Birth Asphyxia in a Nigerian Mission Hospital in Benin City

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Abstract

Background: Although birth asphyxia is a leading cause of neonatal morbidity and mortality in Nigeria, it has received limited attention in terms of policy and funding priority partly because of lack robust perinatal statistics. Survivors have increased risk of long-term neurologic disability.
Objective: To determine the prevalence of birth asphyxia and document the predisposing factors and outcome.
Methods: In this 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. The associated significant factors were preterm and post-term deliveries, primiparity, grand multiparity, maternal age 19 years or 40 years and pregnancy-induced hypertension. Severe birth asphyxia was commonest in breech deliveries. Post-asphyxial seizures occurred in 11.9% of asphyxiated infants with 86.4% of them 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. Overall case fatality rate was 15.7%. Poor suck, persistent hypotonia, drooling of saliva and seizures were the poor prognostic indicators associated with death. The frequency of severe birth asphyxia as well as deaths were higher in males than females.
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.
Keywords: Birth Asphyxia, Prevalence, Outcome, Nigeria.

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] Although birth asphyxia is arguably the most important avoidable cause of permanent neurological injury affecting the full-term newborn infant,[2] it has not received sufficient attention. For instance, the Millennium Development Goal4(MDG-4) involves reduction of under-five mortality by twothirds. To achieve MDG4 requires a more rapid reduction in the risk of early neonatal death (death in the first week of life) which unfortunately has shown the least decline.[3] Birth asphyxia is a major cause of early neonatal deaths. Despite the fact that the estimated numbers of disabilityadjusted life years (DALYs) for birth asphyxia exceed those due to all immunization preventable childhood diseases,[4] birth asphyxia does not feature on most lists of childhood killer diseases and is not a policy or funding priority. Global estimates for asphyxiarelated neonatal deaths vary from 0.7 to 1.2 million annually.[5] In a previous study in Benin City, the reported incidence of birth asphyxia (one-minute Apgar Score 6) was 6.3%.[6] Birth asphyxia contributes greatly to neonatal morbidity and mortality. For instance, at the University of Benin Teaching Hospital (UBTH), birth asphyxia accounted for 22.8% of neonatal admissions and 28.6% of overall neonatal

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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: 40 years 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

deaths.[6] 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. In Benin City, available studies on birth asphyxia were conducted over 30 years ago with details of its epidemiology lacking. The purpose of this study is to determine the prevalence, predisposing factors and outcome of birth asphyxia in a large, busy, centrally located maternity hospital.

Patients and Methods

This 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.[7] 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), 2 Paediatric Senior Medical Officers and 2 Paediatric Medical Officers (with one-year and 3-year post-qualification experience respectively). Obstetric care was provided on a 24-hour basis by 2 Consultant Obstetrician and Gynaecologists and

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**

2 Senior Medical officers. The nursing personnel in SCBU consisted of a trained Paediatric Nurse (in-charge nurse) and 6 Staff Nurse-Midwives. The hospital has facilities for performing ultrasonography, electrocardiography and basic laboratory investigations.

During the 2-year study period, 1st January, 2003 to 31st December, 2004, 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.[8] Similar method of assessment and documentation were used by the trained mid-wife in the case of non-high-risk infants. Each infant was resuscitated in the delivery room as determined by the baby's clinical condition.

Resuscitation Procedures in the Delivery Room

The infant was 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 was placed in a head-down position with the neck slightly extended. The airway was cleared by suctioning. Tactile stimulation was then provided. Ventilation with self-inflating ambu-bag with 100% oxygen was then commenced. Most of the infants responded satisfactorily to this mode of resuscitation with spontaneous regular respiration and heart rate greater than 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) was set up and given at a rate of 60ml/kg in the first 24 hours after birth. Thereafter, it was changed to 4.3% dextrose in 0.18% saline at a rate determined by the baby's clinical condition. The baby was weighed daily and the weights documented. Strict fluid intake/output chart was maintained. Oxygen and warmth (incubator nursing) were 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.

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.*

Furthermore, prolonged labour, difficult delivery, instrumental deliveries and prolonged rupture of membranes (all of which are known to predispose to neonatal sepsis) were 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/kg/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 encouraged oral feeding as soon as the baby's clinical condition permitted. Initial feeding was usually 5% glucose via naso-gastric tube and then breast milk for subsequent feeds. In conformity with a previous studies[6] 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 was one who was delivered before 37 completed weeks of gestation;
(ii) A post-term infant was one who was delivered after 42 completed weeks of gestation;
(iii) A woman was accepted as primiparous if her parity was zero; multiparous if her parity was 1 to 4; and grand multiparous if her parity was 5 and above.
(iv) A teenage mother was one whose age was 19 years;
(v) A booked patient was one who registered for delivery at SPCH and attended antenatal clinic for at least two times;
(vi) An unbooked patient was one without prior registration for delivery at SPCH or attended antenatal clinic less than two times at SPCH.

Results

Overall Prevalence

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 prevalence 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 were at significantly increased risk of birth asphyxia. Further details are shown

in Table 1. Infants of primiparous mothers were at a significantly greater risk of birth asphyxia compared to those of their multiparous counterparts (p<0.01). The same was true when infants of grand multiparous women were compared with those of their multiparous counterparts (p<0.01) Table 2. The commonest obstetric factor associated with birth asphyxia was pre-eclampsia/eclampsia. Further details are shown in Table 3.

Prevalence 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. The prevalence rate varied according to mode of delivery. For instance, prevalence 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.

Prevalence and Gestational Period.

Prevalence 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 finger nails and umbilical cords.

Sex Ratio

There was no gender difference in the overall prevalence 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) p<0.05. 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

Prevalence 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

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 a Case Fatality Rate of 15.7%. The NMR for the same period was 50.7 per 1000 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) p<0.05, 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, 44.8% of which occurred within the first 12 hours after birth.

Discussion

In Benin city, the prevalence rate of birth asphyxia and its contribution to neonatal mortality has not changed over time, suggesting persistence of the specific perinatal risk factors. Both preterm and post-term infants are at a significantly increased risk. However, whether the low Apgar score (birth asphyxia) in preterm

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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[a]	13	59.1
7-12[b]	6	27.3
13-24[c]	2	9.1
25[d]	1	4.5
Total	**22**	**100**

a+b versus c+d Z-statistic = 3.418 p<0.001

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**

Age at death: ≤ 24 hours versus > 24 hours Z-statistic = 0.876; p>0.05

infants relates to developmental immaturity rather than foetal 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[9] in which foetal 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[9] have also observed that meconium in amniotic fluid was more frequently seen in their asphyxiated than in their control group.

Other statistically significant predisposing factors were maternal ages (≤ 19 years or ≥ 40 years), parity (primiparity and gund multiparity), absence of antenatal care and hypertension in pregnancy. Early pregnancy (before the age of 20 years) or late pregnancy (after the age of 35

years) has been identified as a risk factor for development of hypertension disorder in pregnancy,[11-14] which in itself predisposes to birth asphyxia. Teenage mother are still growing, and so, may have inadequate pelvis, leading to birth asphyxia. The higher frequency of birth asphyxia in infants born to mother aged 40 years and above may partly be due to relative secondary contracture of their pelvis and reduce elasticity of the pelvis ligaments.

Of all modes of delivery, vaginal breech delivery was most frequently associated with severe birth asphyxia. It is likely that some of these patients may have arrived too 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.[9]

Majority of the post-asphyxial seizures and neonatal deaths occurred in the first 24 hours after birth, suggesting that there is a critical period when both human and material resources should be deployed to achieve better perinatal outcome. Poor prognostic clinical findings associated with early neonatal deaths in asphyxiated infants included poor suck, persistent hypotonia, drooling of saliva and seizures. Prevalence of severe birth asphyxia and the occurrence of neonatal death were higher in males than females. Frequency of occurrence of birth asphyxia was significantly higher among babies born to unbooked mothers compared to their counterparts born to booked mothers. A possible explanation is that majority of the unbooked mothers were high risk groups referred from other peripheral health institutions with either antepartum or intrapartum complications of pregnancy, which themselves predispose to birth asphyxia.

The strength of the study lies in the fact that cost was relatively small and there was no loss to follow up. The study highlighted the magnitude and the persistent nature of the problem of birth asphyxia in Benin City and provided useful hints for anticipation and prevention of its complications.

One limitation of this study was the use of Apgar score in defining birth asphyxia. The Apgar Scoring System,[8] though very useful in the measurement of birth asphyxia, has its shortcomings in that it does not fully define asphyxia.[15-17] It is known that factors other than asphyxia may affect the Apgar score of an infant.[16-19] However, in the review by Addy he noted that Apgar score was the basis of many papers on the outcome of birth asphyxia.[20] Furthermore, a previous study in Benin City[6] used Apgar score in defining birth asphyxia. To allow for comparison of results, the same definition was used for birth asphyxia in the present study. Thus, justifying the use of Apgar score in defining birth asphyxia in the present study.

The overall prevalence 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.[6] Since their study was retrospective, some case-records may not have been available. Even when available, may contain complete data and so be left out in the final analysis of data resulting in a lower prevalence rate.

In consonance with previous studies, statistically significant predisposing factors to birth asphyxia included prematurity,[6,9] post maturity,[6] maternal age (≤ 19 years or ≥ 40 years),[10-12] parity (primiparity and grand multiparity), [9,13] absence of antenatal care[6] and hypertensive disorders in pregnancy.[9,21]

The contribution of birth asphyxia to neonatal mortality in Benin City remains unchanged over time. It accounted for 28.6% of all neonatal death in a previous study conducted between 1974 and 1976[6] and 25.9% in the present study. The Case Fatality Rate in the present study was 15.7% compared to 20.8% reported from Ilesa.[22] The difference may be explain on the basis of difference in study population. The Ilesa study involved only infants with severe birth asphyxia but the present study involved all categories of birth asphyxia. In keeping with previous reports, [6,9,11,15,23] poor prognostic clinical findings in the present study included poor suck, persistent hypotonia, drooling of saliva and seizures.

In contrast to a previous report, gender difference was observed in neonatal mortality rate of severely asphyxiated infants with a significantly higher rate in males than females. The higher frequency of occurrence of severe form of birth asphyxia in males compared to females in the present study may partly explain the higher death rate in males. The study in Ilesa also reported that males tended to suffer severe birth asphyxia more often than females.[22]

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) 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.

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

(iii) 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.

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

(v) 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.

This study raises these potential areas for future research:

1. How do obstetric factors affect prognosis for the asphyxiated baby?
2. Can early neonatal neurologic examination predict long- term outcome?
3. Why is severe birth asphyxia and post-asphyxial neonatal deaths commoner in male than female babies?
4. What other factors affect the efficiency of perinatal health care delivery in Benin City in view of no change over time in prevalence and mortality rates.

Although some changes in political and socio-economic circumstances were witnessed in Nigeria, the high prevalence rate of birth asphyxia and the associated neonatal mortality have remained unchanged over time.

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