Intrapartum asphyxia and hypoxic ischaemic encephalopathy in a public hospital: Incidence and predictors of poor outcome

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Objective. To determine the incidence of asphyxia and hypoxic ischaemic encephalopathy (HIE) and predictors of poor outcome in a hospital in a developing country.

Methods. Neonates of birth weight ≥2 000 g who required bag-and-mask ventilation and were admitted with a primary diagnosis of asphyxia from January to December 2011 were included. Medical records were retrieved and maternal and infant data collected and analysed. Infants who had severe HIE and/or died were compared with those who survived to hospital discharge with no or mild to moderate HIE.

Results. There were 21 086 liveborn infants with a birth weight of ≥2 000 g over the study period. The incidence of asphyxia ranged from 8.7 to 15.2/1 000 live births and that of HIE from 8.5 to 13.3/1 000, based on the definition of asphyxia used. In 60% of patients with HIE it was moderate to severe. The overall mortality rate was 7.8%. The mortality rate in infants with moderate and severe HIE was 7.1% and 62.5%, respectively. The odds of severe HIE and/or death were high if the Apgar score was <5 at 10 minutes (odds ratio (OR) 19.1; 95% confidence interval (CI) 5.7 - 66.9) and if there was no spontaneous respiration at 20 minutes (OR 27.2; 95% CI 6.9 - 117.4), a need for adrenaline (OR 81.2; 95% CI 13.2 - 647.7) and a pH of <7 (OR 5.33; 95% CI 1.31 - 25.16). Predictors of poor outcome were Apgar score at 10 minutes (p=0.004), need for adrenaline (p=0.034) and low serum bicarbonate (p=0.028).

Conclusion. The incidence of asphyxia in term and near-term infants is higher than that reported in developed countries. Apgar score at 10 minutes and need for adrenaline remain important factors in predicting poor outcome in infants with asphyxia.

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Asphyxia is defined biochemically as a condition of impaired respiratory gaseous exchange that leads to hypoxaemia, hypercapnia and metabolic acidosis. Metabolic acidaemia, defined as a base deficit of >12 mmol/L, has been associated with episodes of intrapartum asphyxia and has therefore been used to define asphyxia biochemically.[1] The problem with this definition is that a number of normal babies have metabolic acidosis, and if it is used on its own there is a risk that the incidence of asphyxia may be overestimated.[2] Clinically, the need for bag-and-mask ventilation (BMV) at birth and/or an Apgar score <7 have been used to define intrapartum asphyxia.[3] Since in many developing countries there are no facilities to do blood gas measurement, base deficit is not used as part of the definition and only the clinical definition is used. Using the clinical definition alone to describe the incidence of asphyxia may also overestimate the incidence, as infants who do not initiate respiration are not always asphyxiated intrapartum. Assessing the incidence of asphyxia using a combination of clinical definition of need for BMV, Apgar score <7 at 5 minutes and metabolic acidaemia (base deficit >12 mmol/L) is therefore more likely to provide information on the true incidence of asphyxia in any population. Neonates with asphyxia often have ongoing respiratory depression requiring respiratory support in a neonatal intensive care unit (NICU). Where resources are limited, there is competition for these interventions, resulting in rationing and their being offered to infants who are more likely to survive with minimal morbidity. It is therefore important to determine factors that are associated with severe morbidity and mortality, as they could assist in decision-making on who should be offered these limited resources.

In this study we sought to assess the incidence of asphyxia and hypoxic ischaemic encephalopathy (HIE) according to the presence of signs that define HIE 1, 2 and 3 in Sarnat staging (Table 1),[4] infant characteristics and factors associated with severe HIE and mortality in term and near-term neonates diagnosed with asphyxia in a setting where there is a high patient load and resources are limited.

Methods

Study design. A retrospective, descriptive study of neonates born at term or near term (defined as birth weight ≥2 000 g) and admitted with a diagnosis of asphyxia.

Study setting. Chris Hani Baragwanath Academic Hospital (CHBAH), Johannesburg, South Africa, a public government hospital. This hospital serves the population of Soweto and the surrounding areas. Until April 2014, it was the only hospital in Soweto. It is also a referral centre for all clinics conducting births in Soweto and surrounding areas. These clinics conduct about 8 000 births per year and the hospital about 23 000 births per year, with a total of just over 30 000 births per year for the cluster. In 2011, the hospital had two operating theatres for obstetric surgery including caesarean sections (CSs), and 12 NICU beds.

Study population. Infants who were born weighing ≥2 000 g, required resuscitation with at least BMV and were admitted with a diagnosis of asphyxia were included. The study period was from 1 January to 31 December 2011. Infants who died in the labour ward soon after birth were excluded, as their records were either missing or had incomplete information. Infants born before arrival to hospital, twins and infants with severe congenital abnormalities were also excluded.

Table 1. Sarnat and Sarnat[4] classification of HIE

	Stage 1	Stage 2	Stage 3
Level of consciousness	Hyperalert	Lethargic or obtunded	Stuporous
Neuromuscular control			
Muscle tone	Normal	Mild hypotonia	Flaccid
Posture	Mild distal flexion	Strong distal flexion	Intermittent decerebration
Stretch reflexes	Overactive	Overactive	Decreased or absent
Segmental myoclonus	Present	Present	Absent
Complex reflexes			
Suck	Weak	Weak or absent	Absent
Moro	Strong; low threshold	Weak; incomplete; high threshold	Absent
Oculovestibular	Normal	Overactive	Weak or absent
Tonic neck	Slight	Strong	Absent
Autonomic function	Generalised sympathetic	Generalised parasympathetic	Both systems depressed
Pupils	Mydriasis	Miosis	Variable; often unequal; poor light reflex
Heart rate	Tachycardia	Bradycardia	Variable
Bronchial and salivary secretions	Sparse	Profuse	Variable
Gastrointestinal motility	Normal or decreased	Increased; diarrhoea	Variable
Seizures	None	Common; focal or multifocal	Uncommon (excluding decerebration)

Table 2. Incidence of asphyxia according to different definitions of asphyxia (N=21 086 live births)

Definition	Infants with asphyxia, n	Incidence /1 000 live births
Need for BMV at birth	321	15.2
BMV + base deficit >12 mmol/L	261	12.4
BMV + Apgar score <7 at 5 min	218	10.3
BMV, Apgar score <7 at 5 min + base deficit >12 mmol/L	183	8.7

Study procedure. Hospital records of newborn infants who met the inclusion criteria were retrieved and the following data were collected: birth weight, gender, gestational age, growth, Apgar score, resuscitation required, time to spontaneous respiration, arterial blood gas done within the first hour of birth, and the diagnosis of HIE. Maternal records were also reviewed for maternal age, parity, maternal HIV results, antenatal care, maternal disease, mode of delivery, presence of fetal distress or meconium-stained amniotic fluid and cardiotocographic records. Means, standard deviations (SDs), medians and ranges were used to describe continuous variables, while frequencies and percentages were used to describe categorical variables. The χ^2 test or Fisher's exact test and Student's t-test were used to compare the categorical variables and continuous variables, respectively, between infants with no or mild to moderate HIE and those with severe HIE or who died. Differences between the two groups were considered statistically significant at a p-value of <0.05.

The extent of association of different variables with severe HIE or death was reported using odds ratios (ORs) and the precision using 95% confidence intervals (CIs). In order to determine predictors of severe HIE or death, variables with p-values <0.05 on the univariate logistic regression were included in the multivariate logistic regression. STATA version 10.0. was used to perform the statistical analysis.

Approval to conduct the study was obtained from the hospital protocol review committee and the University of the Witwatersrand Human Research Ethics Committee.

Results

Incidence of asphyxia

Of a total of 23 035 liveborn infants, 21 086 weighed ≥2 000 g at birth. Of those weighing ≥2 000 g, 357 had a diagnosis of asphyxia, of whom 321 were admitted and 36 died in the labour ward before admission; since records of the latter were either missing or had incomplete information, they were excluded.

The incidence of asphyxia ranged from 8.7 to 15.2/1 000 deliveries, depending on the definition of asphyxia used (Table 2). Using the definition combining need for BMV, Apgar score <7 at 5 minutes and base deficit >12 mmol/L (representing a truer reflection of intrapartum asphyxia) the incidence was 8.7/1 000 live births, compared with 15.2/1 000 using need for BMV alone.

Maternal characteristics

Demographic and clinical characteristics of mothers giving birth to infants diagnosed with asphyxia are shown in Table 3. Most of the mothers (95.6%) had attended antenatal care. The average maternal age was just over 25 years, and 57.6% were pregnant for the first time. Just over a quarter of mothers (25.9%) were positive for HIV. Among the mothers who had maternal illness recorded, the most common diagnosis was pregnancy-induced hypertension, accounting for 75.9% of mothers with recorded illness and 19.6% of mothers giving birth to infants with a diagnosis of asphyxia. Of babies with asphyxia, 34.3% were born to mothers with meconium-stained amniotic fluid. The mode of delivery was CS in 38.0% of cases, with only 8.7% of deliveries being assisted vaginal deliveries. The most common reason for performing CS in mothers of asphyxiated infants was fetal distress (68.9%) followed by prolonged second stage of labour (7.3%), cephalopelvic disproportion (6.3%) and cord prolapse (4.1%). Electronic monitoring with a

Table 3. Characteristics of mothers who gave birth to infants with asphyxia (*N*=321)

Variables	
Maternal age (years), mean (SD)	25.17 (6.4)
First pregnancies, *n* (%)	185 (57.6)
Positive HIV test, *n* (%)	83 (25.9)
Received antenatal care, *n* (%)	307 (95.6)
Maternal illness, *n* (%)	
Hypertension	63 (19.6)
Other medical conditions	20 (6.2)
Mode of delivery, *n* (%)	
CS	122 (38.0)
Vaginal delivery	171 (43.2)
Assisted vaginal delivery	28 (8.7)
Meconium-stained amniotic fluid, *n* (%)	110 (34.3)
Documented electronic monitoring during labour (CTG), *n* (%)	
Yes	249 (77.6)
No	72 (22.4)
Normal CTG in monitored patients, *n* (%)	132/249 (53)
Abnormal CTG in monitored patients, *n* (%)	117/249 (47)
Late decelerations	90/117 (76.9)
Early decelerations	8/117 (6.8)
Fetal bradycardia	8/117 (6.8)
Beat-to-beat variability	3/117 (2.6)
Suspicious tracing	8/117 (6.8)

Table 4. Characteristics of infants diagnosed with asphyxia (*N*=321)

Variables	
Birth weight (g), mean (SD)	3 084 (448)
Gestational age (weeks), mean (SD)	38.5 (2.2)
Gender, *n* (%)	
Male	192 (59.8)
Female	129 (40.2)
Apgar score at 5 min, *n* (%)	
<5	75 (23.4)
5 - 7	143 (44.6)
≥7	101 (31.5)
Apgar score at 10 min, *n* (%)	
Recorded	233 (72.6)
< 5	19/233 (8.2)
5-7	50/233 (21.5)
≥7	164/233 (70.3)
Not recorded, *n* (%)	88 (27.4)
Resuscitation required, *n* (%)	
BMV only	288 (89.7)
BMV + CC	23 (7.2)
BMV + CC + adrenaline	10 (3.1)
Time to spontaneous breathing (min), *n* (%)	
<5	105 (32.7)
5 - 10	89 (27.7)
10 - 20	42 (13.1)
>20	27 (8.4)
Not recorded	58 (18.1)
pH, mean (SD)	7.09 (0.16)
pH <7.00 , *n* (%)	77 (24.0)
pH 7.00 - 7.25, *n* (%)	182 (56.7)
pH >7.25, *n* (%)	38 (11.8)
PaO_2 (mmHg), mean (SD)	145.63 (79.92)
PaO_2 <50, *n* (%)	16 (5)
PaO_2 50 - 100, *n* (%)	92 (28.7)
PaO_2 >100, *n* (%)	186 (57.9)
$PaCO_2$ (mmHg), mean (SD)	36.66 (16.71)
$PaCO_2$ <35, *n* (%)	170 (53.0)
$PaCO_2$ 35 - 45, *n* (%)	64 (19.9)
Base deficit (mmol/L), mean (SD)	18.09 (5.26)
Base deficit ≤12, *n* (%)	35 (10.9)
Base deficit >12, *n* (%)	261 (81.3)

CC = chest compressions.

cardiotocograph (CTG) was performed in 77.6% of cases; tracings were normal in 53% and abnormal in 47%. In mothers with an abnormal CTG, the common abnormality was late deceleration (76.9%), followed by early deceleration (6.8%) and fetal bradycardia (6.8%).

Infant characteristics (Table 4)
Apgar scores
The average birth weight was 3 084 g and the average gestation 38.5 weeks. Apgar scores were done at 1 and 5 minutes in 99.5% of the infants and at 10 minutes in 72.6%. At 5 minutes 68.0% had an Apgar score of <7. Of infants with a documented Apgar score at 10 minutes, 29.7% had a score of <7.

Resuscitation and time to spontaneous respiration
During resuscitation, the majority (89.7%) of infants diagnosed with asphyxia responded to BMV only, while just over 10% required extensive resuscitation in the form of chest compressions (7.2%) and adrenaline (3.1%). Of the infants who had time to spontaneous respiration recorded, 50.0% took >5 minutes to attain spontaneous respiration.

Blood gases
All infants were resuscitated with 100% oxygen. The majority of infants had blood gas analysis performed within an hour after delivery. The mean partial pressure of oxygen (PaO_2) and partial pressure of carbon dioxide ($PaCO_2$) were 145.6 mmHg and 36.6 mmHg, respectively, suggesting overzealous BMV. Only 24.0% of infants had a pH of <7.00, 57.9% had a PaO_2 of >100 mmHg, 53.0%

had hypocarbia ($PaCO_2$ <35 mmHg), and 81.3% had a base deficit of >12 mmol/L. The lactate level, which is a good indicator of tissue hypoxia, was found to be >7.5 mmol/L in 97.4% of cases in which it was documented.

Incidence of HIE and mortality
The incidence of HIE ranged from 8.5 to 13.3/1 000 live births, depending on the definition of asphyxia used (Table 5). Defining

asphyxia as an Apgar score of <7 at 5 minutes and base deficit of >12 mmol/L, 179 patients (97.8%) developed signs of HIE (according to Sarnat and Sarnat staging[4]), giving an incidence of 8.5/1 000 live births, compared with 13.3/1 000 live births when asphyxia was defined as need for BMV. Among those who developed HIE, 59.0% had moderate to severe HIE. The overall mortality rate was 7.8%, with the rates in infants with HIE ranging from 8.9% to 12.3%, depending on the definition of asphyxia. Only 1.4% of infants with HIE 1 died, as opposed to 7.1% and 62.5% of those with HIE 2 and 3 respectively, defining asphyxia with inclusion of metabolic acidaemia. There were no deaths among those who did not develop HIE. Sixty-four per cent of the deaths were primarily due to HIE, and 16.0% were due to HIE and meconium aspiration syndrome, with or without persistent pulmonary hypertension of the newborn; 24.0% were due to sepsis. Comorbidities in patients with asphyxia included renal

dysfunction in 31.2%, clinically presumed sepsis in 24.6% and positive blood culture in 13.9%.

Comparison of mild to moderate HIE with severe HIE and/or death

Comparison was made between the above two groups for all patients with asphyxia defined as need for BMV. On univariate analysis, there were no statistically significant differences in maternal characteristics and CTG readings between those with no HIE or mild to moderate HIE compared with those with severe HIE or who died. The infant factors that were associated with poor outcome (severe HIE or death) were low Apgar scores at 5 and 10 minutes, need for adrenaline, delay in time to spontaneous respiration, low pH, high base deficit and low bicarbonate. The median Apgar score was lower at 5 minutes (4 v. 6; $p<0.01$) and 10 minutes (5 v. 8; $p<0.01$), time to spontaneous respiration was longer (15.4 minutes v. 6.2

minutes; $p<0.01$), pH was lower (6.9 v. 7.1; $p<0.01$), base excess was higher (22.8 v. 17.5 mmol/L) and bicarbonate was lower (8.9 v. 11.6 mmol/L) in infants with severe HIE and/or who died compared with those with either no HIE or mild to moderate HIE who survived to hospital discharge (Table 6). Factors showing statistically significant differences in univariate analysis were included in a multivariate analysis to determine factors that could predict severity of HIE or death. Only Apgar score at 10 minutes ($p=0.004$), need for adrenaline ($p=0.034$) and bicarbonate levels in the first hour after birth ($p=0.02$) were found to be predictors of severe HIE and/or death.

Severity of different variables and odds of developing severe HIE or death

The odds of having severe HIE or death increased if the Apgar score at 5 minutes was <5 (OR 9.33; 95% CI 2.83 - 34.03); if the Apgar score at 10 minutes was 5 - 7 (OR 3.05; 95% CI 1.08 - 8.62) or <5 (OR 19.1; 95% CI 5.66 - 66.9) compared with a score of >7; if chest compressions were required (OR 4.51; 95% CI 1.06 - 18.0); if adrenaline was required (OR 81.2; 95% CI 13.2 - 647.7); and if time to spontaneous respiration was >20 minutes (OR 27.2; 95% CI 6.89 - 117.4). When arterial blood gas was measured within the first hour of life, the odds of having severe HIE and/or death were increased if the pH was <7 (OR 5.33; 95% CI 1.31 - 25.16); if the PaO_2 was <60 mmHg (OR 9.65; 95% CI 1.87 - 55.12); and if the $PaCO_2$ was >45 mmHg (OR 3.98; 95% CI 1.11 - 15.56) (Table 7).

Table 5. Incidence of HIE and mortality rate in patients with asphyxia, depending on definition used

	Need for BMV			Apgar score <7 + base deficit >12 mmol/L		
	Total N (%)	Incidence /1 000 live births	Died n (%)	Total N (%)	Incidence /1 000 live births	Died n (%)
No HIE	40 (12.5)	-	0 (0)	4 (2.2)	-	0 (0)
HIE 1	131 (40.8)	6.2	2 (1.5)	71 (38.8)	3.4	1 (1.4)
HIE 2	122 (38.0)	5.8	7 (5.7)	84 (45.9)	4.0	6 (7.1)
HIE 3	28 (8.7)	1.3	16 (57.1)	24 (13.1)	1.1	15 (62.5)
All HIEs	281 (87.5)	13.3	25 (8.9)	179 (97.8)	8.5	22 (12.3)

Table 6. Factors associated with severe HIE or death in infants with asphyxia

	No HIE or HIE 1 - 2 (N=256)	HIE 3 and/or death (N=37)	p-value Univariate	p-value Multivariate
Median Apgar at 5 min	6	4	<0.001	0.99
Median Apgar at 10 min	8	5	<0.001	0.004
Need for CCs, n (%)	19/256 (7.4)	4/37 (10.8)	0.36	N/A
Need for adrenaline, n (%)	1/256 (0.4)	9/37 (24.3)	<0.001	0.03
Time to spontaneous respiration (min), mean (SD)	6.17 (5.71)	15.36 (11.93)	<0.001	0.69
pH, mean (SD)	7.11 (0.14)	6.92 (0.18)	<0.001	0.54
PaO_2 (mmHg), mean (SD)	145.25 (76.00)	148.75 (108.35)	0.82	N/A
$PaCO_2$ (mmHg), mean (SD)	34.94 (13.89)	49.91 (27.69)	<0.001	0.33
Bicarbonate (mmol/L), mean (SD)	11.59 (2.84)	8.91 (2.72)	<0.001	0.03
Base deficit (mmol/L), mean (SD)	17.48 (4.88)	22.74 (5.79)	<0.001	0.30
Lactate (mmol/L), mean (SD)	15.90 (4.34)	18.26 (3.68)	0.008	0.61
Glucose (mmol/L), mean (SD)	6.79 (2.30)	6.93 (4.48)	0.82	N/A

CCs = chest compressions; N/A = not applicable.

Table 7. ORs for developing severe hypoxic ischaemic encephalopathy and/or death according to severity of different variables

Variables	OR (95% CI)
Apgar score at 5 min	
>7	Reference
5 - 7	1.79 (0.50 - 7.01)
<5	9.33 (2.83 - 34.03)
Apgar score at 10 min	
>7	Reference
5 - 7	3.05 (1.08 - 8.62)
<5	19.13 (5.66 - 66.89)
Extent of resuscitation	
BMV only	Reference
CCs	4.51 (1.06 - 17.98)
Adrenaline	81.2 (13.17 - 647.7)
Time to spontaneous respiration (min)	
<5	Reference
5 - 10	2.18 (0.55 - 9.24)
10 - 20	3.41 (0.74 - 16.20)
>20	27.19 (6.89 - 117.38)
pH	
>7.25	Reference
7.0 - 7.2	1.48 (0.35 - 7.13)
<7.0	5.33 (1.31 - 25.16)
PaO_2 (mmHg)	
60 - 100	Reference
>100	2.40 (0.63 - 10.74)
<60	9.65 (1.87 - 55.12)
$PaCO_2$ (mmHg)	
35 - 45	Reference
>45	3.98 (1.11 - 15.56)
<35	0.75 (0.19 - 3.07)

CCs = chest compressions.

Discussion

The recent development of induced hypothermia having been reported to reduce the risk of mortality and disability in infants with asphyxia prompted this study to assess the burden of asphyxia (incidence of asphyxia) and the numbers of infants who might require cooling (incidence of moderate to severe HIE) in a setting where there is a high patient load but limited resources. Defining asphyxia as need for BMV, Apgar score <7 at 5 minutes and base deficit >12 mmol/L within an hour after delivery, the incidence of asphyxia was 8.7/1 000 live births. Most studies reporting on the incidence of birth asphyxia in developing countries do not include blood gases, and use only need for assistance with respiration and/or Apgar score <7 at 5 minutes. Using the definition of need for assistance with breathing or Apgar score <7 at 5 minutes, our incidence of asphyxia was 15.2/1 000 or 10.3/1 000 live births, respectively. Whichever definition is used, the incidence of asphyxia in this study is very high compared with that reported in developed countries (~1 - 5/1 000 live births.[5,6] We did not collect data on modifiable factors that may have contributed to this high incidence. A perinatal care survey conducted in South Africa in 2008/9 reported that delay in seeking medical care, inadequate monitoring during labour and lack of facilities were modifiable factors identified in neonates who died from asphyxia.[7] We postulate that these same factors contribute to our high incidence of asphyxia.

The overall incidence of encephalopathy in infants with Apgar scores <7 and base deficit >12 mmol/L was 8.5/1 000 live births, with 60% of cases being due to HIE 2 and 3. The overall incidence reported in this study was higher than the incidences reported from Sweden, a developed country, and Kathmandu in Nepal, a developing country (1.8 and 6.4/1 000 live births, respectively[8,9]), but lower than the 28.1/1 000 reported for Sarlahi, Nepal.[10]

It has been reported that of infants diagnosed with HIE, 15 - 20% will die in the neonatal period and 25 - 30% of survivors will develop permanent neurodevelopmental abnormalities including cerebral palsy.[6] In our study, 62% and 7% of infants with HIE 3 and HIE 2, respectively, died before hospital discharge. Some infants with HIE 2 and most of those with HIE 3 who survive to hospital discharge will have a poor neurological outcome.

The high incidence of HIE in this report is a major concern and highlights the need for improvements in the healthcare system to reduce the incidence of asphyxia and HIE. It is also important to treat infants with moderate to severe HIE with induced hypothermia to reduce neurological disability and mortality.

Predictors of severe HIE and/or death were low Apgar score at 10 minutes, need for adrenaline and low bicarbonate. Shah *et al.*[11] reported similar findings, and that need for chest compressions, high base deficit and delay in onset of respiration beyond 20 minutes in infants with asphyxia were associated with poor outcome; rates of severe adverse effects were 64% if one predictor was present, 76% if two were present and 93% if all three were present. The finding that Apgar score at 10 minutes is a predictor of poor outcome is helpful, as the Apgar score is easy to perform and widely used in labour and delivery rooms in both developed and developing countries. Although the Apgar score has not been helpful in predicting which infants will develop cerebral palsy, especially in the era of induced hypothermia for asphyxia,[12] it has been shown to be very useful in predicting survival in both preterm and term infants. Laptook *et al.*[13] reported that each point decrease in Apgar score at 10 minutes was associated with a 45% increase in the odds of death or disability, and concluded that Apgar score at 10 minutes provided useful prognostic information before other evaluations are available.

The presence of metabolic acidaemia or low bicarbonate is of additional value to the Apgar score in predicting poor outcome, as the Apgar score is often not done in real time and can therefore be unreliable. A base deficit of >16 mmol/L has previously been reported to be a predictor of poor outcome.[10] Casey *et al.*[14] also reported that a combination of low Apgar score (0 - 3) and cord pH of <7.0 increased the risk of mortality in both preterm and term infants, whereas Sehdev *et al.*[15] reported that a base deficit of >16 and a 5-minute Apgar score of <7 together identified 79% of neonates who would develop complications.

Study limitations

The chief limitation of this study is that it is retrospective. The diagnoses of asphyxia and encephalopathy were therefore mainly based on what was recorded in the patient's hospital file. This may have led to overestimation of the true incidence of HIE, especially as some clinical findings could be affected by the experience of a person conducting the examination, e.g. junior doctors commonly labelling infants with asphyxia as having HIE. Our study nevertheless serves to highlight the burden of asphyxia and encephalopathy at CHBAH, which possibly reflects the incidence in other public sector hospitals in South Africa.

Conclusion

The incidences of both asphyxia and HIE are very high at CHBAH. As moderate to severe HIE is likely to be associated with abnormal neurodevelopmental outcomes and disability, factors that contribute to a high incidence of asphyxia must be avoided. Infants at risk of developing encephalopathy must be offered neuroprotective treatment, i.e. induced hypothermia. Where arterial blood gas analysis, NICU facilities and induced hypothermia are not available or are limited, an Apgar score of <5 at 10 minutes and the need for adrenaline can be of assistance in predicting poor outcome.

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