.48 to 1.98). Shoulder dystocia was similar between groups (OR 1.07, 95%CI 0.41 to 2.81). Perinatal morbidity was infrequent and similar between groups. Two cases of brachial plexus injury and 4 cases of fracture were reported in the expectant management group. Three and two cases of intracranial haemorrhage were diagnosed by sonography in the induced and expectant group, respectively.<sup>7</sup> No information is available regarding mother's views on their care, urinary and fecal incontinence, and sexual dysfunction or on other long-term morbidity. There is presently no evidence that induction of labour for suspected fetal macrosomia modifies the risk of caesarean section or instrumental delivery. The studies were however of too small sample size to exclude even large effects on these outcomes. There is also very limited evidence to quantify the effect on maternal and neonatal morbidity.<sup>9</sup>

## **6 Risks associated with induction of labour**

The results of numerous observational studies suggested that induction of labour is associated with an increased risk of caesarean section<sup>14, 53-57</sup>. Two other observational studies, conducted in contexts of very high rates of labour induction (35 and 50% respectively), reported a 30% reduction in the risk of caesarean section associated with labour induction<sup>58, 59</sup>. It is likely that residual confounding by the indication may bias the above estimates of risk associated with labour induction.

We conducted a retrospective cohort study in a tertiary care hospital in Quebec (Canada).<sup>14</sup> A total of 7430 women, not referred from another institution, with a single baby in vertex presentation and delivering between 38 and 40 weeks of pregnancy were included. Among these women, 3546 were excluded for pre-labour pregnancy complications. Relative risks (RR), adjusted for parity, were computed to compare 3353 women who went into labour spontaneously with 531 women whose labour was induced. Induction of labour was found to be associated with a higher risk of caesarean section (RR 2.4, 95%CI 1.8, 3.4). Use of non-epidural (RR 1.5, 95%CI 1.2, 1.8) and of epidural analgesia (RR 1.4, 95%CI 1.1, 1.7) was more frequent after labour induction. Resuscitation (RR 1.2, 95%CI 1.0, 1.5), admission to the intensive care unit (RR 1.6, 95%CI 1.0, 2.4) and phototherapy (RR 1.3, 95%CI 1.0, 1.6) were more frequent after induction of labour. Results were similar when controlling simultaneously for parity, maternal age, gestational age, year of delivery, birthweight and the physician in charge of delivery in a logistic regression analysis. The results of this study suggest that induction of labour is associated with a higher risk of caesarean section and of some perinatal adverse outcomes. Induction of labour should be reserved for cases where maternal and perinatal benefits outweigh the risk of these complications. It is therefore

important to evaluate the balance between the benefits and the risks in specific situations, e.g. in suspected macrosomia.

In contrast with the results of observational studies, randomised controlled trials evaluating labour induction do not show an increased morbidity associated with the intervention. A systematic review of randomised controlled trials showed elective induction of labour before 41 weeks to be associated with more use of non-epidural analgesia, more operative vaginal deliveries and a reduction of meconium stained amniotic fluid, but there was no difference in caesarean section between groups<sup>11</sup>. A large trial, including women with post-term pregnancy (>41 weeks), showed a reduction in the risk of caesarean section when induction of labour was performed, compared to expectant management.<sup>12</sup> A systematic review of the randomised controlled trials suggested that routine induction of labour after 41 weeks reduced perinatal mortality (OR 0.2, 95%CI 0.1 to 0.9) and the risk of caesarean section (OR 0.9, 95%CI 0.8 to 1.0). In women with prelabour rupture of membranes, induction of labour was shown to decrease the risk of infection, of perinatal mortality without increase in caesarean section, when compared to expectant management.<sup>13</sup> For pregnant diabetic women treated with insulin, a relatively small trial suggested that induction of labour at 38 weeks do not increase the risk of caesarean section<sup>60, 61</sup>.

We have conducted a systematic review on “Induction of labour for suspected macrosomia” for the Cochrane Collaboration.<sup>9</sup> We searched Medline and the Cochrane Controlled Trials Register. The results of this systematic review and meta-analysis are reported above. It should be noted that, in both trials, groups had similar mean gestational age at delivery and mean birthweight. Both studies included pregnancies around 40 weeks with an estimated fetal weight of more than 4000 g. The intervention was possibly not performed early enough to avoid excessive birthweight and to decrease maternal and neonatal risks associated with macrosomia.<sup>9</sup> Our conclusions were that there is presently no evidence that induction of labour for suspected fetal macrosomia decreases or increases the risk of caesarean section, instrumental delivery, and maternal or neonatal morbidity. The studies were however of too small sample size to exclude even large effects on these outcomes.<sup>9</sup> We performed also a systematic review on “Elective delivery in diabetic pregnant women”, a subject related to macrosomia<sup>61</sup>. This review also showed that evidence is limited for evidence-based decision-making in this situation.

There is at present no valid evidence that induction of labour for specific indications increases the risk of caesarean section or of other maternal or neonatal morbidity.

## References

1. Thiery M, Baines C, Keirse M. The development of methods for inducing labour. In: Chalmers I, Enkins M, Keirse MJNC, editors. *Effective care in pregnancy and childbirth*. Oxford: Oxford University Press; 1989. p. 969-80.
2. Irion O, Hirsbrunner Almagbaly P, Morabia A. Planned vaginal delivery versus elective caesarean section: a study of 705 singleton term breech presentations. *Br J Obstet Gynaecol* 1998;105:710-7.
3. Goffinet F. [Is cesarean section indicated for suspected macrosomia?]. *J Gynecol Obstet Biol Reprod* 2000;29 Suppl 2:22-9.
4. Perlow JH, Wigton T, Hart J, Strassner HT, Nageotte MP, Wolk BM. Birth trauma. A five-year review of incidence and associated perinatal factors. *J Reprod Med* 1996;41:754-60.
5. Weeks JW, Pitman T, Spinnato JA, 2nd. Fetal macrosomia: does antenatal prediction affect delivery route and birth outcome? *Am J Obstet Gynecol* 1995;173:1215-9.
6. Friesen CD, Miller AM, Rayburn WF. Influence of spontaneous or induced labor on delivering the macrosomic fetus. *Am J Perinatol* 1995;12:63-6.
7. Gonen O, Rosen DJ, Dolfin Z, Tepper R, Markov S, Fejgin MD. Induction of labor versus expectant management in macrosomia: a randomized study. *Obstet Gynecol* 1997;89:913-7.
8. Tey A, Eriksen N, Blanco J. A prospective randomized trial of induction versus expectant management in nondiabetic pregnancies with fetal macrosomia. *Am J Obstet Gynecol* 1995;172:293.
9. Irion O, Boulvain M. Induction of labour for suspected fetal macrosomia. *Cochrane Database Syst Rev* 2000;2.
10. Johnstone FD, Prescott RJ, Steel JM, Mao JH, Chambers S, Muir N. Clinical and ultrasound prediction of macrosomia in diabetic pregnancy. *Br J Obstet Gynaecol* 1996;103:747-54.
11. Crowley P. Interventions for preventing or improving the outcome of delivery at or beyond term. *Cochrane Database Syst Rev* 2000;2.
12. Hannah ME, Hannah WJ, Hellmann J, Hewson S, Milner R, Willan A. Induction of labor as compared with serial antenatal monitoring in post-term pregnancy. A randomized controlled trial. *N Engl J Med* 1992;326:1587-92.
13. Hannah ME, Ohlsson A, Farine D, Hewson SA, Hodnett ED, Myhr TL, et al. Induction of labor compared with expectant management for prelabor rupture of the membranes at term. *N Engl J Med* 1996;334:1005-10.
14. Boulvain M, Marcoux S, Bureau M, Fortier M, Fraser W. Risks of induction of labour in uncomplicated term pregnancies. *Paediatr Perinat Epidemiol* 2001;15:131-8.
15. Delpapa EH, Mueller-Heubach E. Pregnancy outcome following ultrasound diagnosis of macrosomia. *Obstet Gynecol* 1991;78:340-3.
16. Fetal macrosomia. ACOG Technical Bulletin Number 159. *Int J Gynaecol Obstet* 1992;39:341-5.
17. Shoulder dystocia. ACOG Practice Patterns. Number 7. *Int J Gynaecol Obstet* 1998;60:306-13.
18. Mamelie N, Munoz F, Grandjean H. [Fetal growth from the AUDIPOG study. I. Establishment of reference curves]. *J Gynecol Obstet Biol Reprod* 1996;25:61-70.

19. Anderson GD, Blidner IN, McClemon S, Sinclair JC. Determinants of size at birth in a Canadian population. *Am J Obstet Gynecol* 1984;150:236-44.
20. Modanlou HD, Komatsu G, Dorchester W, Freeman RK, Bosu SK. Large-for-gestational-age neonates: anthropometric reasons for shoulder dystocia. *Obstet Gynecol* 1982;60:417-23.
21. Boyd ME, Usher RH, McLean FH. Fetal macrosomia: prediction, risks, proposed management. *Obstet Gynecol* 1983;61:715-22.
22. Hill DE. Fetal effects of insulin. *Obstet Gynecol Annu* 1982;11:133-49.
23. Faltin DL, Sangalli MR, Curtin F, Morabia A, Weil A. Prevalence of anal incontinence and other anorectal symptoms in women. *Int Urogynecol J Pelvic Floor Dysfunct* 2001;12:117-120.
24. Hojberg KE, Salvig JD, Winslow NA, Bek KM, Laurberg S, Secher NJ. Flatus and faecal incontinence: prevalence and risk factors at 16 weeks of gestation. *Br J Obstet Gynaecol* 2000;107:1097-103.
25. Spellacy WN, Miller S, Winegar A, Peterson PQ. Macrosomia--maternal characteristics and infant complications. *Obstet Gynecol* 1985;66:158-61.
26. Lilford RJ, van Coeverden de Groot HA, Moore PJ, Bingham P. The relative risks of caesarean section (intrapartum and elective) and vaginal delivery: a detailed analysis to exclude the effects of medical disorders and other acute pre-existing physiological disturbances. *Br J Obstet Gynaecol* 1990;97:883-92.
27. McFarland M, Hod M, Piper JM, Xenakis EM, Langer O. Are labor abnormalities more common in shoulder dystocia? *Am J Obstet Gynecol* 1995;173:1211-4.
28. Bleichenbacher M, Haenel AF. Das perinatale Risiko bei erheblicher Makrosomie. Untersuchung aufgrund der Datenbank der Arbeitsgemeinschaft schweizerischer Frauenkliniken (ASF) 1983-1992. *Geburtshilfe und Frauenheilkunde* 1995;55:339-44.
29. Benedetti TJ, Gabbe SG. Shoulder dystocia. A complication of fetal macrosomia and prolonged second stage of labor with midpelvic delivery. *Obstet Gynecol* 1978;52:526-9.
30. Beall MH, Spong C, McKay J, Ross MG. Objective definition of shoulder dystocia: a prospective evaluation. *Am J Obstet Gynecol* 1998;179:934-7.
31. Spong CY, Beall M, Rodrigues D, Ross MG. An objective definition of shoulder dystocia: prolonged head-to-body delivery intervals and/or the use of ancillary obstetric maneuvers. *Obstet Gynecol* 1995;86:433-6.
32. Nesbitt TS, Gilbert WM, Herrchen B. Shoulder dystocia and associated risk factors with macrosomic infants born in California. *Am J Obstet Gynecol* 1998;179:476-80.
33. Smith RB, Lane C, Pearson JF. Shoulder dystocia: what happens at the next delivery? *Br J Obstet Gynaecol* 1994;101:713-5.
34. Nocon JJ, McKenzie DK, Thomas LJ, Hansell RS. Shoulder dystocia: an analysis of risks and obstetric maneuvers. *Am J Obstet Gynecol* 1993;168:1732-7; discussion 1737-9.
35. el Madany AA, Jallad KB, Radi FA, el Hamdan H, O'Deh H M. Shoulder dystocia: anticipation and outcome. *Int J Gynaecol Obstet* 1991;34:7-12.
36. Goldaber KG, Wendel PJ, McIntire DD, Wendel GD. Postpartum perineal morbidity after fourth-degree perineal repair. *Am J Obstet Gynecol* 1993;168:489-93.
37. Sandmire HF, O'Halloin TJ. Shoulder dystocia: its incidence and associated risk factors. *Int J Gynaecol Obstet* 1988;26:65-73.

38. Buschmann WR, Sager G. Orthopaedic considerations in obstetric brachial plexus palsy. *Orthop Rev* 1987;16:290-2.
39. Curran JS. Birth-associated injury. *Clin Perinatol* 1981;8:111-29.
40. Hope P, Breslin S, Lamont L, Lucas A, Martin D, Moore I, et al. Fatal shoulder dystocia: a review of 56 cases reported to the Confidential Enquiry into Stillbirths and Deaths in Infancy. *Br J Obstet Gynaecol* 1998;105:1256-61.
41. Patterson CA, Graves WL, Bugg G, Sasso SC, Brann AW, Jr. Antenatal and intrapartum factors associated with the occurrence of seizures in term infant. *Obstet Gynecol* 1989;74:361-5.
42. Rouse DJ, Owen J, Goldenberg RL, Cliver SP. The effectiveness and costs of elective cesarean delivery for fetal macrosomia diagnosed by ultrasound. *JAMA* 1996;276:1480-6.
43. Chauhan SP, Lutton PM, Bailey KJ, Guerrieri JP, Morrison JC. Intrapartum clinical, sonographic, and parous patients' estimates of newborn birth weight. *Obstet Gynecol* 1992;79:956-8.
44. Persson B, Stangenberg M, Lunell NO, Brodin U, Holmberg NG, Vaclavinkova V. Prediction of size of infants at birth by measurement of symphysis fundus height. *Br J Obstet Gynaecol* 1986;93:206-11.
45. Loeffler FE. Clinical foetal weight prediction. *J Obstet Gynaecol Br Commonw* 1967;74:675-7.
46. Chauhan SP, Cowan BD, Magann EF, Bradford TH, Roberts WE, Morrison JC. Intrapartum detection of a macrosomic fetus: clinical versus 8 sonographic models. *Aust N Z J Obstet Gynaecol* 1995;35:266-70.
47. O'Reilly-Green C, Divon M. Sonographic and clinical methods in the diagnosis of macrosomia. *Clin Obstet Gynecol* 2000;43:309-20.
48. Walraven GE, Mkanje RJ, van Roosmalen J, van Dongen PW, van Asten HA, Dolmans WM. Single pre-delivery symphysis-fundal height measurement as a predictor of birthweight and multiple pregnancy. *Br J Obstet Gynaecol* 1995;102:525-9.
49. Grover V, Usha R, Kalra S, Sachdeva S. Altered fetal growth: antenatal diagnosis by symphysis-fundal height in India and comparison with western charts. *Int J Gynaecol Obstet* 1991;35:231-4.
50. Chauhan S, Hendrix N, Magann E, Morrison J, Kenney S, Devoe L. Limitations of clinical and sonographic estimates of birth weight: experience with 1034 parturients. *Obstet Gynecol* 1998;91:72-7.
51. Sherman DJ, Arieli S, Tovbin J, Siegel G, Caspi E, Bukovsky I. A comparison of clinical and ultrasonic estimation of fetal weight. *Obstet Gynecol* 1998;91:212-7.
52. Weeks J, Pitman T, Spinnato J. Fetal macrosomia: Does antenatal prediction affect delivery route and birth outcome? *Am J Obstet Gynecol* 1995;173:1215-9.
53. Vierhout ME, Out JJ, Wallenburg HC. Elective induction of labor: a prospective clinical study, I: Obstetric and neonatal effects. *J Perinat Med* 1985;13:155-62.
54. Yudkin P, Frumar AM, Anderson AB, Turnbull AC. A retrospective study of induction of labour. *Br J Obstet Gynaecol* 1979;86:257-65.
55. Macer JA, Macer CL, Chan LS. Elective induction versus spontaneous labor: a retrospective study of complications and outcome. *Am J Obstet Gynecol* 1992;166:1690-6; discussion 1696-7.
56. Smith LP, Nagourney BA, McLean FH, Usher RH. Hazards and benefits of elective induction of labor. *Am J Obstet Gynecol* 1984;148:579-85.

57. Yeast JD, Jones A, Poskin M. Induction of labor and the relationship to cesarean delivery: A review of 7001 consecutive inductions [see comments]. *Am J Obstet Gynecol* 1999;180:628-33.
58. Boisselier P, Peter J, Trouslard D. La programmation de l'accouchement. Bilan de 5 années d'activité et 1752 déclenchements du travail. *J Gynecol Obstet Biol Reprod* 1991;20:1131-40.
59. Clinch J. Induction of labour--a six year review. *Br J Obstet Gynaecol* 1979;86:340-2.
60. Kjos SL, Henry OA, Montoro M, Buchanan TA, Mestman JH. Insulin-requiring diabetes in pregnancy: a randomized trial of active induction of labor and expectant management. *Am J Obstet Gynecol* 1993;169:611-5.
61. Boulvain M, Stan C, Irion O. Elective delivery in diabetic pregnant women. *Cochrane Database Syst Rev* 2000;2.