Obstetric and perinatal outcomes in type 1 diabetic pregnancies – a large, population-based study

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Objective: to perform comparative analyses of obstetric and perinatal outcomes between type 1 diabetic pregnancies (T1DM) and the general obstetric population in Sweden between 1991 and 2003.

Research design and Methods: Population based study. Data were obtained from the Medical Birth Registry, covering more than 98% of all pregnancies in Sweden. A total of 5,089 T1DM pregnancies and 1,260,207 controls were included. Odds ratios (OR) were adjusted for group differences in maternal age, parity, BMI, chronic hypertensive disease, smoking habits and ethnicity.

Results: In T1DM, preeclampsia was significantly more frequent [OR=4.47 (3.77–5.31)] as well as delivery with Cesarean section [OR=5.31 (4.97–5.69)] compared to the general population. Stillbirth [OR=3.34 (2.46–4.55)], perinatal mortality [OR=3.29 (2.50–4.33)] and major malformations [OR=2.50 (2.13–2.94)] were more common in T1DM than in controls. Risk of very preterm birth (<32 gestational weeks) was also higher among T1DM women [OR=3.08 (2.45–3.87)]. The incidence of fetal macrosomia (birth weight 2 SD or more above the mean) was much increased in the diabetes group [OR=11.45 (10.61–12.36)].

Conclusion: T1DM in pregnancy is still associated with considerably increased rates of adverse obstetric and perinatal outcomes. The 8-fold increased risk for fetal macrosomia in T1DM pregnancies is unexpected and warrants further investigation.
Type 1 diabetes (T1DM) in pregnancy is associated with increased risks of maternal and fetal complications. In a Swedish study from 1982 to 1985, the perinatal mortality (3.1%) and stillbirth rate (2.1%) were 4-5 times that of the general population (1). Since then and following the introduction of tight glycemic control, outcomes of T1DM pregnancies are considered to have improved significantly. However, objective and unanimous estimates of improvement in care are difficult to find. More recent studies on T1DM pregnancies have reported varying results – stillbirth and perinatal mortality were still significantly increased in some studies (2-6), the risk for major malformations varied from 2 to 10 times normal (2, 4, 6) and the incidence of fetal macrosomia remained markedly increased, despite apparently good metabolic control with HbA1C levels close to the normal range (7, 8). Accordingly, the aim of the ST Vincent declaration from 1989 (9) – i.e. to abolish the over risks associated with pregnancy in women with T1DM by the end of the last century - has not been achieved. There is no clear explanation for the wide variation in perinatal outcomes for diabetic pregnancies. Differences in organisation of healthcare, socioeconomic factors, maternal characteristics and patient compliance could account for some of the observed differences. More favourable results reported from centres of excellence may reflect some selection bias. Given the relatively low incidence of congenital malformations and perinatal mortality, large studies are required for accurate risk estimates. And variations in outcomes for T1DM pregnancies may not only be associated with diabetes but also with other risk factor variations in the general obstetric population.

The primary aim of this study was to provide solid evidence for obstetric and perinatal outcomes in T1DM pregnancies. We compared outcomes in over 5,000 type 1 diabetic pregnancies with that of the general obstetric population in Sweden over a time period of thirteen years (1991 – 2003). The large numbers included in the study offered a possibility to obtain objective and precise estimates of complications in diabetic pregnancies and to adjust for potential confounders. To facilitate interpretation of our results in relation to organization of care and change over time, we compared hospitals in relation to the number of T1DM pregnancies managed per year, and sub-grouped outcomes in relation to calendar period of birth.

**RESEARCH DESIGN AND METHODS**

This prospective study was based on information from the Swedish Medical Birth Registry (MBR) between 1991 and 2003. MBR is regularly evaluated by the Swedish National Board of Health and in the latest evaluation, it was found to capture > 98% of all pregnancies in Sweden. The quality of data has also been found reliable (10). The study included all T1DM pregnancies and pregnant women without a diagnosis of T1DM, served as controls. In both groups, only singletons were included.

We used the International Classification of Diseases (ICD), version 9 and 10. Since 1991, pregestational diabetes can be separated from gestational diabetes (GDM) by ICD 9 and from 1997 diabetes type 1, type 2 (T2DM) and GDM can be separated from each other by ICD 10. The Swedish rate of T2DM in pregnancy is ≤ 5% (MBR, personal communication). Therefore, the overall contribution from any T2DM woman included in the diabetic population during the first six years of the study period was considered small and insignificant.

In Sweden, pregnancy care is standardized and free of charge. Virtually all
pregnant women comply with 7-9 antenatal visits, 1-2 to a physician and the remainders to a midwife. In diabetic pregnancies, the number of antenatal visits is increased. 97% of all pregnant women have an ultrasound examination at 17-18 postmenstrual weeks to determine gestational age.

At the first antenatal visit the woman was interviewed about her medical history, previous births, and complications during previous pregnancy, delivery, and neonatal period. Anthropometric data and smoking habits were recorded. Pregnancy and delivery data were prospectively entered into the MBR which also included neonatal anthropometry and diagnoses. For this purpose, standardised individual obstetric and pediatric forms were used. Maternal characteristics were age, parity, pre-pregnancy weight, height, smoking habits, and whether the mother was born in the Nordic countries (Sweden, Finland, Denmark, Norway or Iceland) or elsewhere. These variables, except ethnicity, were all collected from the MBR. Country of birth was established by linking the MBR to the Civil Registration. Maternal pre-pregnancy body mass index (BMI) was calculated (kg/m²). We defined smoking categories as no smoking, smoking <10 cigarettes per day, or ≥10 cigarettes per day. Maternal complications during pregnancy and delivery, as well as fetal and neonatal complications were classified according ICD 9 and 10. All diagnoses were made by a physician at the time of hospital discharge and copies of obstetric and pediatric records were forwarded to the MBR. Chronic hypertensive disease (ICD-9: 401 or 642 A, ICD-10: I10 or O10.0) was defined as blood pressure (BP) ≥140/90 mmHg diagnosed before pregnancy or before 20 weeks of gestation. Pregnancy induced hypertension (PIH) was defined as resting BP ≥140/90 mm Hg in the second half of pregnancy (ICD -9: 642 D3, ICD -10: 013). Preeclampsia (PE) was defined as PIH and proteinuria (≥0.3 g/day or ≥1 + on a urine dipstick), (ICD-9:642 E and 642 F, ICD-10: O14.0, O14.1 and O15). Mild PE was defined as diastolic BP from 90 to 109 mm Hg combined with proteinuria of <5g/day or 1 to 2 + on a urine dipstick (ICD 9 code 642E and ICD 10 code O14.0). Severe PE was defined as preeclampsia with either a diastolic BP ≥110 mmHg or proteinuria ≥5 g/day, or both (ICD 9 code 642 F ICD 10 codes O14.1 or O15). Preterm birth was defined as delivery before 37 gestational weeks, and very preterm birth as delivery before 32 weeks. During the study period, stillbirth was in Sweden defined as intrauterine death occurring after 28 weeks of gestation.

Perinatal death was defined as the combined rate of stillbirth and mortality within the first week of life. Neonatal mortality was defined as death within the first 7 days of life (early neonatal death) and within the first 28 days of life. Fetal distress was present whenever vacuum extraction or Cesarean section were performed as a result of suspected or manifest fetal hypoxia. Large for gestational age (LGA) and small for gestational age (SGA) were defined as birth weights 2 SD or more above or below the mean for normal fetal growth according to Swedish reference data, respectively (11). The corresponding percentile limits for LGA and SGA are ≥ 97.5 and ≤ 2.5 percentiles; respectively. Apgar scores between 0-3 at 5 min of postnatal age were recorded as well as brachial plexus injury in vaginally delivered infants. Neonatal respiratory disorders were classified according to Hjalmarsen (12). Transient tachypnea, respiratory distress and other diagnoses of respiratory disturbances (ICD 9 codes 769,770G and ICD 10 codes: all codes starting with P 22, P 24, P 25 and P 26) were included in the analysis. A malformation was classified by the MBR as major if it was fatal or potentially life threatening or if it was likely to lead to serious handicap or major cosmetic defect if not surgically corrected. We compared the outcomes between hospitals
managing less than ten (total number of patients = 2,190), ten to nineteen (n = 1,910) and more than nineteen (n = 989) T1DM pregnancies per year. We also categorized the study period into: the first seven years and (1991-97) and the next six years (1998-2003).

Statistical methods. Means and standard deviations were calculated. Student’s t-test and chi-square test were used for comparison of group means and proportions. Logistic regression was used to evaluate any association between maternal T1DM and outcomes. In multivariate analyses, the point estimates for outcomes were adjusted for maternal age, BMI, parity, chronic hypertensive disorder, smoking habits, and ethnicity. Due to missing data on pre-pregnancy BMI, multivariate analyses were limited to 954,292 mothers. All statistical analyses were performed using SPSS version 15.0.

RESULTS

In total 5,089 pregnant women with and 1,260,207 pregnant women without T1DM were included.

Maternal characteristics - women with T1DM suffered more often from pre-pregnancy hypertension, had more often a Nordic origin, as well as a higher BMI compared to the general obstetric population. Maternal age, parity and smoking habits showed minor but statistically significant differences between women with diabetes and controls, Table 1.

Pregnancy complications - The risks for PIH and PE, fetal distress and fetal loss, instrumental delivery and Cesarean section were all increased in the diabetes cohort, also after adjusting for potential confounders, Table 2 and 3. In the diabetic cohort, the majority of stillbirths (58/69) occurred between 34 and 40 gestational weeks. In vaginal deliveries, shoulder dystocia occurred in 13.7% of infants delivered by diabetic mothers as compared to 0.2% in control infants (p < 0.001) which corresponds to an adjusted OR of 11.08 (8.22-14.93).

Fetal growth - Infants of diabetic mothers were born at a lower gestational age (mean 267 versus 278 days, p < 0.001) than control infants. In spite of this, birth weights (3684 gr versus 3551 gr, p < 0.001) were higher in infants of diabetic mothers. Birth weights were close to normally distributed in the diabetes cohort with only a minor difference between the mean and median birth weight. After correction for gestational age and gender, birth weights were much increased in the diabetes group: 31% of the infants to T1DM mothers were LGA as compared to 3.6 % in the population. Birth weights equal to or above 4.5 and 5 kg, respectively, were significantly more common in the diabetes group (12.6% and 2.7 %,) than among control infants (3.9% and 0.5%, p< 0.0001). The adjusted OR for SGA was significantly lower in the T1DM group compared to controls, Table 3.

Malformations - In the diabetes group there was a two fold increase in the incidence of major malformations, Table 3. Major malformations were the leading cause of neonatal death (10/36) in the diabetic cohort.

Neonatal mortality and morbidity - Perinatal and neonatal mortalities were significantly increased in diabetic pregnancies. Low Apgar scores (0-3) at 5 minutes of postnatal age, Erb’s palsy and respiratory distress were all significantly and much more common in infants of diabetic mothers, Table 3.

Outcomes in relation to hospital size - Fetal distress and transient tachypnea were more common in larger hospitals, caring for more than nineteen T1DM pregnancies per year. Except from these two outcomes, there were no significant differences in obstetric and perinatal outcome between hospitals of different size.

Outcomes in relation to calendar period of birth - The incidence of LGA
increased in the diabetic group from 27.6% in the first to 35.0% in the second period of the study (p<0.001). The proportion of LGA infants increased significantly also in the general obstetric population, from 3.38% in the first to 3.77% in the last study period (p<0.001).

Over time, the proportion of women with a BMI \( \geq 30 \) increased in both groups. In the first study period, 13.2% of T1DM women were obese as compared to 18.4% during the second period (p<0.001). The corresponding figures for control women were 7.3% (period one) and 11.3% (period two).

**DISCUSSION**

This study showed that pregnant T1DM women still suffer from markedly elevated incidences of obstetric and fetal complications such as PE, prematurity, malformations, perinatal mortality, and neonatal morbidity. The risk for a LGA infant was particularly increased among women with diabetes.

The strength of the present study is the population based cohort, including both still-born and live-born infants. Population based data are essential for assessing solid estimates of complication rates, for planning of health care and for patient counselling, as well as for comparisons between countries and populations. The large numbers of patients allowed for detailed risk assessments, also as regards rare complications such as perinatal mortality and malformations. Potential confounding by other risk factors – such as age, parity, BMI, and chronic hypertensive disease – could be controlled for.

We cannot exclude that during the first study period some T2DM patients could have been included in the study population since the ICD-9 codes just differ between pregestational and gestational diabetes. However the rate of T2DM in pregnancy is low in Sweden. If anything, the small fraction of T2DM that might have been included most likely contributed to a dilution of the complication rates observed in T1DM. Accordingly, this potential misclassification bias does not invalidate our findings.

One limitation is that the Medical Birth Registry does not contain data on duration of diabetes, prevalence of pre-existing microangiopathy, or glycemic control during pregnancy. Another limitation is the lack of data regarding the number of induced abortions due to malformations. The present analysis confirms the elevated risks for PE and PIH in women with T1DM. This is in accordance with findings in other comparable studies (4, 13). The precise explanation for this finding is not known. However, chronic hypertensive disease, nephropathy and poor glycemic control are likely contributing factors (14-16).

Stillbirths in diabetic pregnancies (1.5%) were five times that of the background population. Although markedly reduced compared to data from forty years ago (17), the over-risk for stillbirth in diabetic pregnancies is in agreement with contemporary studies (4, 13, 18). The underlying mechanisms for stillbirth in diabetic pregnancies are not fully understood. Maternal and fetal hyperglycemia are associated with chronic fetal hypoxia, as indicated by a correlation between the concentration of erythropoetin in amniotic fluid and antenatal glycemic control (19). Lauenborg et al reported 25 cases of stillbirth in T1DM pregnancies, and found no other cause of death than poor maternal glycemic control in nine cases (20). Similarly, Hanson et al recorded significantly higher HbA1C levels in the last trimester in 5/10 diabetic stillbirths (1). Fetal hypoxia may also explain why fetal distress as an indication for instrumental delivery or Cesarean section was nearly three times more common in diabetic pregnancies compared to controls.

Major malformations occurred 2-3 times more often in the diabetes group. This is
comparable to some (4, 6) but not all reports -
higher rates of major malformations in
comparison to controls have been reported
from the UK (2). Differences in metabolic
control (21), in definitions of major
malformation, in the number of early induced
abortions, and in intrapartum deaths may
contribute to this discrepancy. The
malformation rate should also be interpreted
in view of the fact that in Sweden, there is no
food-fortification with folic acid. Pre-
pregnancy counselling includes the
recommendation of folic acid
supplementation to women with T1DM but
we have no data regarding compliance with
this recommendation or folic acid intake
before conception.

Rates of instrumental delivery (9.6%)
and Cesarean section (46%) were much
higher in the diabetes group. This is in
agreement with other reports (4). Studies on
T1DM pregnancies from Finland and
Denmark report even higher rates of C-
sections; 63.5% and 55.9%, respectively (5,
13). The lower C-section rate in Sweden could
partly be explained by a more expectative
policy, where spontaneous initiation of
delivery is encouraged. This suggestion is
supported by higher numbers of preterm
deliveries reported from Finland and
Denmark – 30% and 42%, respectively (5, 13)
versus 22 % in our study. Many of the
preterm deliveries in other studies were
performed before start or after induction of
labour (13). Perinatal mortality in T1DM
pregnancies in Sweden has decreased from
3.1% in 1982-85 (1) to 2.0% in the present
study, and it is in the lower range of
previously recorded rates, varying from 1.3%
to 6.6% (4-6,13, 18, 22). Nevertheless,
perinatal mortality in T1DM exceeds that in
controls by four times, mainly because of the
over-risk for stillbirth. To a lesser extent,
neonatal mortality rates were also increased in
infants of diabetic women, most likely
because of their higher incidence of
malformations, preterm delivery and birth
asphyxia. An unexpected observation was the
markedly increased incidence of fetal
macrosomia in diabetic pregnancies. The
incidence of LGA (31%) was more than 8
times that of the control group. In comparison
with Swedish data from 1982-85, the
incidence of LGA has increased with 11% in
absolute values, and relatively around 50 %
(1). The risk for LGA increased significantly
over time in the present study. High rates of
macrosomia have also been reported from
Holland (28.4%) and Finland (34.7%) (4, 5).
It is unlikely that the increased LGA-
incidence is a consequence of deterioration in
metabolic control, as both the stillbirth and
major malformation rates have declined. One
could speculate that a contributing factor to
the increasing incidence of LGA is a
decreasing rate of microangiopathy (23).
Interactions with other determinants of birth
weight – such as pre-pregnancy BMI – may
also contribute to this finding. However, the
over-risk for an LGA infant remained high
[OR=11, 45] even after adjusting for maternal
BMI.

Very preterm delivery was three times
that of the controls, contributing to the higher
incidence of neonatal morbidity such as RDS
in the offspring of diabetic mothers. We also
found an 8 times higher incidence of Erb’s
palsy in the diabetic offspring than in the
healthy infant. It is well recognized that
infants of mothers with diabetes have a
smaller head to shoulder ratio. The
disproportionate body constitution increases
the propensity of shoulder dystocia and Erb’s
palsy and the risk of these complications also
increases with larger size of the baby. There is
no clear explanation for the variation in
obstetric and perinatal outcomes of type 1
diabetic pregnancies between different
countries. The discrepancy in reported rates of
complications could partly be a result of
differences in the organisation of health care,
socioeconomic factors and patient
compliance. In Sweden, antenatal care of pregnant women with diabetes is usually carried out in hospitals. The population is scattered over a large geographical area which precludes centralisation of care to regional centres. Almost half of the pregnant women with diabetes attended small clinics, caring for less than 10 T1DM pregnancies per year. The care is generally provided by multidisciplinary teams with special interest in diabetes. All patients are given insulin and equipment for insulin administration, as well as equipment for home monitoring of blood glucose. The importance of pre-pregnancy planning is emphasised, starting already at the pediatric clinic. National recommendations concerning standardisation of care are available. Before and during the study period, diabetologists have provided health care personnel with information and guidelines for medical management of pregnant diabetic women. This work seems to have been successful as major outcomes did not vary in relation to hospital size. Similar experience has been reported from Finland where decentralization of care for diabetic patients did not significantly increase the number of adverse pregnancy outcomes (5). However, we cannot exclude that the higher incidence of fetal distress and transient tachypnea that was observed in larger hospitals caring for >19 T1DM pregnancies a year reflect a selection with the most complicated pregnancies transferred to the larger hospitals. Given such selection, smaller hospitals would be expected to have better results. The number of preterm births and cases of severe PE did not differ between large and smaller clinics, but without a more detailed analysis of the case-mix of diabetic women in different hospitals, the issue of centralisation or not has to be left open. In conclusion, the present study demonstrates that incidences of obstetric and perinatal complications are still high in the diabetic pregnancy. An intriguing finding is the high incidence of fetal macrosomia and that the incidence is increasing over time. The etiology behind the increased risk of fetal death and fetal macrosomia in the T1DM pregnancy are not fully understood and warrants further investigation.

Acknowledgements

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References


### Table 1. Maternal characteristics in type 1 diabetes and the general obstetric population in Sweden 1991-2003

<table>
<thead>
<tr>
<th></th>
<th>Type 1 diabetes</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>N= 5,089</td>
<td>N= 1,260,207</td>
</tr>
<tr>
<td>Nordic origin (%)</td>
<td>92.6</td>
<td>86.8</td>
</tr>
<tr>
<td>Maternal age (years)</td>
<td>29.6 ± 5.1</td>
<td>29.0 ± 5.1</td>
</tr>
<tr>
<td>Primapara (%)</td>
<td>44.5</td>
<td>42.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>166 ± 6.3</td>
<td>166 ± 6.2</td>
</tr>
<tr>
<td>Prepregnancy weight (kg)</td>
<td>71.4 ± 13.4</td>
<td>66.5 ± 12.1</td>
</tr>
<tr>
<td>Prepregnancy BMI (kg/m2)</td>
<td>25.9 ± 4.6</td>
<td>24.0 ± 4.1</td>
</tr>
<tr>
<td>Chronic hypertensive disease (%)</td>
<td>2.1</td>
<td>0.24</td>
</tr>
<tr>
<td>No smoking in pregnancy (%)</td>
<td>82.0</td>
<td>83.8</td>
</tr>
<tr>
<td>Smoking - &lt; 10 cig/day(%)</td>
<td>10.9</td>
<td>10.6</td>
</tr>
<tr>
<td>Smoking - ≥ 10 cig/day(%)</td>
<td>7.1</td>
<td>5.6</td>
</tr>
</tbody>
</table>

Data are mean ± SD values or proportions in %. With the exception of maternal height, all differences between type 1 diabetics and controls are statistically significant with p-values < 0.001 (Students t-test or chi square test).
Table 2. Pregnancy complications and mode of delivery in type 1 diabetic pregnancies (n=5,089) and controls (n=1,260,207)

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>Type 1 diabetes</th>
<th>Non-diabetes</th>
<th>Crude (95% CI)</th>
<th>Adjusted (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PIH</td>
<td>1.6</td>
<td>0.87</td>
<td>1.93 (1.50 - 2.49)</td>
<td>1.53 (1.18 - 1.99)</td>
</tr>
<tr>
<td>Preeclampsia, mild</td>
<td>9.7</td>
<td>2.0</td>
<td>5.37 (4.81 - 6.00)</td>
<td>4.30 (3.83 - 4.83)</td>
</tr>
<tr>
<td>Preeclampsia, severe</td>
<td>4.3</td>
<td>0.8</td>
<td>5.58 (4.75 - 6.57)</td>
<td>4.47 (3.77 - 5.31)</td>
</tr>
<tr>
<td>Cesarean section</td>
<td>46</td>
<td>12</td>
<td>5.85 (5.49 - 6.25)</td>
<td>5.31 (4.97 - 5.69)</td>
</tr>
<tr>
<td>VE/ forceps</td>
<td>9.6</td>
<td>6.6</td>
<td>1.48 (1.33 - 1.66)</td>
<td>1.41 (1.25 - 1.58)</td>
</tr>
</tbody>
</table>

OR = odds ratio, CI = confidence interval, adjusted OR = OR adjusted for group differences in maternal age, body mass index, parity, chronic hypertensive disorder, smoking habits, and ethnicity. PIH = pregnancy induced hypertension, VE = vacuum extraction.

Table 3. Fetal and neonatal complications in type 1 diabetic pregnancies and the general obstetric population

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>Type 1 DM</th>
<th>Controls</th>
<th>Crude (95% CI)</th>
<th>Adjusted (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stillbirth</td>
<td>1.5</td>
<td>0.3</td>
<td>4.04 (3.02-5.40)</td>
<td>3.34 (2.46-4.55)</td>
</tr>
<tr>
<td>Fetal distress</td>
<td>14</td>
<td>6.2</td>
<td>2.45 (2.24-2.69)</td>
<td>2.34 (2.12-2.58)</td>
</tr>
<tr>
<td>Perinatal mortality, %</td>
<td>20</td>
<td>4.8</td>
<td>4.02 (3.11-5.20)</td>
<td>3.29 (2.50-4.33)</td>
</tr>
<tr>
<td>Neonatal mort., 0–7 d, %</td>
<td>5.1</td>
<td>1.8</td>
<td>2.91 (1.97-4.28)</td>
<td>3.05 (1.68-5.55)</td>
</tr>
<tr>
<td>Neonatal mort., 0-28 d, %</td>
<td>7.0</td>
<td>2.2</td>
<td>3.08 (2.02-4.70)</td>
<td>2.67 (1.72-4.16)</td>
</tr>
<tr>
<td>Birth &lt; 37 weeks GA</td>
<td>21</td>
<td>5.1</td>
<td>5.27 (4.88-5.71)</td>
<td>4.86 (4.47-5.28)</td>
</tr>
<tr>
<td>Birth &lt; 32 weeks GA</td>
<td>2.3</td>
<td>0.7</td>
<td>3.58 (2.89-4.44)</td>
<td>3.08 (2.45-3.87)</td>
</tr>
<tr>
<td>LGA</td>
<td>31</td>
<td>3.6</td>
<td>12.2 (11.4-13.1)</td>
<td>11.4 (10.6-12.4)</td>
</tr>
<tr>
<td>SGA</td>
<td>2.3</td>
<td>2.5</td>
<td>0.80 (0.63-1.02)</td>
<td>0.71 (0.55-0.91)</td>
</tr>
<tr>
<td>Major malformations</td>
<td>4.7</td>
<td>1.8</td>
<td>2.70 (2.37-3.08)</td>
<td>2.50 (2.13-2.94)</td>
</tr>
<tr>
<td>Apgar &lt; 7 at 5 min</td>
<td>3.1</td>
<td>1.1</td>
<td>2.98 (2.54-3.50)</td>
<td>2.60 (2.14-3.17)</td>
</tr>
<tr>
<td>Apgar &lt; 4 at 5 min</td>
<td>0.80</td>
<td>0.30</td>
<td>2.60 (1.79-3.78)</td>
<td>2.39 (1.64-3.51)</td>
</tr>
<tr>
<td>Erb’s palsy*</td>
<td>2.1</td>
<td>0.25</td>
<td>7.91 (5.77-10.8)</td>
<td>6.69 (4.81-9.31)</td>
</tr>
<tr>
<td>RDS</td>
<td>1.0</td>
<td>0.20</td>
<td>4.88 (3.51-6.81)</td>
<td>4.65 (2.20-9.84)</td>
</tr>
<tr>
<td>Respiratory disorders</td>
<td>9.5</td>
<td>2.6</td>
<td>4.02 (3.67-4.42)</td>
<td>3.42 (3.04-3.85)</td>
</tr>
</tbody>
</table>

OR = odds ratio, adjusted OR = OR adjusted for group differences in maternal age, body mass index, parity, chronic hypertensive disorder, smoking habits, and ethnicity. LGA = large for gestational age, SGA = small for gestational age, RDS = respiratory distress syndrome. *= vaginal deliveries only. GA= gestational age.