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cols and errors in implementing guidelines are common (4).

INTRODUCTION
Experience over the last century has demonstrated that peri-
natal mortality can be reduced by improved obstetrical and
neonatal care (1). With the aim to avoid errors in care by
implementing system-based changes, a systematic review of
the pitfalls and mistakes in the clinical practice of perinatal
medicine can be useful.

In a previous Swedish nationwide report, we identified
177 newborn infants over a 16-year period who had suf-
fered from severe birth asphyxia presumably due to delivery-
related malpractice. The most common causes of obstetrical
errors were that established guidelines for foetal surveillance
were not followed (i.e. negligence to supervise foetal well-
being), signs of foetal asphyxia were overlooked, misuse of
oxytocin, and a non-optimal choice of mode of delivery. We
concluded that foetal surveillance and attention to signs of
asphyxia must be improved, that there is a need to improve
cooperation between professionals in the labour unit and, to
create security barriers (2).

Less is known about potential flaws in the immediate
management of the asphyxiated newborn infant. Although
national guidelines for neonatal resuscitation have been in
place for many years (3), compliance with and the effective-
ness of these guidelines has not been studied. Video uptakes
from emergency rooms indicate that deviations from proto-
cols and errors in implementing guidelines are common (4).
The aim of this study was to scrutinize the initial resusci-
tation procedure of the previously identified 177 newborn
infants with severe asphyxia (2).

METHODS
We retrieved information on all 472 claims from 1990 to
2005 concerning suspected malpractice during pregnancy,
delivery and the neonatal period, sent to the County Coun-
cil’s Mutual Insurance Company under the Patients’ Advi-
sory Committee, Sweden (PAC, Person Skade Reglering AB
in Swedish). We included infants with a gestational age of
>33 completed gestational weeks, planned vaginal delivery,
a normal CTG tracing at admission to the delivery unit, and
severe asphyxia-related neurological outcomes. We defined
labour-related asphyxia as an Apgar score of < 7 at 5 min
and, if the acid base status was measured in the umbilical
cord at delivery or shortly thereafter, metabolic acidosis as a
pH of < 7.05 and/or a base excess (BE) of < –12 (5,6). In all,
we included 177 infants with labour-related asphyxia, pre-
sumably caused by malpractice in connection with labour.
The selections of the cases and the investigation procedure
have previously been described in detail (2).

A structured protocol, including clinical data retrieved
from the obstetric and neonatal records, was used. We col-
lected information on pregnancy and maternity care, onset
of labour, presentation and mode of delivery, time of pag-
ing of a physician, the date and time of birth, gestational
age, gender, birth weight, Apgar scores at 1, 5 and 10 min,
Stimulation of the newborn
Improvement of status
Give the infant to mother

### Primary apnoea

| Minutes | Asystolia |
|---------|-----------|
| 0       | Intubation |

### Secondary apnoea

- Mask ventilation
- Intubation

### Persistent bradycardia

- Increase insufflations pressure
- Increase inspiration time
- Control of chest movements

| Minutes | Asystolia |
|---------|-----------|
| 1       | Chest compressions |
| 2       | Persistent asystolia |

### Persistent bradycardia

- Intubation
- Ventilation
- Adrenaline

| Minutes | Asystolia |
|---------|-----------|
| 3       | Continue ventilation |
| 5       | Persistent asystolia |

### Persistent bradycardia

- Correction of acidosis
- Repeat Adrenaline 1-2 times

| Minutes | Asystolia |
|---------|-----------|
| 15      | Persistent asystolia |

Consider cessation/discontinuation of resuscitation

### Persistent apnoea

| Minutes | Asystolia |
|---------|-----------|
| 30      | Persistent apnoea |

Consider cessation/discontinuation of resuscitation

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**Figure 1** National Swedish guidelines for neonatal resuscitation 1996–2006.

Umbilical cord acid base status, acts of resuscitation, the length of stay (LOS) in the neonatal intensive care unit (NICU), degree of hypoxic ischemic encephalopathy (HIE) and investigations during the first weeks of age. We also retrieved information on the long-term follow-up, including information on morbidity (cerebral palsy [CP] and other neurological disorders), degree of disability or death from the paediatric medical records.

All medical documents were scrutinized and computerized by one of us (SB), a specialist in obstetrics for a period of 15 years, a graduate of the American Neonatal Resuscitation Provider Program, and also an instructor of team training in neonatal resuscitation at the Centre for Education in Pediatric Simulator (CEPS) at Södersjukhuset in Stockholm (1). The reviewer SB needed access to all information from each case record and was therefore not blinded to the final outcome.

National guidelines on neonatal resuscitation in place during the period of the investigation are presented in Figure 1. (3) According to these guidelines, (revised in 2006), clearing the airways from meconium and initiating bag and mask ventilation with initially 40% oxygen should be performed immediately in all newborn infants with a heart rate below 100 beats per min (bpm). In cases of asystolia,
Endo-tracheal intubation and administration of thoracic compressions should immediately be performed. Early intubation (within the first 2–3 min after birth) was recommended in cases with persistent bradycardia to ensure free airways and satisfactory ventilation. Adrenaline, either through the endo-tracheal tube or intravenously, should be administered if the heart rate did not rise despite satisfactory ventilation. Correction of metabolic acidosis should be performed in cases of refractory bradycardia and terminal apnoea (2 mmol buffer/kg i.v. for 5 min). Interruption of resuscitation should be considered if there is no evidence of heart activity after 15 min or if no spontaneous breathing or body movements have occurred during the first 30 min after birth.

According to national clinical practice in obstetrics, the paediatrician should be paged before the birth in cases of immediate when deliveries are complicated by shoulder dystocia. In twenty-five deliveries, trial of labour with vacuum extraction or forceps was performed before converting to emergency CS.

The median Apgar score was 5 (range 0–7) 10 min after birth, and at an older post-natal age there was rarely any notation of an Apgar score. A total number of 52 (18%) infants died, 16 in the neonatal period and 16 during later follow-up.

Information about the time of the initiation of artificial ventilation was noted in 167 of all 177 infants. Despite clear indications, artificial ventilation was not initiated within 1 min after birth in seven infants (4.2%) while waiting for a physicist. Two of these had an Apgar score of ≤3 at 10 min and one infant died (Table 1). The median time for mask ventilation was 8 min (range 0–30 min), and mask ventilation proceeded for more than 15 min in 34 infants. Eight of these infants had an Apgar score of ≤3 at 10 min and seven infants died.

Endotracheal intubation was performed in 112 cases, and the time of intubation was noted in 95 infants. Median time for intubation was 6 min (range 0–180 min). In 72 infants, intubation was not, despite clear indications, performed within 3 min after birth. Twenty-six of these infants had an Apgar score of ≤3 at 10 min and 17 died (Table 1). Twenty-eight infants were intubated after more than 10 min of age. The time of the initiation of spontaneous breathing was noted for 119 infants of all 177 infants (67%), and the median time was 18 min (range 1–70 min). Forty-one infants were subjected to chest compressions. Despite asystole at one min of age (n = 33), thoracic compressions were not performed on five infants, one of which had an Apgar score of ≤3 at 10 min (Table 1).

Adrenaline was not provided in 14 of 33 infants with asystolia at one min and nor was it provided in seven of 24 infants with persistent bradycardia. In all 21 infants where adrenaline was not provided, six infants had an Apgar score of ≤3 at 10 min and five of these infants died (Table 1). An acid base status at birth or shortly after birth was analyzed in 107 of all the infants (60%). The median pH at or shortly after birth was 6.83 (Range 6.57–7.20) and BE −22.5 (−10 to −40) mmol/L. One hundred thirty-five infants were treated with buffer. The time for the correction of acidosis was noted in 219 of all 219 infants and the median time for correction was 15 min (range 1–120 min). In two infants with asystolia or persistent bradycardia there was no correction of metabolic acidosis. Both had an Apgar score of ≤3 at 10 min and one of these infants died (Table 1). Four infants had a post-natal asphyxia after a traumatic delivery with a pH ≥7.05, a BE ≥−12 mmol/L, but an Apgar score of ≤7 at 5 min.

Information relating to the attending physician and his/her time of arrival at the resuscitation was not routinely noted. Of 92 vaginal-risk deliveries (65 instrumental, 18 with shoulder dystocia and nine breech deliveries), the presence
Table 1  Failure events during neonatal resuscitation of 177 asphyxiated newborn infants and outcome

| Failure Event                                                                 | n (%) | Apgar score 0–3 at 10 min (n) | Deaths1 n (%) |
|-------------------------------------------------------------------------------|-------|-------------------------------|---------------|
| 1. Non satisfactory/unsatisfactory resuscitation                              |       |                               |               |
| (a) Artificial ventilation not started within 1 min after birth               | 84 (47) |                               |               |
| (b) Endotracheal intubation not performed within 3 min after birth           | 72 (41) | 26                            | 17 (24)       |
| (c) Thoracic compressions not performed despite asystole2                    | 5 (3)  | 1                             | 0 (0)         |
| 2. Not satisfactory drug administration                                       | 23 (13) |                               |               |
| (d) Adrenaline not provided despite asystole1                                 | 14 (8)  | 6                             | 5 (24)        |
| or persistent bradycardia3                                                   | 7 (4)  |                               |               |
| (e) No correction of metabolic acidosis3 despite asystole or persistent bradycardia | 2 (1)  | 2                             | 1 (50)        |
| 3. Late initiation of resuscitation                                          | 19 (11) |                               |               |
| (f) Paediatrician, neonatologist or anaesthesiologist not present before the birth in spite of known delivery complication | 11 (6)  | 2                             | 3 (27)        |
| (g) Paediatrician, neonatologist or anaesthesiologist arrived within 4 min after birth in cases of unexpected asphyxia (n = 48) | 8 (5)  | 4                             | 4 (50)        |
| 4. Resuscitation not interrupted                                             | 38 (22) |                               |               |
| (h) Resuscitation not interrupted:                                           |       |                               |               |
| o In spite of asystole for more than 15 min                                  | 8 (5)  | 8                             | 7 (88)        |
| o In spite of lack of spontaneous breathing during the first 30 min after birth | 30 (17) | 15                            | 8 (27)        |

1There were 16 neonatal deaths, and another 16 during follow-up (total deaths 32/177 = 18%).
233 neonates had asystole at 1 min of age.
324 neonates had persistent bradycardia at 5 min.

All infants were admitted to the NICU. Neonatal outcomes following resuscitation are summarized in Table 2. The median length of stay in the NICU was 24 days (range 1–120). Of 128 infants born at hospitals without facilities for ventilator treatment, 48 (38%) were transferred to a university hospital shortly after birth. Seventy-eight (44%) infants were treated on ventilators for more than 24 h. Almost all (94%) infants suffered from convulsions during the neonatal period. EEG was performed on all infants and was pathological in 80%.

Examination by cerebral CT scan was performed on 137 (77%) of all infants, and the scan was assessed as pathological in 96 infants (70% of those scanned). In addition, 58 (33%) were examined with magnetic resonance imaging (MRI) of the brain, and the MRI was abnormal in 49 infants.

The degree of HIE was diagnosed in 137 infants. Among those, HIE I was diagnosed in 10, HIE II in 69 and HIE III in 58 infants (Table 2).

Mortality and long-term morbidity

Information on mortality, neurological disorders, co-morbidity and degree of impairment has been published previously (2). Briefly, there were 16 neonatal deaths, and another 16 infants died during the follow-up. Information
on morbidity was for 45 infants based on case records with a limited follow-up period. These infants were considered to be suffering from an unspecified CP syndrome with diagnosed encephalopathy during the neonatal period. Nine of these infants died before a final diagnosis could be made. Sixty-nine of the 116 children with information about the type of CP had dyskinetic CP, 18 spastic tetraplegia, 21 spastic diplegia and 8 had hemiplegia.

DISCUSSION
We are very much aware of the difficulty of evaluating the efforts of resuscitation in these most severely asphyxiated infants since the final outcome was poor for all, but nevertheless we could evaluate compliance with the guidelines relating to neonatal resuscitation. In accordance with our previous findings on insufficient adherence to current guidelines on foetal surveillance (2), we found indications of similar insufficiencies with respect to neonatal resuscitation and that resuscitation was not performed according to guidelines in the case of 84 infants (47%) (Table 1). Since ventilation is the most important issue in cases of neonatal resuscitation, we consider the most perturbing findings to be a delayed initiation of extensive resuscitation, lack of satisfactory ventilation by insurance of free airways by an early intubation, and untimely interruption of resuscitation (Fig. 1 and Table 1).

Foetal surveillance at admission to the delivery ward registered no irregularities in any of the cases, presumably indicating a healthy foetus. After delivery, umbilical cord blood gas and acid-base assessment are the most objective determinations of the foetal metabolic condition, but acid-base status at birth or shortly thereafter was available only in 60% of all cases. On the other hand, Apgar score at 5 min, which is considered to be a reliable indicator of asphyxia in the absence of malformations, was noted in all cases (7). Median pH at birth was 6.83, median BE was −22.5 and the median Apgar score at 5 min was 5, indicating that the neonates in the study group suffered from profound asphyxia at birth and were in great need of an immediate initiation of neonatal resuscitation. The documentation of the resuscitation was generally poor and incomplete, which is a warning sign itself that ought to be elucidated. Despite the difficulty of prioritizing documentation in these stressful situations, documentation is necessary, not least in severe cases where infants might have been injured by malpractice in conjunction with labour. A simple and straightforward pre-printed protocol simplifies documentation in cases of neonatal resuscitation (8). The poor documentation also emphasizes that the results from our evaluation must be interpreted with caution.

Although there were several shortcomings during the neonatal resuscitation, we consider it most likely that the infants were primarily damaged by asphyxia in conjunction with labour. Immediately after birth, the most important shortcomings noted were, according to the national guidelines for neonatal resuscitation, unsatisfactory ventilation or drug administration and a late initiation of or untimely interruption of resuscitation.

Even if the majority of failure events defined in this study is uncontroversial, failure to interrupt resuscitation after 15 or 30 min is more contestable. There could be clinical situations when the attending physician would have considered continuing resuscitation despite a longstanding asystolia or persistent apnoea. For example, the parents may have opposed discontinuation of resuscitation. Drugs administered to the mother immediately before delivery may have depressed spontaneous breathing in the newborn infant, demanding continued artificial ventilation after birth. An unclear diagnosis of intrapartum asphyxia—for other reasons than depressant drugs—may also have added to a decision of continued resuscitation beyond the stipulated points of return.

Although the value of routine correction of acidosis in newborn infants has been rightfully questioned, we note that in our study of severely asphyxiated infants, there were two patients with asystolia or persistent bradycardia who did not receive any correction of metabolic acidosis (9,10). Both had an Apgar score of ≤3 at 10 min and one of these infants died. An acid-base status was available in 107 out of 177 infants and 135 infants were treated with buffer. The time for correction of acidosis was noted in only 47 (35%) infants. Among these infants, correction of acidosis was in many cases delayed (median time for correction 15 min (range 1–120 min) after birth). The point of time for correction of acidosis was missing in too many cases to provide meaningful indication whether an earlier correction could have improved the outcome.

Post-asphyxial management at the neonatal intensive care unit is also of vital importance for the chance of intact survival (11,12). Aggressive prevention and treatment of complications such as recurrent acidosis and hypoxia, severe hypoglycaemia, systemic hypotension, convulsions and brain oedema are necessary (13,14). Recently, moderate hypothermia has been reported as an evolving therapy with promising results in cases of neonatal asphyxia (13,15). Quality assurance of the post-asphyxial management remains to be done.

Neuroimaging gives the possibility to time-relate a brain injury. Clinical consequences are dependent on the topography of the lesion. In cases of asphyxia during labour, basal ganglia and thalamus lesions occur, predominantly because of acute and severe perfusion failure in the term or near term born infant. Newborn infants with pure basal ganglia and thalamus lesions tend to have dyskinetic CP at follow-up, often with severe motor problems but less pronounced cognitive deficits. Infants with additional central and especially hippocampal involvement are usually severely retarded in both their motor and cognitive development and develop severe bilateral spastic CP (16,17). The dominating types that occurred among our cases were dyskinetic CP and spastic tetraplegia, which are thought to be the most common subtypes of CP that are associated with intrapartum hypoxic events (14). Although some infants in the study-group may have had encephalopathy prior to labour, we find it probable that suboptimal care around delivery aggravated the encephalopathy in these infants. The median LOS at NICU
was 24 days, 44% were treated for more than 24 h in a ventilator, and almost all had convulsions with pathological EEG during the neonatal period, which indicates how severely injured these infants were at birth. Neonatal resuscitation is important in cases of asphyxia. Even if the percentage of newborns requiring assistance may be small, the implications of not receiving correctly performed neonatal resuscitation can cause lifelong impairment or even death.

This report does not intend to cover all neonatal resuscitation in connection with malpractice during delivery. However, given our outcome definition of asphyxia at birth and the long-term outcomes of these infants, the study probably mirrors the initial care of the most severely asphyctic neonates.

Ten percent of all newborns require some assistance to adjust to the extra uterine environment, while 1% needs extensive resuscitative measures to survive (1). The crucial time for improvement in cases of severe asphyxia at birth is around 10 min, and it is necessary that labour staff available initiates resuscitation and that more skilled staff participates within a few minutes (1). Asphyxia was unexpected in 48 of the infants, and in eight of these cases neither a paediatrician, nor a neonatologist or an anaesthesiologist had arrived to assist within 4 min of the birth, which may have aggravated the asphyxia. Four of these infants had an Apgar score of ≤ 3 at 10 min and died later. In addition, resuscitation was noted delayed by the absence of skilled person in 11 of the 92 cases of known delivery complications, but notation about presence before birth was missing in another 34 complicated deliveries. Due to the possibility of unexpected asphyxia, it is necessary that all staff attending childbirths continuously train how to anticipate and handle complications in conjunction with labour (18) while waiting for skilled personnel to arrive.

CONCLUSIONS

There are possibilities for improvement in the immediate neonatal resuscitation within our labour units. The most important contributions may be made by improving compliance with the guidelines concerning ventilation, and the waiting for the early assistance of skilled personnel in cases of imminent asphyxia. It is crucial that all of the staff on the labour ward is familiar with how to initiate extensive neonatal resuscitation. Every case of unexpected asphyxia, also those that recover without sequelae, should be scrutinized to enable the creation of security barriers and improvements in each labour unit, concerning both obstetrical care and neonatal resuscitation (19). In addition, the documentation of neonatal resuscitation must be improved to enable accurate and reliable evaluation.

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DISCLOSURE OF INTERESTS

No conflicts of interests.

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