

Influence of maternal, obstetric and fetal risk factors on the prevalence of birth asphyxia at term in a Swedish urban population

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Background. The influence of maternal, obstetric and fetal risk factors on the prevalence of birth asphyxia at term in a Swedish urban population.

Objective. To investigate risk factors for Apgar score-defined birth asphyxia, birth asphyxia with hypoxic-ischemic encephalopathy and birth asphyxia-related death/disability.

Material and methods. Retrospective case-control study in term neonates with birth asphyxia defined as Apgar score <7 at 5 min. Cases originating from nonasphyctic causes (e.g. infection, maternal sedation) were excluded. Hypoxic-ischemic encephalopathy was diagnosed according to criteria by Sarnat. Maternal, obstetric and fetal risk factors were registered in 225 cases of birth asphyxia diagnosed in 42203 live births occurring in the urban Swedish population studied. A matched control group was used for statistical evaluation.

Results. Asphyxia was associated with single civil status, OR = 7.1 (95%CI 2.0, 27.6); intrauterine meconium release, OR = 4.1 (95%CI 1.8, 9.8); operative delivery, OR = 8.7 (95%CI 3.4, 24.6); breech delivery, OR = 20.3 (95%CI 3.0, 416.5); oxytocin augmentation, OR = 2.9 (95%CI 1.4, 6.3); cord complication, OR = 15.8 (95%CI 2.1, 341.5); external compression to assist delivery OR = 6.2 (95%CI 1.3, 45.7); and cardiotocography score, OR = 0.5 (95%CI 0.4, 0.6). Normal fetal heart rate variability, OR = 0.4 (95%CI 0.2, 0.6), repeated late decelerations irrespective of amplitude or repeated variable decelerations, OR = 29.4 (95%CI 5.7, 540.8) or occasional late or variable decelerations, OR = 2.2 (95%CI 1.3, 3.8), and no accelerations, OR = 5.2 (95%CI 2.0, 16.4), were associated with asphyxia. Operative or instrumental delivery was more common in all three asphyxia groups compared with controls. Leanness was a risk factor for asphyxia and for hypoxic-ischemic encephalopathy. Maternal age, smoking and illnesses, time of delivery (day/night, seasonal) and previous caesarean section were not associated with birth asphyxia.

Conclusions. An association between neonatal asphyxia and cardiotocography parameters, intrauterine meconium release, operative delivery, breech delivery, single civil status, oxytocin augmentation, cord complication, external compression to assist delivery and neonatal leanness was found. Abnormal fetal heart rate variability, repeated late decelerations irrespective of amplitude or repeated variable decelerations, occasional late or variable decelerations and no accelerations were associated with asphyxia.

Keywords: asphyxia, epidemiology, hypoxic-ischemic encephalopathy, newborn, risk factors

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Perinatal mortality, morbidity and long-term sequelae have not only been related to maternal risk factors such as age, maternal illness, smoking and socioeconomic status (1,2) but also to obstetric risk factors, i.e. factors related to the management

of labor and fetal abnormalities such as malformations and intrauterine growth retardation (3). Birth asphyxia is a perinatal event, in serious cases leading to a dismal outcome with risk of death or permanent sequelae. Its incidence may be influ-

enced by the same risk factors as detailed earlier or by unexpected catastrophic events, as well as by a combination of the two (4–9).

The aim of this retrospective case-control study was to investigate the incidence and importance of potential maternal, obstetrical and fetal risk factors, as well as fetal heart rate (FHR) changes in term-asphyxiated newborns from an urban Swedish population (10). Asphyxia was defined by a low Apgar score and according to the development of hypoxic-ischemic encephalopathy (HIE) and asphyxia-related death/damage (disability), which were ascertained on clinical grounds after excluding other possible causes.

Materials and methods

This material originates from the population of the city of Göteborg, the second largest city in Sweden (population 430 000) with a birth rate of approximately 6000/year, and comprising 5% of all infants born in Sweden. Three delivery units at the hospitals Sahlgrenska, Östra and Mölndal serve the city. The two former hospitals are teaching hospitals and Mölndal Hospital is a community hospital. There were no planned home deliveries during the period. The study was approved by the Ethics Committee, Faculty of Medicine, Göteborg University.

All births in Sweden are recorded in the Swedish Medical Birth Register (MBR), which was the main source of our material. The register is based on standardized medical records for maternal, obstetric and neonatal healthcare from all deliveries in Sweden, and includes the hospital code and community affiliation of the mother. Deliveries are also registered in the Swedish Vital Statistics Register, based on birth certificates.

Population and inclusion criteria

During the 7-year period from 1985 to 1991, 42 203 live infants were born to mothers residing in the city of Göteborg. Infants born during this period with a gestational age (GA) of 37 weeks, and Apgar scores <7 at 5 min were identified via the MBR. Gestational age was estimated from the last menstrual period, and in the majority of cases also by ultrasound at 15–16 weeks. To increase the accuracy of the material, data obtained from the MBR were cross-checked and supplemented by hospital diagnosis registers and the Swedish Vital Statistics Register.

A total of 292 infants with a 5-min Apgar score <7 were identified. In order to estimate the incidence of 'pure' birth asphyxia based on a definition of an Apgar score <7, infants considered

to have a low Apgar score for reasons other than asphyxia were excluded: opioid/anesthesia-related low Apgar score ($n=29$), congenital malformations and chromosomal disorders ($n=20$), congenital infections ($n=13$), congenital neuromuscular disorders ($n=2$) and subarachnoid hemorrhage ($n=1$).

Asphyxia group. Of the remaining infants ($n=227$), 136 were boys and 91 were girls (60%/40%). This group of infants, referred to as the 'asphyxia group', formed the basis of our study. The clinical course and neurological outcome of this material has been reported in a previous paper (10). The 'control group' was chosen as follows: the next infant born on the same delivery unit, of the same gender, GA = 37 weeks and with Apgar score = 7 at 5 min. If this infant did not fulfil the criteria for the control group, the infant born immediately before the asphyxiated infant was chosen, etc. Two infants in the asphyxia group were excluded: one was born outside the city of Göteborg and a control case was not obtainable. There was one case of duplex with asphyxia in both and for these two infants a single control was selected. Accordingly, one of the twins was excluded, leaving 225 infants in the asphyxia group.

Hypoxic-ischemic encephalopathy group. A retrospective HIE classification was performed, according to notes in the medical records, and the infants were classified as having none, mild, moderate or severe HIE according to the criteria of Sarnat and Sarnat (11), modified by Fenichel (12). Mild HIE is characterized by hyper-alertness and irritability, normal muscle tone, normal or hyperactive reflexes, ankle clonus and no seizures. Moderate HIE includes lethargy, decreased spontaneous movements, proximal muscular weakness, depressed primitive reflexes and seizures. Severe HIE includes stupor or coma, markedly reduced muscle tone or flaccidity and absent primitive reflexes. Seizures are often frequent and may be difficult to control, but may also be totally absent. No infant with encephalopathy was found among the control cases. Infants with HIE are referred to as the 'asphyxia-HIE group' ($n=75$), and was a subgroup of the 'asphyxia group'. Medical records of surviving infants were followed ≥ 18 months. Infants who were considered to be neurologically disabled at 18 months were followed for a further 1.5–3 years to obtain a full perspective of their disability (10).

Asphyxia-death/disability group. Infants who died or developed a neurological disability related to asphyxia are referred to as the 'asphyxia-death/disability group' ($n=22$). This was also a subgroup of the 'asphyxia group'.

Relevant maternal/obstetrical and fetal/neonatal

data were obtained from hospital records for all the index and control cases included ($n = 450$). Fetal heart activity during delivery had been recorded by external or internal cardiotocography and fetal heart rate recordings were available in the majority of included cases (comparisons were possible in 131 of the 225 matched asphyxia pairs; 41 of the 75 asphyxia-HIE pairs and 15 of the 22 asphyxia-death/disability pairs). The fetal heart rate recordings were retrospectively evaluated by two obstetricians using a standardized scoring system (Table I), which is a modification of a scoring system described previously by Käär (13). The obstetricians were not aware of the fetal outcome when the recordings were analyzed. Standard deviation scores (SDS) for birth weight (BWSDS) and birth length (BLSDS; 14) were used to evaluate weight in relation to length normalized for sex and gestational age in the newborns. The leanness score (LeanSDS) (15), which is independent of gender and gestational age, was calculated from BWSDS and BLSDS, and is a measure of dysmaturity (leanness) or fatness in the newborn infant. The infants in this study were compared with a reference population based on 100 000 term infants (15).

Statistical analysis

Univariate logistic regression was used for analysis of risk factors, and a stepwise logistic regression analysis was used to suggest the predictor variables, which consisted of apparently independent and significant predictors of asphyxia. Logit estimators used a correction of 0.5 in every cell of tables that contained a zero. Fisher's exact test was used to test differences when comparing proportions, and Wilcoxon's rank-sum test was used for testing differences between means ($p < 0.05$ was considered to be statistically significant). SAS statistical software version 6.12 (SAS Institute Inc., Cary, NC, USA) was used for the analysis.

Results

During the 7-year period 1985–91, the incidences of low Apgar score (< 7 at 5 min), pure birth asphyxia and birth asphyxia with HIE in this population were 6.9, 5.4 and 1.8 per 1000 live born infants, respectively (10). The asphyxia-related neonatal mortality was 0.26/1000 live births, i.e. 13 infants died in the neonatal period and 10 infants (0.2/1000) had a neurological impairment at the follow up performed at 18 months of age. One infant in this group had no HIE and developed a mild hemiplegia not considered to be of perinatal origin.

Maternal factors

Asphyxia group. Mothers of asphyctic infants were more often single, of foreign nationality and primigravida (Table II). Maternal age, smoking habits, time of delivery (day/night, seasonal) and previous pregnancy history (e.g. history of abortions, previous caesarean and extrauterine pregnancy) were not related to an increased risk of birth asphyxia. Nor was there any over-representation of concomitant maternal illnesses in the asphyxia group compared with the control group (*asphyxia group*: hypothyreosis 4, viral hepatitis 4, diabetes mellitus 3, asthma 3, hyperthyreosis 1, inflammatory bowel disorder 1, hypertension 1, sarcoidosis 1, anxiety disorder 1; *control group*: viral hepatitis 1, asthma 1, inflammatory bowel disorder 2, hypertonia 2, sarcoidosis 1, depression 1, collagenosis 1, psoriasis 1, infectious disorder 2, cardiomyopathy 1, and seizure disorder 1). However, in this respect it should be noted that there were four cases of hypothyreosis and four cases of diabetes mellitus (three manifest and one gestational diabetes) in the asphyxia group compared with none in the control group.

Table I. Scoring system (modified after Käär; 13) used for the evaluation of fetal heart rate

Variable	Score			
	0	1	2	3
Baseline fetal heart rate (b.p.m.)	< 119 > 160	120–160	–	–
Fetal heart rate Variability (b.p.m.)	< 5 –	5–9 > 30	10–30 –	– –
Accelerations (a)	0	a > 15 b.p.m.	5a > 15 b.p.m. for > 15 s; 1a > 20 b.p.m. for > 15 s	–
Decelerations (d)	Repeated late d irrespective; amplitude; repeated variable d	Occasional late or variable d	Non-characteristic or suspect d	None

Table II. Analyses of the association between potential antenatal risk factors for asphyxia

	Asphyxia group <i>n</i> = 225 <i>n</i> (%)	Control group <i>n</i> = 225 <i>n</i> (%)	Asphyxia vs. Control OR (95%CI)	<i>p</i> -value
Civil status: single	32 (14.4)	13 (5.8)	2.7 (1.4, 5.3)	0.003
Foreign nationality	28 (12.5)	13 (5.8)	2.3 (1.2, 4.6)	0.02
Primigravida	154 (68.0)	100 (44.4)	2.7 (1.8, 3.9)	< 0.001
Normal pregnancy	201 (89.3)	219 (97.8)	0.2 (0.1, 0.5)	< 0.001

The pregnancy was considered to be abnormal (premature contractions, hemorrhage, preeclampsia, diabetes mellitus, infection or multiple pregnancies) in 24/225 (10.7%) women from the asphyxia group compared with 6/225 (2.2%) in the control group (OR = 5.2, 95%CI: 2.0, 14.0). Hemorrhage was associated with asphyxia in 6/225 compared with 0/225 in the control group ($p = 0.01$). Pregnancy-induced hypertension/preeclampsia and hemorrhage were more often recorded in the asphyxia group 5.3% compared with 1.3% in the control group (OR = 4.2, 95%CI 1.2, 14.9). The incidence of diabetes was too low for a statistical analysis (4/225 in the asphyxia group compared with 0/225).

Hypoxic-ischemic encephalopathy group. Mothers of infants who developed HIE were more often single: 10/75 vs. 3/75 (OR = 3.8, 95%CI 1.0, 14.2). When the HIE group was compared with their controls, 8/75 had complications during pregnancy compared with 3/75 (OR = 2.9, 95%CI 0.7, 11.3).

Obstetric factors

Asphyxia group. Emergency caesarean section was more frequent in the asphyxia group, 86/225 compared with 11/225 in the control group (Table III; OR = 12.0, 95%CI 6.2, 23.4). Vacuum extraction is more often used than forceps in the obstetric units involved and an over-representation of vacuum extraction deliveries was recorded in the asphyxia and asphyxia-HIE groups (Table III). Abruptio placenta, umbilical cord complications (i.e. cord prolapse and compression), oxytocin augmentation, and breech delivery were more frequently recorded in the asphyxia group (Table III). The women with deliveries resulting in asphyctic infants had more often received an epidural blockade compared with the controls (Table III). Even if only vaginal deliveries were analyzed the association between epidural anesthesia and asphyxia remained (OR = 4.2, 95%CI 2.3, 7.6).

Hypoxic-ischemic encephalopathy group. Emergency caesarean section was more frequent in the

HIE-asphyxia group: 35/75 compared with 2/75 in the control group (OR = 17.5, 95%CI 4.4, 70.2). Elective caesarean section was more commonly encountered in the control group 5/75 compared with the asphyxia group (0/75) without reaching statistical significance ($p = 0.06$). An over-representation of vacuum extraction deliveries was recorded in the asphyxia-HIE group: 16/75 compared with 4/75 in the control group (OR = 4.0, 95%CI 1.4, 11.4). There were three cases of uterine rupture; all these infants had HIE, and one infant is now considered to be normal while two have severe neurological damage. Two of these women had a previous caesarean section and one woman had a previous vaginal delivery.

Oxytocin augmentation was more common in the asphyxia-HIE group (38/75) compared with the control group (23/75), OR = 2.5, 95%CI 1.3, 4.9. Umbilical cord complications (i.e. cord prolapse and compression) were more commonly encountered in the asphyxia-HIE group: 7/75 compared with 1/75 in the control group (OR = 9.6, 95%CI 1.2, 80.7). Abruptio placenta was more common in the HIE-asphyxia group, 8/75 vs. 0/75, without reaching statistical significance (OR = 13.0, 95%CI 0.8, 226.7).

Perinatal factors

Asphyxia group. A comparison of the FHR scores between the control and the asphyxia group appears in Table IV. Marked differences were recorded between the asphyxia group and the control group, regarding the FHR parameters analyzed. A stepwise logistic regression analysis was performed on all the variables in Table IV to analyze the factors monitored by cardiotocography (CTG) that were independently associated with asphyxia. Asphyxia was associated with a normal fetal heart rate variability OR of 0.4 (95%CI 0.2, 0.6), a deceleration (repeated late decelerations irrespective of amplitude or repeated variable decelerations) OR of 29.4 (95%CI 5.7, 540.8), an occasional late or variable deceleration OR of 2.2 (95%CI 1.3, 3.8) and a no acceleration OR of 5.2, 95%CI 2.0, 16.4).

Table III. Analyses of the association between potential intrapartur risk factors for asphyxia

	Asfyxia group		Control group		Asfyxia vs. Control OR (95%CI)	p-value
	n = 225 n	(%)	n = 225 n	(%)		
Vaginal delivery	127	(56.4)	20	(8.9)	0.2 (0.1, 0.3)	< 0.001
Elective CS	6	(2.7)	14	(6.2)	0.4 (0.2, 1.07)	0.107
Acute CS	55	(24.4)	10	(4.4)	7.0 (3.4, 14.1)	< 0.001
Extremely acute CS	31	(13.8)	1	(0.4)	35.7 (4.8, 264.6)	< 0.001
Vacuum extraction	41	(18.2)	16	(7.1)	2.9 (1.6, 5.4)	< 0.001
External compression	24	(10.7)	3	(1.3)	8.8 (2.6, 29.7)	< 0.001
Abruptio placenta	11	(4.9)	1	(0.4)	11.5 (2.2, 59.3)	0.006
Cord complication	25	(11.1)	3	(1.4)	10.8 (3.2, 36.2)	< 0.001
Oxytocin augmentation	127	(58.5)	64	(29.5)	3.4 (2.3, 5.0)	< 0.001
Intrauterine meconium release	105	(47.5)	29	(13.1)	6.0 (3.8, 9.7)	< 0.001
Vertex presentation	199	(88.4)	21	(9.4)	0.3 (0.1, 0.6)	0.002
Breech presentation	21	(9.3)	2	(0.9)	11.5 (2.7, 49.6)	< 0.001
No analgesia	70	(31.1)	60	(26.7)	1.2 (0.9, 1.6)	0.349
Epidural blockade	56	(25.8)	25	(11.4)	2.3 (1.5, 3.5)	< 0.001
Paracervical blockade	33	(15.2)	28	(12.8)	1.2 (0.8, 1.9)	0.492
Nitrous oxide	136	(62.7)	154	(70.3)	0.9 (0.8, 1.0)	0.104

CS, caesarean section.

Table IV. A comparison of an evaluation of the intrapartur fetal heart rate trace

	Asfyxia group		Control group		Sensitivity	Specificity	Pos pred value	Neg pred value
	n = 179 n	(%)	n = 153 n	(%)				
Abnormal basal fetal heart rate (< 120 or > 160 b.p.m)	49	(27.4)	10	(6.5)	27%	93%	83	52
Normal fetal heart rate variability (10–30 b.p.m.)	63	(35.3)	118	(77.1)	35%	23%	35	23
Fetal heart rate variability (5–9 b.p.m. or > 30 b.p.m.)	82	(45.8)	34	(22.2)	46%	78%	71	55
Fetal heart rate variability (< 5 b.p.m.)	34	(19.0)	1	(0.7)	19%	99%	97	51
Accelerations, score = 2	47	(26.3)	101	(66.0)	26%	34%	32	28
Accelerations, score = 1	73	(40.8)	47	(30.7)	41%	69%	61	50
No accelerations	59	(33.0)	5	(3.3)	33%	97%	92	55
No decelerations	3	(1.7)	17	(11.1)	2%	89%	15	44
Decelerations score = 2	53	(29.6)	86	(56.2)	30%	44%	38	35
Decelerations score = 1	91	(50.8)	49	(32.0)	51%	68%	65	54
Decelerations score = 0	32	(17.9)	1	(0.7)	18%	99%	97	51

There was no difference in placenta weight between the asphyxia group and the control group. Postmaturity, i.e. GA = 42 weeks, occurred in 34/225 of the infants in the asphyxia group compared with 18/225 in the control group (OR = 2.0, 95%CI 1.1, 3.8). There was 17/225 small for gestational age (SGA) infants in the asphyxia group compared with 3/225 in the control group (OR = 5.7, 95%CI 1.7, 19.1). Fourteen of 225 infants in the asphyxia group were large for gestational age (LGA) compared with 10/225 in the control group (OR = 0.7, 95%CI 0.3, 1.6). The infants in the asphyxia group were significantly leaner than the reference population and the control group, as shown in Fig. 1.

Birthweight (in SDS or gram) in the asphyxia group revealed less difference compared with the control group (Fig. 2). There was a lack of information concerning length in six of the infants with death/damage, so we also calculated BWSDS to confirm that this group was not a group of small infants.

Hypoxic-ischemic encephalopathy group. Marked differences were recorded between the HIE group and the control group regarding all the FHR parameters analyzed in the same way as done for the asfyxia-group. Intrauterine meconium release was significantly more common in the HIE group than in the control group: 43/75 vs. 8/75

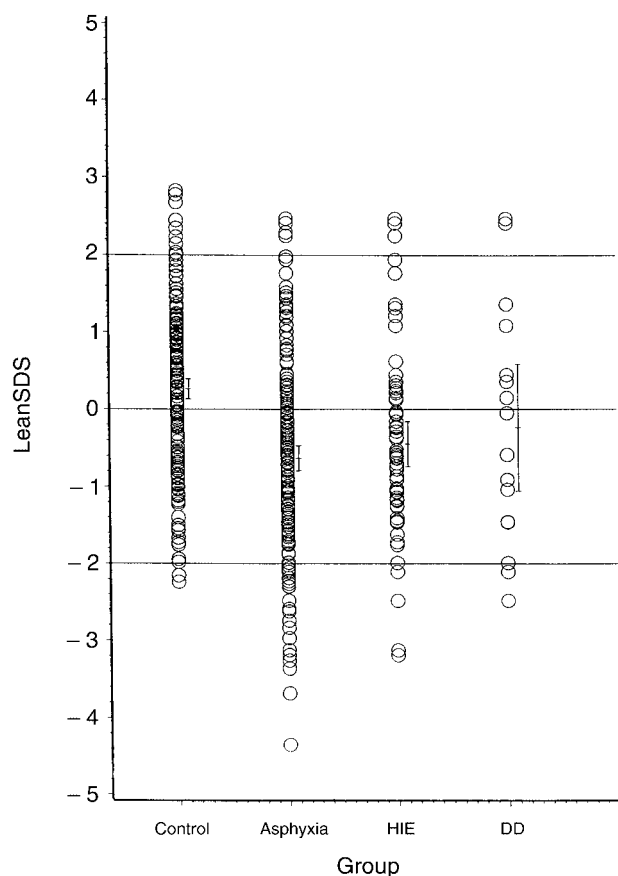


Fig. 1. Comparison (standard deviation scores) for leanness (LeanSDS) between the asphyxia, the hypoxic-ischemic encephalopathy (HIE) and the asphyxia related death/damage (DD) groups and the respective control group, superimposed on a Swedish reference (15) standard of term infants (95%CI around the mean).

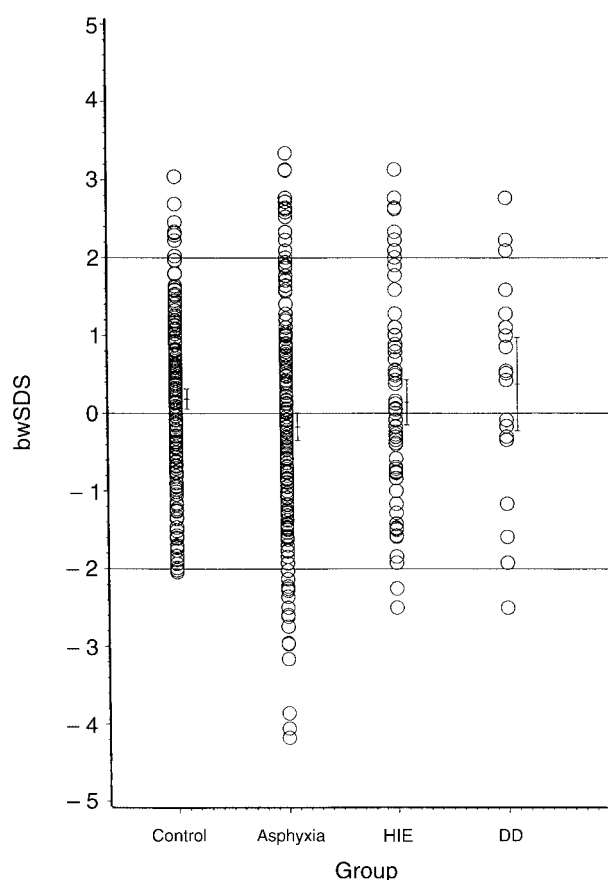


Fig. 2. Comparison (standard deviation scores) for birth weight (BWSDS) between the asphyxia, the hypoxic-ischemic encephalopathy (HIE) and the asphyxia related death/damage (DD) groups and the respective control group, superimposed on a Swedish reference (14) standard of term infants (95%CI around the mean).

(OR = 11.4, 95%CI 5.2, 25.3). In the HIE group 8/75 were postmature compared with 5/75 in the HIE control group ($p=0.6$). In the HIE group only 2/75 infants were SGA compared with 0/75 in the HIE control group. In the HIE group 7/75 were LGA compared with 0/75 in the control group. The asphyxia-HIE group was also significantly different regarding LeanSDS (Fig. 1), which was lower than in their controls, but no differences were found in BWSDS (Fig. 2).

Asphyxia-death/disability-group. The asphyxia-death/disability group comprised 22 neonates; 12/22 had a caesarean section compared with 0/22 in the control group (OR = 53.5, 95%CI 2.9, 993.4). Cord complication was found in 4/22 in the asphyxia-death/disability group compared with 0 in the control group, and uterine rupture occurred in 2/22 cases vs. 0 in the control group. The asphyxia-related death-damage group did not differ from the control group (larger confidence interval) regarding leanness (Fig. 1) or birthweight (Fig. 2).

Maternal, obstetric and fetal risk factors for asphyxia and hypoxic-ischemic encephalopathy

A stepwise multiple logistic regression analysis was performed to find antenatal and intrapartum risk factors for asphyxia. The analyses of FHR were included in the analysis as the CTG score. Independent risk factors associated with asphyxia were CTG score, intrauterine meconium release, operative delivery, breech delivery, single civil status, oxytocin augmentation, cord complication and external compression to assist delivery (Table V). Independent risk factors for HIE were intrauterine meconium release, OR = 28.3 (95%CI 5.4, 238.0); CTG score, OR = 0.3 (95%CI 0.2, 0.6); and operative delivery, OR = 32.0 (95%CI 3.9, 455.8).

Discussion

This population-based study demonstrated an association between birth asphyxia, defined as an

Table V. Stepwise multiple logistic regression analyzes of antenatal and intrapartum risk factors for asphyxia

	Odds ratio (95%CI)	p-value
CTG score (0–10)	0.5 (0.4, 0.6)	<0.0001
Intrauterine meconium release	4.1 (1.8, 9.8)	<0.0001
Operative delivery	8.7 (3.4, 24.6)	<0.0001
Breech delivery	20.3 (3.0, 416.5)	0.0004
Civil status: single	7.1 (2.0, 27.6)	0.0014
Oxytocin augmentation	2.9 (1.4, 6.3)	0.0019
Cord complication	15.8 (2.1, 341.5)	0.0067
External compression	6.2 (1.3, 45.7)	0.0251

CTG, cardiotocography.

Apgar score <7 at 5 min, and the CTG score, intrauterine meconium release, operative delivery, breech delivery, single civil status, oxytocin augmentation, cord complication, external compression to assist delivery and neonatal leanness. Prevention of birth asphyxia and its ensuing neonatal complications is dependent on the early identification of pregnancies at risk for asphyxia, with appropriate intervention in selected cases. The use of various clinical risk-scoring systems, fetal heart rate monitoring and fetal scalp blood sampling have all been evaluated in the prediction of birth asphyxia (16–21). The construction of risk-scoring systems has been based on our present knowledge of risk factors. This study is one of the few population-based studies on risk factors for asphyxia (4–7) and, as far as we are aware, none of the previous population-based studies have separately investigated the importance of risk factors for asphyxia, HIE and asphyxia-related death/disability.

The incidences of birth asphyxia, HIE and asphyxia-related death/disability were low in this study. In an Australian study (22), the incidence of moderate or severe newborn encephalopathy was reported to be 3.8/1000 term live births, which compares with an HIE incidence of 1.8 per 1000 term live births in the present study. In our study the occurrence of Apgar-defined asphyxia was associated with socioeconomic factors, single mother and foreign nationality. In the Australian study (22) the socioeconomic factor of unemployment was associated with asphyxia. However, factors such as maternal age, smoking and illness, time of delivery (day/night, seasonal) and previous caesarean section did not affect the incidence of birth asphyxia in the present study. In the study (22) by Badawi *et al.* thyroid disease, severe preeclampsia and bleeding were associated with asphyxia, which was also the case in our study.

In the present study asphyxia was associated with abnormal fetal heart rate variability, lack of accelerations and decelerations. The limitations in

the clinical prediction of intrapartum fetal asphyxia have previously been extensively evaluated and discussed (16–21), and several authors have questioned the generalized use of FHR monitoring in low-risk populations (18–20). The predictive value of clinical-risk scoring was evaluated in 1909 consecutive pregnancies where the incidence of intrapartum asphyxia was 2.3% (based on blood-gas analyzes) (21). The predictive value of clinical-risk scoring and periodic fetal heart rate assessment was found to be low. However, electronic FHR monitoring has been shown to be superior to intermittent auscultation in detecting fetal acidemia at birth (e.g. Vintzileos *et al.*; 23). Low (24) found that FHR variables associated with fetal asphyxia included absent and minimal baseline variability and late and prolonged decelerations. Fetal heart rate patterns with absent baseline variability were the most specific but identified only 17% of the asphyxia group. In the present study there was a marked difference in the results of FHR assessment between all three asphyxia groups and the relevant control groups regarding the fetal heart rate parameters analyzed. Thus in this study population there was a significant over-representation of pathological FHR findings compared with the matched control population, when the infant developed asphyxia. Multiple regression analysis revealed an association between asphyxia and the CTG score. This implies that CTG, despite its limitations, could be of importance even in low-risk populations such as the one in this study where 89% of the women delivering an asphyxiated neonate had experienced a normal pregnancy. Intrauterine meconium release was also associated with asphyxia, indicating that CTG should always be used in deliveries with intrauterine meconium release. Oxytocin augmentation was also associated with asphyxia, indicating that women with oxytocin-augmented labor are another group that should be surveyed with CTG.

Operative delivery was more common in the asphyxia group. The incidence of caesarean section for the control group was 11%, which can be compared with a mean caesarean section rate of 12% (elective 6–7%, emergency 5–6%) during this period in the city of Göteborg. In the asphyxia group the emergency caesarean section rate was 38%, which is in accordance with the results of Badawi *et al.* (25) An abnormal CTG was over-represented in the asphyxia group, and thus it is possible that caesarean sections were performed in an attempt to minimize the problem of an asphyxiated newborn. This did not however, explain all the observed cases of birth asphyxia in this study, and it is possible that some fetuses were damaged before the CTG changes occurred or that appro-

priate measures were not taken when the CTG was abnormal. Previous authors (26) have also indicated that neonatal encephalopathy may also originate in the antepartum period. The fact that breech delivery was associated with asphyxia is difficult to interpret. Is the breech child already compromised before delivery or is a fetus exposed to a vaginal breech delivery at increased risk, as a randomized study recently reported (27)? External compression to assist delivery is a method that has been used for many years and this study demonstrated an increased risk of asphyxia associated with its use. Thus the question arises as to whether or not it is time to abandon this method.

Neonates in the asphyxia group were longer and leaner than the controls according to the analysis of BLSDS and LeanSDS. This finding is in accordance with previous studies that have indicated an over-representation of intrauterine growth retardation in infants with birth asphyxia (28). Previous studies have shown that SGA infants with a low Ponderal index were more likely to develop asphyxia than SGA infants with an adequate Ponderal index (29–31). As the Ponderal index changes with gestational age we have used LeanSDS in this study because it is easier to interpret, and besides being a continuous measure it also gives the relation to the statistical distribution. Furthermore, individual values can be added for more powerful statistical analysis in contrast to the Ponderal index, which is given as percentiles. This study has shown that leanness rather than being light for date (SGA) was related to asphyxia as defined by a low Apgar score. This is also in accordance with a study by Karlberg *et al.* (32) who evaluated leanSDS in newborn infants delivered in Sweden during the period 1987–88 (approximately 200 000 deliveries). That study demonstrated a graded negative 'dose-response' for leanness and Apgar score at 1 and 5 min. Thus, leanness is a risk factor for asphyxia presumably because of its decreased energy stores and supply. However, it should be noted that an increased degree of leanness could not be demonstrated in the more severely asphyxiated group (HIE) or in the asphyxia-related death/disability group, where asphyxia was often an unexpected catastrophic event during labor. Twelve of the 22 infants in the asphyxia-related death/disability group had experienced a catastrophic event, but only one infant was growth retarded.

Conclusion

This study has shown an association between neonatal asphyxia and CTG score, intrauterine meconium release, operative delivery, breech delivery, single civil status, oxytocin augmentation, cord

complication, external compression to assist delivery and neonatal leanness.

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