Birth Asphyxia in a Mission Hospital in Benin City, Nigeria.

Alphonsus N. Onyiriuka
Department of Child Health, College of Medical Sciences, University of Benin, Benin City, Nigeria.

Abstract

Background: Birth asphyxia is a leading cause of neonatal morbidity and mortality in Nigeria. Survivors have increased risk of long-term neurologic disability.
Objective: To determine the incidence and document the predisposing factors and outcome.
Methods: In this descriptive (cross-sectional) study at St Philomena Catholic Hospital, the one-and-five-minute Apgar scores of 2,208 live-births were recorded. Those with low Apgar scores (6 at one minute) were studied and their data analyzed.
Results: Birth asphyxia occurred in 83.8 per 1000 live-births with preterm and post-term deliveries, primiparity, grand multiparity, maternal age 19 years or 40 years and pregnancy-induced hypertension being the associated significant factors. Severe birth asphyxia was commonest in breech deliveries. Post-asphyxial seizures occurred in 11.9% of asphyxiated infants with 86.4% of these seizures occurring within the first 12 hours after birth. Birth asphyxia accounted for 25.9% of total neonatal deaths with 58.7% of these deaths occurring within the first 24 hours after birth. The rates of both severe birth asphyxia and deaths were higher in male that female neonates.
Conclusion: Disease-specific burden of birth asphyxia remains enormous and will be alleviated if careful attention is paid to management of labour of high-risk expectant mothers, coupled with availability of personnel skilled in neonatal resuscitation at these deliveries.

Key Words: Birth asphyxia, incidence, outcome.

Introduction

In developing countries, birth asphyxia is the commonest labour room neonatal emergency and one of the leading causes of perinatal morbidity and mortality as well as permanent damage to the brain.[1] Estimates of its incidence in Britain and the United States have varied from 1.2% to 5% of births.[2-4] (and of severe asphyxia 0.4% to 1.6%[4-7]). Its incidence varies with gestational age. Thus, it is reported to occur in 9.0% of infants less than 36 weeks gestation.[8] In a previous study in Benin City, the reported incidence of birth asphyxia (one-minute Apgar Score 6) was 6.3%.[9] Birth asphyxia contributes greatly to neonatal morbidity and mortality. For example, at the University of Benin Teaching Hospital (UBTH) birth asphyxia accounted for 22.8% of neonatal admissions and 28.6% of overall neonatal deaths.[9] It is clear, therefore, that developing countries, such as Nigeria, must develop a realistic perinatal health care programme aimed at reducing the incidence and contribution of birth asphyxia to neonatal morbidity and mortality. For this to succeed, an accurate knowledge of the current magnitude of the problem as well as its predisposing factors is a prerequisite. Analysis of data collected from individual hospitals on birth asphyxia will serve to highlight some of the problems. Also shortcomings in perinatal and neonatal care will become apparent; preventable predisposing factors operative in birth asphyxia will be identified and corrected. To the best of my knowledge, no study has in recent times examined in details the epidemiology of birth asphyxia in Benin City. The purpose of this study is to determine the incidence, predisposing factors and outcome of birth asphyxia in a large busy semi-private health facility.

Patients and Methods

This descriptive (cross-sectional) study was conducted at St Philomena Catholic Hospital (SPCH), Benin City. The hospital has a large maternity unit with a delivery rate of 1,344 per annum.[10] It was chosen for the study because of its central location and its non-selection of cases. The hospital has a Special Care Baby Unit (SCBU) with a capacity for 8 cots and 3 well-functioning incubators with a 24-hour coverage provided by a Consultant Paediatrician (the author, with special interest in Neonatology), 2 Paediatric Senior Medical Officers and 2 Paediatric Medical Officers (with one-year and 3-year post qualification experience respectively). Obstetric care is provided on a 24-hour basis by 2 Consultant Obstetrician and Gynaecologists and 2 Senior Medical officers. The nursing personnel in SCBU consists of a trained Paediatric Nurse (in-charge nurse) and 6 Staff Nurse-Midwives. The hospital has facilities for performing ultrasonography and basic laboratory investigations.

During the 2-year study period, 1st January, 2002 to 31st December, 2003, either the Consultant Paediatrician or the Senior Medical Officer in Paediatrics was present at the delivery of all high-risk infants. They assessed and documented the 1-minute and 5-minute Apgar scores of these babies using the Apgar Scoring System.[11] Similar method of assessment and documentation were used by the trained mid-wife in the case of non-high-risk infants.

Correspondence: *Dr. A. N. Onyiriuka, Department of Child Health, University of Benin Teaching Hospital PMB 1111 Benin City, Nigeria.*
E-mail: *ndidiony2000@yahoo.com*

Each infant was resuscitated in the delivery room as determined by the body's clinical condition.

Resuscitation Procedures in the Delivery Room

The infant is placed on the resuscitation table fitted with a source of radiant heat (two 100 watts electric bulbs), the body was quickly dried of amniotic fluid and blood and the wet linen removed. The infant is place in a head-down position with the neck slightly extended. The airway is cleared by suctioning. Tactile stimulation is then provided. Ventilation with self-inflating ambu-bag with 100% oxygen is then commenced. Most of the infants responded satisfactorily to this mode of resuscitation with spontaneous regular respiration and heart rate greater then 100 beats per minute, following which ventilation was discontinued. If heart rate remained below 80 beats per minute despite adequate ventilation with 100% oxygen, medications such as sodium bicarbonate 2mEq/kg (concentration 0.5mEq/ml) and epinephrine 0.01mg/kg (concentration 1:1000; 0.1mg/ml) were administered intravenously.

Post-Resuscitation Care in SCBU

The infants were transferred to SCBU and their clinical condition fully documented. Intravenous fluid (10% dextrose in water) is set up and given at a rate of 60ml/kg in the first 24 hours after birth. Thereafter, it is changed to 4.3% dextrose in 0.18% saline at a rate determined by the baby's clinical condition. The baby is weighed daily and the weights documented. Strict fluid intake/output chart is maintained. Oxygen and warmth (incubator nursing) are provided as determined by the baby's clinical condition. We routinely use antibiotics (cefotaxime and cloxacillin combined) in view of the unavoidable over handling and manipulations during resuscitative procedures. Furthermore, prolonged labour, difficult delivery, instrumental deliveries and prolonged rupture of membranes all of which are known to predispose to neonatal sepsis are often present. Following delivery and resuscitation, infants whose 1-minute or 5-minute Apgar Scores were 3 or 5 respectively and had seizures were treated with phenobarbitone 20mg/kg stat and subsequently maintained on 8mg/day 8 hourly. Where despite this mode of initial therapy there were protracted seizures for which no aetiologic factor could be identified, diazepam 0.25mg/kg stat was added to the therapy. We try to encourage oral feeding as soon as the baby's clinical condition permits. Initial feeding is usually 5% glucose via naso-gastric tube and then breast milk for subsequent feeds. In conformity with a previous studies[9] and for purpose of analysis of data and comparison of results, birth asphyxia was defined as 1-minute Apgar score of 6.

Statistical Methods

The results were analyzed by determining percentages, ratios and confidence intervals. Chi-square test was used in ascertaining the level of significance of differences, which was set at $p < 0.05$.

Definition of Terms

(i) A preterm infant is one who was delivered before 37 completed weeks of gestation; (ii) a post term infant is one who was delivered after 42 completed weeks of gestation; (iii) a primiparous woman is one whose parity is zero; (iv) a multiparous woman is one whose parity is 1 to 4: (v) a grand multiparous woman is on whose parity is 5; (vi) a teenage mother is one whose age is 19 years; (vii) a booked patient is one who registered for delivery at SPCH and attended antenatal clinic for at least two times; (viii) an unbooked patient is one without prior registration for delivery at SPCH or attended antenatal clinic less than two times at SPCH.

Results

Incidence

There were 2,208 live-births during the 2-year study period. Of these, 185 infants had 1-minute Apgar score of 6, giving an overall incidence rate of birth asphyxia of 83.8 per 1000 live-births or 8.38%. Both maternal age and parity significantly influenced the incidence rates of birth asphyxia (Tables 1 and 2). Infants born to teenage mothers and mothers aged 40 years together had a higher incidence rate (24.6%) compared with their counterparts born to mothers aged 20-39 years. Further details are shown in Table 1. Similarly, infants of primiparous and grand multiparous mothers together had a higher incidence rate (13.0%) than their counterparts born to multiparous mothers (4.7%). The commonest obstetric factor associated with birth asphyxia was pre-eclampsia/eclampsia. Others are shown in Table 3.

Incidence of severe birth asphyxia

Forty one out of 185 infants (22.2%) had an Apgar Score of 3 at 1 minute; 9 (4.9%) others had a 5-minute Agpar Score 5, these were mainly those who were initially moderately or mildly asphyxiated at 1 minute but subsequently deteriorated by 5 minutes; thus a total of 27.0% (50/185) were classified as those who suffered severe birth asphyxia. This incidence varied according to mode of delivery. For instance, incidence rates of severe birth asphyxia per 1000 live-births were as follows: (i) Vaginal breech delivery 90.9; (ii) Caesarean section 49.1; (iii) Vacuum extraction 20.8; (iv) Forceps delivery 19.8; and (v) Spontaneous vaginal delivery; 4.1. Eighty two (44.3%) of asphyxiated infants were delivered by Caesarean section; comprising of 75 (91.5%) emergency Caesarean sections and 7 (8.5%) elective Caesarean sections.

Incidence and gestational period.

Incidence rate of birth asphyxia were as follows: (i) Full-term 5.3% (86/1637); (ii) preterm 14.5% (70/483); and (iii) post-term 33.0% (29/88). Preterm versus full-term $= 46.702$ $P < 0.001$. Post-term versus full-term $= 97.482$ $P < 0.001$. Ten out of the twenty-nine (34.5%) post-term infants had meconium stained fingernails and umbilical cords.

Table 1:
Maternal Age and Birth Asphyxia

Maternal age groups in year	Total no of live births	No of asphyxiated Infants	Incidence per 1000 live births
15-19	96	21	218.7
20-24	756	48	63.5
25-29	783	43	54.9
30-34	399	46	115.3
35-39	148	18	121.6
40	26	9	346.2
Total	**2208**	**185**	**83.8**

Mother's age: 15-19 years versus 20-39 years = 24.742 P<0.001
Mother's age: 40years versus 20-39 years = 25.662 P < 0.001.

Table 2:
Maternal Parity and Asphyxia

Maternal parity	Total no of live births	No of Asphyxiated Infants	Incidence per 1000 live births
0	485	75	154.6
1-4	228	58	47.2
5	495	52	105.1
Total	**2208**	**185**	**83.8**

Para zero versus paras 1-4 = 56.009 < 0.001
Para 5 versus paras 1-4 = 19.734 < 0.001

Sex Ratio
There was no gender difference in the overall incidence rate of birth asphyxia; male to female ratio was 1:1. Of the 50 infants classified as having suffered severe birth asphyxia, 31 (62.0%) were males while 19 (38.0%) were females (95% confidence interval CI = 0.486-0.755). Male to female ratio was 1.6:1. Among the 2,208 total live-births, 1157 (52.4%) were males while the remaining 1051 (47.6%) were females. Sex ratio was 1.1:1 in favour of males.

Booking Status
Incidence rates of birth asphyxia in infants of booked and unbooked mothers were 7.2% (146/2038) and 22.9% respectively (39/170) (= 50.655 p < 0.001). The commonest clinical finding in asphyxiated babies was respiratory distress. Others are shown in Table 4. As shown in Table 5, majority (86.4%) of the babies who had post-asphyxial seizures did so within the first 12 hours after birth.

Neonatal mortality
Birth asphyxia was responsible for 185 (20.9%) of the 886 admissions into SCBU. Twenty-nine neonatal deaths were associated with birth asphyxia which accounted for a neonatal mortality rate (NMR) of 13.1 per 1000 live births; corresponding to 15.7% of asphyxiated infants. The NMR for the same period was 50.7 per live births. Birth asphyxia was responsible for 25.9% (29/112) of overall neonatal deaths. Of the 29 asphyxiated infants who died, 19 (65.5%) were males while 10 (34.5%) were females (95% CI = 0.482-0.828) giving male to female ratio of 1.9: 1.

Common clinical findings in asphyxiated infants who died
Common clinical findings in 29 asphyxiated infants who died were poor suck (71.0%), persistent hypotonia (50.7%), drooling of saliva (37.7%) and seizure (21.7%). Others were cyanosis with oxygen dependence and a history of meconium stained amniotic fluid. As shown Table 6, 58.6% of deaths among asphyxiated babies occurred within the first 24 hours after birth with majority (44.8%) occurring within the first 12 hours after birth.

Discussion

The incidence of birth asphyxia is still high in Benin City with preterm and post-term infants being particularly predisposed. Other important predisposing factors include maternal age (19 years or 40years), parity (primiparity or grand multiparity), absence of antenatal care and presence of pregnancy-induced hypertension (PIH). Birth asphyxia is an important cause of neonatal morbidity and mortality in Benin City with majority of the deaths occurring within the first 24 hours after birth. Poor suck, persistent hypotonia, drooling of saliva and seizures were the common clinical findings in babies who died. Majority of the post-asphyxial seizures occurred in the first 12 hours after birth. Gender differentials were observed in the incidence rates of severe birth asphyxia and neonatal deaths.

The strength of the study lies in the fact that cost is small and there was no loss to follow up. The study also highlighted the magnitude of the problem of birth asphyxia and provided useful hints for anticipation and prevention of its complications.
One limitation of this study is the use of Apgar score in defining birth asphyxia. The Apgar Scoring System[11] though very useful in the measurement of birth asphyxia has its short- comings in that it does not fully define asphyxia.[4,12,13] It is known that factors other than asphyxia may affect the Apgar score of an infant.[12-14] However, in the review by Addy he noted that Apgar score was the basis of many papers on the outcome of birth asphyxia.[15] Furthermore, a previous study in Benin City[9] used Apgar score in defining birth asphyxia. To allow for comparison of results the same definition for birth asphyxia was used in this study. Thus, justifying the use of Apgar score in defining birth asphyxia in the present study.

Table 3:
Obstetric Factors Associated With Birth Asphyxia In 185 Infants

Obstetric factors	No of Asphyxiated Infants	% of total Asphyxiated Infants
Pre-eclampsia and eclampsia	37	20.0
Fetal distress of unidentified cause	33	17.8
Malpresentation	29	15.7
Cephalo-pelvic disproportion	28	15.1
Antepartum haemorrhage	20	10.8
Cord complications	16	8.6
Prolonged ruptured of fetal membrane	7	3.8
Retained second twin	5	2.7
Uterine dysfunction	4	2.2
Congenital malformation (Hydrocephalus)	2	1.1
Others	4	2.2
Total	**185**	**100**

The overall incidence rate of birth asphyxia highlighted in this study is higher than 63.0 per 1000 live-births previously reported by Omene and Diejomaoh in Benin City.[9] Their study was retrospective, therefore some case-records may not be available, even when available, may contain incomplete data and so be left out in the final analysis of data resulting in lower incidence rate.

In consonance with two previous studies,[9,16] preterm and post-term infants are particularly predisposed to birth asphyxia.
However, whether the low Apgar score (birth asphyxia) in preterm infants relates to developmental immaturity rather than fetal distress-related depression around the time of birth is unclear. It is likely that it represents true hypoxia because in the study by Low et al[16] in which fetal acid-base data and cardiotocographic abnormalities were used as basis for defining birth asphyxia, a higher incidence was also observed among preterms. The increased predisposition of post-term infants to asphyxia may be explained by the fact that they may have suffered chronic intrauterine hypoxia as a result of placental insufficiency occasioned by aging placenta thereby making them more vulnerable to asphyxia following the additional stress of labour and delivery. This view is supported by the fact that in 34.5% of our post-term infants there was meconium stained finger nails and umbilical cords, suggesting that they may have suffered chronic intrauterine hypoxia. Low et al[16] have also observed that meconium in amniotic fluid was more frequently seen in their asphyxiated than in their control group.

Of all modes of delivery, vaginal breech delivery was most frequently associated with severe birth asphyxia; further confirming the results of previous studies.[9,16] It is likely that some of these patients may have arrived late in labour to have influenced the choice of mode of delivery since it is known that Caesarean section for breech deliveries have better outcome.[17] Data from the present study showed that the overall incidence rate of birth asphyxia was higher in infants delivered by Caesarean section than in their counterparts delivered by spontaneous vaginal delivery. This is in agreement with data form other studies.[9,16] As indicated in the present report and others,[9,16] majority of these Caesarean deliveries were emergencies for neonatal asphyxiating conditions such as severe pre-eclampsia, eclampsia, obstructed labour, antepartum haemorrhage and cord prolapse; suggesting that the same cause which led to the Caesarean delivery might have caused antepartum/intrapartum asphyxia which manifested at birth.

In this study, as in others, maternal age (19 years or 40 years)[18,19] parity (primiparous and grand multiparous)[16,20] absence of antenatal care[9] and pregnancy-induced hypertension[4,6,9,16,21] were the statistically significant predisposing factors. However, two of these studies[4,6] considered only severe form of birth asphyxia. The contribution of birth asphyxia to neonatal mortality in Benin City has not changed over time. For instance, it accounted for 28.6% of all neonatal deaths at the University of Benin Teaching Hospital (1974-1976).[9] The corresponding figure in the present study was 25.9% with majority of the deaths occurring in the first 24 hours after birth. Comparison was not possible because time of death was not indicated in the previous report.[9] In consonance with previous reports[2,4,6,9,16,18] poor prognostic clinical findings associated with neonatal death in asphyxiated infants include poor suck,

Table 4:
Clinical Findings in 185 Asphyxiated Infants

* Clinical findings	No of cases	% of total Asphyxiated Infants
Respiratory Distress	86	46.5
Apnoeic attacks	10	5.4
Cyanosis requiring continuous oxygen therapy	9	4.9
Hypotonia	32	17.3
Hypertonia	13	7.0
Difficulty in feeding	15	8.1
Drooling of saliva	14	7.6
Convulsions	22	11.9
Jitteriness	4	2.2
Subnormal temperature (35.5^{0}C)	5	2.7

** Some infants had more that one of these clinical findings.*

Table 5:
Age at Onset of Neonatal Seizures in 22 Asphyxiated Infants

Age in hours at Onset of seizures	No of Cases Who had seizures	% of total Asphyxiated infants who had Seizures
6	13	59.1
7-12	6	27.3
13-24	2	9.1
25	1	4.5
Total	**22**	**100**

Table 6:
Age at which Death occurred in 29 Asphyxiated Infants

Age at death (Hours)	No of asphyxiated dead infants	% of total asphyxiated dead infants
6	7	24.1
6-12	6	20.7
13-24	4	13.8
25-36	3	10.3
37-48	3	10.3
49-72	2	6.9
73-169	0	0
169	4	13.8
Total	**29**	**100**

persistent hypotonia, drooling of saliva and seizures. However, the percentages for these clinical findings were different in the various studies.[2,4,6,9,16,18] in one of these studies,[6] as in the present one, majority of these seizures occurred in the first 12 hours after birth. The usefulness of these clinical findings as indicators of occurrence of neonatal deaths in asphyxiated infants is reinforced by the report of Levene et al,[21] who showed that progressive assessment of post-asphyxial neurologic status of the infant improves prognostication when combined with Apgar scores. In constrast to a previous report,[17] gender difference was observed in neonatal mortality rates of severely asphyxiated infants with higher rates in males than females. This gender difference in neonatal mortality rates may be explained by the higher incidence of severe form of birth asphyxia in male than female infants in this study.

The leading role of birth asphyxia as a cause of neonatal morbidity and mortality has not changed over time. This implies that any effort aimed at reducing neonatal morbidity and mortality rates in Benin City (and perhaps other parts of Nigeria) must strengthen strategies for prevention and treatment of birth asphyxia. Some ways of achieving this goal include:

(i) Personnel skilled in neonatal resuscitation should be present at the delivery of all high-risk infants such as preterm and post-term infants as well as those whose mothers have biological and obstetric factors that predispose to birth asphyxia.

(ii) Acquisition of practical skill in neonatal resuscitation should be emphasized in all medical schools and schools of mid-wifery in Nigeria.

(iii) Women in labour whose babies have breech presentation should be offered delivery by Caesarean section especially if another risk factor for birth asphyxia is present.

(iv) Teenagers, elderly women (40 years old) and grand multiparous women should be provided with family planning services to avoid pregnancy.

(v) Since majority of the post-asphyxial seizures and neonatal deaths occurred in the first 24 hours after birth, greater attention, both human and material resources, should be devoted to asphyxiated infants in the first 24 hours after birth to ensure early recognition, prompt and adequate treatment of post-asphyxial complications.

Some unanswered questions and specific areas of future research include:

(i) What is the risk of death or permanent severe disability for the infant with severe birth asphyxia?

(ii) What is the risk of minor disability in survivors of severe birth asphyxia?

(iii) For how long should we persist in attempting to resuscitate a baby who does not breathe at birth?

(iv) How do obstetric factors affect prognosis for the asphyxiated baby?
(v) Can neonatal neurologic examination predict long-term outcome?
(vi) Why is severe birth asphyxia and post-asphyxial neonatal deaths commoner in male than female infants?

Conclusions

The present study has shown that birth asphyxia is an important cause of morbidity and mortality in the first one week after birth. Birth asphyxia, therefore, should be viewed as an acute neonatal emergency requiring intimate co-operation between the paediatrician, obstetrician and the anesthetist.

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