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Original paper

Identification of peripartum near-miss for perinatal audit

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Abstract

Introduction: **Today, perinatal audit focuses basically on cases of perinatal mortality. In most centres in Western Europe, perinatal mortality is low. Identification of metabolic acidosis at birth may increase index cases eligible for evaluation of perinatal care, and this might improve quality of perinatal audit. The aim of this study is to assess the incidence of metabolic acidosis at birth in order to estimate its impact on perinatal audit.**
Patients and Methods: **Cord blood was analysed for every neonate born between January 1, 2010 and December 31, 2012 in Ziekenhuis Oost-Limburg, Genk. Acidosis was defined as an umbilical arterial pH ≤ 7.05 with or without a venous pH ≤ 7.17. Respiratory acidosis (RA) was defined as acidosis with normal base excess, and metabolic acidosis (MA) was defined as acidosis with an arterial or venous base excess ≤ -10 mmol/L. In case of failed cord blood sampling, 5 minute Apgar score ≤ 6 was considered as the clinical equivalent of MA. Retrospective chart review of obstetric and paediatric files was performed for all cases of MA, together with review of paediatric follow-up charts from at least 6 months after birth. Perinatal asphyxia was defined as biochemical evidence for MA at birth, associated with early onset neonatal encephalopathy and long-term symptoms of cerebral palsy.**
Results: **In a total of 6614 babies, perinatal death up to 7 days of life occurred in 40 babies (6.0‰). Acidosis was present in 183 neonates (2.8%), of which 130 (2.0%) had RA and 53 (0.8%) had MA. Of the 173 neonates with unknown pH values, 6 had Apgar scores ≤ 6. Of 59 babies born with MA or its clinical equivalent, 52 (88.1%) showed no neurologic symptoms at birth. Two (3.4%) died in the early neonatal period, one after abruptio placentae and one due to chorioamnionitis and severe prematurity. Five (8.5%) MA babies had symptoms of early onset neonatal encephalopathy, which recovered in three (5.1%), and persisted long-term in two others (3.4%). The two babies with cerebral palsy (prevalence 1/3300) were both born after instrumental vaginal delivery for foetal distress.**
Conclusion: **In our study cohort, the incidence of perinatal mortality is 6‰. The incidence of metabolic acidosis is 9‰. Addition of cases of metabolic acidosis to those of mortality doubles index cases eligible for perinatal audit. The incidence of babies surviving with cerebral palsy after metabolic acidosis at birth is very low (0.3‰). Our results suggest that instrumental delivery for foetal distress might be a risk factor for metabolic acidosis with persisting neurologic dysfunction. Our study illustrates that identification of peripartum near-miss is useful for perinatal audit.**

Key words: **Perinatal audit, peripartum near-miss, metabolic acidosis, cerebral palsy, instrumental delivery, perinatal asphyxia.**

Introduction

Perinatal audit is an important measure for improvement of obstetric care (Pattinson et al., 2005). Although today no trials exist to prove the importance of perinatal audit, it is suggested that information on causes of death in pregnant women or their babies provide essential information to identify problem areas and to formulate feedback.

Nowadays, audit is basically focused on cases of perinatal mortality. Perinatal mortality has become low in most centres in Europe. Perinatal mortality rates (up to 27 days of life) in Europe range from 1.2‰ in Iceland to 5.5‰ in Romania (EURO-

PERISTAT, 2013). In 2011, perinatal mortality in neonates with a birth weight of at least 500 g in Flanders was reported to be 6.6‰. If only neonates with a birth weight of 1000 g or more were taken into account, perinatal mortality was as low as 3.8‰ (SPE, 2011). As such, a very low number of index cases are available for perinatal audit.

Umbilical cord pH at birth is commonly used as an outcome measure in obstetric care. The Sundström criteria define acidosis as an arterial pH ≤ 7.05 or a venous pH ≤ 7.17 (Sundström et al., 2000). Respiratory acidosis is defined as acidosis with a normal base excess (> -10 mmol/L). It results from hypoxemia and hypercapnia in the early stages of impaired blood supply to the foetus. In case of continued impaired blood supply, anaerobic metabolism is activated in the foetus. Lactate is formed which leads to an increased base excess (≤ -10 mmol/L) and as such to metabolic acidosis.

Neonates born with metabolic acidosis are considered at risk for developing early onset encephalopathy or long-term cerebral palsy. The incidence of neonatal encephalopathy is reported to range from 2.0 to 6.0‰ (Sundström et al., 2000. Badawi et al., 1998. Ellis et al., 2000. Brown et al., 1974). Identification of metabolic acidosis at birth may increase index cases eligible for perinatal audit.

The aim of this study is to assess the incidence of intrapartum metabolic acidosis in order to establish whether diagnosis of metabolic acidosis at birth is useful for perinatal audit.

Patients and Methods

All babies born in our hospital between January 1, 2010 and December 31, 2012 were included in this single-centre prospective study. Cases of termination of pregnancy for congenital malformations were excluded. Perinatal mortality was defined following the WHO definition as the sum of intrauterine death of a foetus weighing at least 500 g, intrapartum death and early neonatal death up to 7 days of life.

Cord blood was sampled and analysed for every live-born neonate, including premature neonates and twins or higher order multiple pregnancies. Immediately after delivery, the umbilical cord was clamped on two sides. Arterial and venous cord blood was separately sampled from the clamped part of the umbilical cord. After sampling, air bubbles were removed from the syringes and blood samples were immediately sent to the laboratory for analysis of arterial and venous pH and base excess.

The Sundström criteria were used to define acidosis (Sundström et al., 2000). Acidosis was considered as an arterial pH ≤ 7.05 with or without a venous pH ≤ 7.17. A distinction was made between respiratory acidosis (RA) and metabolic acidosis (MA), by means of arterial or venous base excess. A base excess of -10 mmol/L was used as cut-off value. RA was considered as acidosis with a base excess of -10 mmol/L or above. MA was considered as acidosis with a base excess below -10 mmol/L. If there was a failure in cord blood sampling or analysis, a 5-minute Apgar score ≤ 6 was considered as the clinical equivalent of MA.

As such, all cases of metabolic acidosis at birth were identified for perinatal audit. Retrospective chart review of obstetric and paediatric files was performed for every case of MA. For babies with significant neurologic morbidity during hospital admission, paediatric follow up charts from at least 6 months after birth were studied. The criteria of the ACOG Task force on Neonatal Encephalopathy and Cerebral Palsy from 2003 (Table I) were used for defining birth asphyxia. It should be emphasized that the criteria for pH and base excess are more

Table I. — ACOG Task force criteria on Neonatal Encephalopathy and Cerebral Palsy (2003).

Essential criteria (must meet all four):
1. Evidence of a metabolic acidosis in foetal umbilical cord arterial blood obtained at delivery (pH < 7, 00 and base deficit < 12 mmol/L)
2. Early onset of severe or moderate neonatal encephalopathy in infants born at 34 or more weeks of gestation
3. Cerebral palsy of the spastic quadriplegic or dyskinetic type
4. Exclusion of other identifiable aetiologies such as trauma, coagulation disorders, infectious conditions, or genetic disorders.
Criteria that collectively suggest an intrapartum timing (within close proximity to labour and delivery, eg. 0–48 hours) but that are nonspecific to asphyxial insults:
1. A sentinel (signal) hypoxic event occurring immediately before or during labour
2. A sudden and sustained foetal bradycardia or the absence of foetal heart rate variability in the presence of persistent, late, or variable decelerations, usually after a hypoxic sentinel event when the pattern was previously normal
3. Apgar scores of 0–3 beyond 5 minutes
4. Onset of multisystem involvement within 72 hours of birth
5. Early imaging study showing evidence of acute nonfocal cerebral abnormality.

Table II. — Perinatal mortality: n = 40 (6.0‰).

Year	Intra-uterine death	Intrapartum death	Neonatal death ≤ 7 d	**Perinatal mortality**
2010	8	4	3	**15/2117 (7.1 ‰)**
2011	7	3	2	**12/2231 (5.4 ‰)**
2012	7	0	6	**13/2266 (5.7 ‰)**
Total	22	7	11	**40/6614 (6.0‰)**

stringent than the criteria used in our study (ACOG, 2003).

For every case of MA, the obstetric file was retrospectively reviewed to identify risk factors for metabolic acidosis at birth. Data on gestational age at birth, onset of labour, mode of delivery and indication for instrumental or caesarean delivery were collected. Also, information concerning pregnancy complications such as diabetes mellitus (gestational or pre-existing) and hypertensive disorders of pregnancy (essential hypertension or pre-clampsia) was obtained.

Results

During the three year study period, 6614 babies were born for a total of 6459 deliveries: 6305 singletons, 153 twins and 1 triplet. Perinatal death occurred in 40 of 6614 neonates (6.0‰). Data on perinatal mortality are summarized in Table II.

Acidosis was present in 183 neonates (2.8%). Respiratory acidosis was found in 130 (2.0%) and metabolic acidosis in 53 neonates (0.8%). For 173 babies (2,6%) we didn't have data on cord blood due to failed sampling or analysis. Six of them, including 1 preterm neonate, had a 5-minute Apgar score of 6 or less and were therefore considered as clinically equivalent of metabolic acidosis.

Of 59 babies born with MA or its clinical equivalent, 52 (88.1%) showed no neurologic symptoms at birth. Two (3.4%) MA babies died in the early neonatal period, one after placental abruption and one due to chorioamnionitis and severe prematurity. Five (8.5%) MA babies had symptoms of early onset neonatal encephalopathy, which recovered in three (5.1%), and persisted

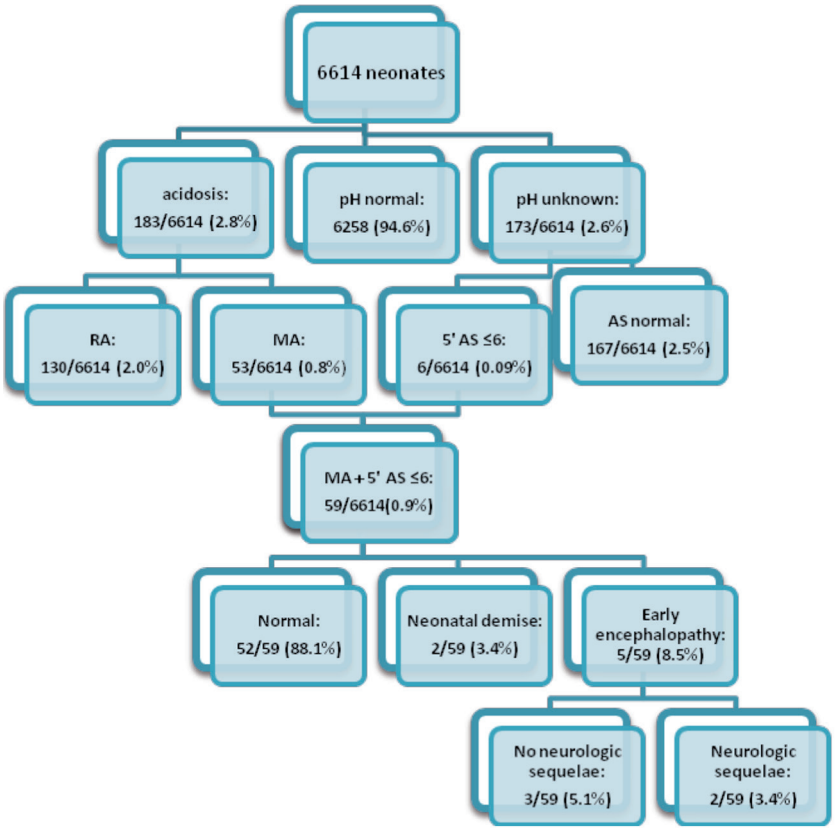

Fig. 1. — Incidence of acidosis at birth and neonatal outcome.

Table III. — Mode of delivery and pregnancy complications in MA pregnancies, n (%).

Gestational age at birth	
Preterm (< 37 weeks)	6 (11.3%)
Term (37-41 weeks)	38(71.7%)
Postterm (≥ 41 weeks)	9 (17.0%)
Pregnancy complications	
Diabetes Gestational diabetes Juvenile diabetes	3 (5.7%) 2 1
Pre-eclampsia/hypertension/APC resistance syndrome	5 (9.4%)
No pregnancy complication	45 (85.0%)
Mode of delivery	
Spontaneous vaginal delivery	28 (52.8%)
Instrumental virginal delivery (Vacuum extraction/Forceps extraction)	14 (26.4%)
Caesarean section	11 (20.8%)
Onset of labour	
Spontaneous onset of labour	38 (71.7%)
Induction of labour	12 (22.6%)
Primary caesarean section	3 (5.7%)

long-term in two others (3.4%). The two babies with cerebral palsy (prevalence 1/3300) were both born after instrumental vaginal delivery for foetal distress.

Table III summarizes data on mode of delivery and pregnancy complications in the 53 pregnancies featuring biochemical metabolic acidosis at birth. There were three primary caesarean sections. Of these, 2 were performed for clinical suspicion of abruption placentae and 1 was an elective repeat caesarean section. Among the 8 cases of caesarean section during labour, 7 were performed for foetal distress and 1 was performed for foetal intolerance to syntocinon. Vacuum extraction was performed in 14 cases, of which foetal distress was the reason in 10 and delayed expulsion in 4.

Discussion

The results of our study demonstrate that identification of metabolic acidosis at birth is feasible for improvement of perinatal audit. Adding cases of peripartum near-miss to cases of perinatal mortality doubles index cases available for audit. Our results suggest that instrumental delivery for foetal distress, specifically in technically difficult cases, might contribute to poor neurologic outcome in neonates born with metabolic acidosis.

An advantage of our study is that it is a prospective, single-centre study. Therefore, selection and sampling bias are minimized. Data on cord blood gases were missing for 173 babies (2,6%).

A major disadvantage of our study is that we do not have follow-up after the age of 6 months for every baby born with metabolic acidosis. Developmental problems sometimes become evident later on in childhood. So in fact, we might have underestimated the incidence of neurologic disability in infants born with metabolic acidosis. Another weakness is that we do not have data on neurologic outcome in neonates born without acidosis. Neurologic disability is known to have other causes than acidosis as well. If the incidence of long-term neurologic sequelae would be the same in the non-acidosis group, conclusions on the contribution of peripartum MA to poor neurologic outcome cannot be drawn.

Several studies found an association between acidosis and poor neonatal outcome. On the short term, low umbilical cord pH is reported to be associated with more Apgar score below 7 at 5 minutes, NICU admissions, neonatal mortality, hypoxic ischemic encephalopathy and intraventricular haemorrhage or periventricular leucomalacia (Victory et al., 2004. Malin et al., 2010). On the long term, more cerebral palsy (Malin et al., 2010), speech problems (Ingemarsson et al, 1997) and developmental problems at the age of 6.5 years are seen (Hafström et al., 2012).

The incidence of metabolic acidosis in our cohort was 0.9%. This is in line with the incidence found in other high-income countries. An Australian observational study reported an incidence of MA, defined as arterial pH < 7.0 and BE < 12 mEq/L, of

Table IV. — Literature review on antepartum and intrapartum risk factors for neonatal near-miss.

Study	Outcome measure	Antepartum risk factors	Intrapartum risk factors
Badawi et al., 1998	Moderate or severe encephalopathy	Increasing maternal age Decreasing parity Socio-economic status Family history of neurologic disease Fertility treatment Maternal thyroid disease during pregnancy Severe pre-eclampsia Moderate/severe vaginal bleeding Clinically diagnosed viral illness	Induction of labour Operative vaginal delivery Emergency caesarean section
Maisonneuve et al., 2011	Umbilical artery pH < 7.00	Maternal age > 35 Prior neonatal death Prior caesarean delivery	General anaesthesia Thick meconium Uterine rupture Abnormal foetal heart rate
Westerhuis, 2012	Metabolic acidosis	Gestational age at delivery Nulliparity Previous caesarean section Maternal diabetes	Spontaneous onset of labour Meconium

0.54% in their cohort of 12 345 live-born babies from 20 weeks GA onwards (White et al., 2010). In a Dutch multicentre study, the incidence of metabolic acidosis at birth, arterial pH < 7.05 and BE < 12 mEq/L, was 1.9% among 5667 high risk singleton pregnancies with cephalic presentation beyond 36 weeks of gestation (Westerhuis et al., 2012). The prevalence of cerebral palsy in live born children is reported 2 per 1000 in Europe (Blair et al., 2010; SCPE, 2000). In our cohort we found a prevalence of only 0.3 per 1000 live born neonates with metabolic acidosis. However as explained, we cannot exclude missed cases due to missing long-term neonatal follow-up and we do not have data on neurologic outcome in babies without metabolic acidosis at birth.

When risk factors for neonatal near-miss can be identified, appropriate measures to avoid its consequences can be installed. Table IV summarizes the reported risk factors for neonatal near-miss studied in literature.

Fourteen of 53 babies with MA at birth were born with instrumental delivery. The two babies with MA at birth and persistent neurologic sequelae, were both born after instrumental vaginal delivery for foetal distress. As such, our study suggests that instrumental delivery for foetal distress might contribute to poor neonatal outcome, specifically in technically difficult cases. Therefore, alternative management options might be considered for foetal distress at full cervical dilatation, such as an emergency caesarean section or intra-uterine resuscitation when foetal wellbeing is confirmed by foetal scalp blood sampling.

The systematic review of McIntyre et al. (2013) identified only one eligible study that investigated the association between forceps delivery and CP in which a significant association was found. No eligible studies on vacuum extraction and the incidence of CP were found. In this review ten consistent risk factors for CP were identified: placental abnormalities, major and minor birth defects, low birth weight, meconium aspiration, instrumental/emergency caesarean delivery, birth asphyxia, neonatal seizures, respiratory distress syndrome, hypoglycaemia and neonatal infections (Milsom et al., 2002).

A Swedish retrospective case-control study investigated risk factors in 225 cases of birth asphyxia, defined as Apgar Score < 7 at 5 minutes. An overrepresentation of vacuum extraction deliveries was found in the asphyxia group (OR 4.0, 95% CI 1.4-11.4). It is impossible to assess whether the vacuum extraction was the primary cause or the consequence of foetal asphyxia because of an abnormal CTG monitoring (Leung et al., 2003).

The results of our study demonstrate that adding cases of metabolic acidosis and birth asphyxia is useful for perinatal audit. It is still questionable whether this will improve perinatal care itself. Also, literature resources are still scarce on this topic. Findings of the available studies on this topic are summarized in Table V.

Table V. — Literature review on usefulness of identifying metabolic acidosis (MA) and birth asphyxia for perinatal audit.

Study	Study design	Findings
Leung et al., 2003	Observational study to evaluate implementation of a code sheet in clinical practice to reduce birth trauma and birth asphyxia due to instrumental delivery.	Significant decrease in birth trauma and asphyxia from 2.8% to 0.6%.
Jonsson et al., 2009	Case-control study: 161 cases (pHa < 7.05 and BE ≥ 12 mmol/l) versus 322 controls (pHa > 7.05)	Significant reduction (49 to 13%) of suboptimal care (oxytocin misuse or failure to respond to pathological CTG pattern)
White et al., 2010	Observational study on 19 646 deliveries.	Significant reduction in neonates with pHa < 7.10 and lactate > 6.1 mmol/L after implementation of umbilical cord blood analysis in.
Low et al., 2011	Retrospective reassessment of foetal heart rate record in 166 term pregnancies with arterial BE > 12 mmol/L.	FHR predictive in 109 cases. FHR nonpredictive in 29 cases. No FHR record available in 28 cases.
Westerhuis et al., 2012	Multicentre randomized clinical trial: Retrograde reassessment of CTG and/or STAN data for 61 cases of MA, perinatal death or moderate/severe hypoxic ischemic encephalopathy.	Foetal indication to intervene in 42 cases, in 24 cases indication was already present 20 minutes before delivery.
Dehaene et al., 2014	Retrograde reassessment of perinatal care in 19 case of proven metabolic acidosis.	Three cases were possibly preventable because of absent foetal monitoring during second stage of labour. Four cases were considered preventable: there was a delay of intervention following a significant ST event during second stage of labour in two and during first stage of labour in one.

Conclusion

In our study cohort in a total of 6459 deliveries, the incidence of perinatal mortality is 6‰, and the incidence of metabolic acidosis is 9‰. Addition of cases of metabolic acidosis to those of mortality doubles index cases eligible for perinatal audit. The incidence of babies surviving with cerebral palsy is very low (0.3‰). Our data suggest that instrumental delivery for foetal distress might be a contributing risk factor for persisting neurologic dysfunction. Our study illustrates that identification of peripartum near-miss is useful for perinatal audit.

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