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Analysis of 226 Asphyxiated Newborn Infants at the University of Benin Teaching Hospital (1974 - 1976)

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Summary

Omene, J. A., and Diejomaoh, P. M. E. (1978). *Nigerian Journal of Paediatrics*, **5** (**2**), 25. **Analysis of 226 Asphyxiated Newborn Infants at The University of Benin Teaching Hospital.** Two hundred and twenty-six asphyxiated infants with low (1 minute) Apgar scores were analysed. The incidence of asphyxia was 63 per 1000 live births. It was an important cause of neonatal mortality accounting for 28.6 per cent of neonatal deaths during the study period. Pulmonary pathology was responsible for asphyxia in all the small low birthweight infants, while in the normal birthweight infants the common factor was an extra-pulmonary pathology incompatible with extra-uterine life. Abnormal presentation, cephalo-pelvic disproportion, uterine dysfunction and ante-partum haemorrhage were the major obstetric causes of asphyxia. The modes of delivery frequently associated with severely asphyxiated infants were breech and caesarean section. The most important clinical findings were hypotonia, hypothermia, absence of the Moro reflex and apnoeic attacks, while hypoglycaemia was an important complication.

Advances in obstetrics and neonatal care in developed countries have led to the reduction of perinatal mortality to a minimum. It has also become clear that perinatal events profoundly influence subsequent intellectual performance (Hadberg, Hadberg and Olow, 1975). It seems therefore appropriate to use perinatal morbidity and its sequelae for evaluating maternal and neonatal care. Birth asphyxia has long been recognised as an important cause of brain damage (Griffiths, and Lawrence 1974; Hadberg, Hadberg and Olow, 1975). The purpose of the present study was to determine the frequency, clinical features, predisposing factors, complications and necropsy findings in neonatal asphyxia in the University of Benin Teaching Hospital (UBTH).

Materials and Methods

Infants described in this study were delivered in the department of Obstetrics and Gynaecology (UBTH) between April 1st, 1974 and March

31st, 1976. During this two-year period, paediatricians and paediatric resident staff were present at the delivery of all high risk infants, such as low birthweight babies; infants of toxaemic or diabetic mothers, emergency and elective caesarean sections and foetal or maternal distress.

All high risk infants were assessed, using the Apgar scoring system (Apgar, *et al.*, 1958) at 1 and 5 minutes respectively . Infants with low Apgar scores were resuscitated as follows:

(*i*) Air-way was cleared and ventilation was accomplished by head extension, lateral positioning and face-mask positive pressure ventilation as described by Higgins (1968).

(*ii*) If the infant improved as shown by increased heart rate to 120 per minute, good muscle tone and less cyanosis, mask ventilation was continued until spontaneous respiration was established.

(*iii*) If there was no clinical improvement after three minutes of face-mask breathing, endotracheal tube was inserted to ensure adequate oxygenation with hand-assisted ventilation. The movement of the thorax was observed and both lung fields were auscultated for adequate breath sounds.

(*iv*) If there was still bradycardia despite the above measures, external cardiac massage was started and the infant was re-evaluated by Apgar score at five minutes.

(*v*) If the Apgar score improved but the heart rate was less than 80 per minute, sodium bicarbonate (3mEq per kg) was given slowly through an umbilical venous catheter, after dilution with equal amount of 10 per cent glucose solution.

(*vi*) In most cases marked improvement was observed after step V. However, in some instances, bradycardia persisted (Heart rate less than 80 per minute). In such cases, 1 ml of 1:1000 adrenaline diluted with 10 ml of 10 per cent dextrose solution was given intravenously while monitoring the heart rate.

(*vii*) Four ml per kg of 25 per cent glucose solution was then administered as intravenous bolus injection. This was then followed by 10 per cent dextrose drip (85 ml per kg per day).

(*viii*) This rescuscitative procedure was carried out under radiant heaters in order to maintain the skin temperature at 36.5^0C.

(*ix*) The infants were transferred to the Special-care-baby unit (SCBU) when they were clinically stable. Portable x-ray films were taken within 6 hours of admission to localise the proper positioning of the endotracheal tube and the umbilical catheter. Gestational age was estimated using physical characteristics (Usher, Mclean and Scott, 1966) and neurological evaluation carried out as described by Dubowitz, Dubowitz and Goldberg (1970).

Results

There were 3,550 live births during the 2 year period. Of these, 226 infants had 1 - minute Apgar score of 6 or less, giving an incidence of neonatal asphyxia of 63 per 1,000 live births. Neonatal asphyxia was responsible for 226 (22.8 per cent) of the 991 admissions into the SCBU. There were 26 neonatal deaths associated with asphyxia which accounted for a neonatal mortality rate of 7.3 per 1,000 live births or 11.5 per cent of asphyxiated babies. The neonatal mortality rate for the same period was 25.6 per 1,000 live births. Asphyxia was responsible for 26/91 (28.6 per cent) of the overall neonatal deaths; the corresponding figure for 1973-74 was 18/31 (58 per cent).

Table I shows the clinical findings in the 226 asphyxiated infants. Apnoeic attacks, lethargy and feeding difficulties were common during the first 24-72 hours. Four infants were tube-fed for periods varying between one and two weeks.

The neurological findings were mainly hypotonia, hypertonia, absence of Moro reflex, neural-hyperexcitability and convulsion. Nine of the 26 (34.6 per cent) infants that died had persistent hypotonia.

TABLE I

Clinical findings in 226 Asphyxiated Infants

Findings	*No. of Cases*
Hypotonia	61
Hypothermia (Temp. ⩽ 35°C)	50
Difficulty in feeding	40
Absent Moro reflex	39
Apnoeic attacks	38
Lethargy	13
Neural hyper-excitability (Twitching)	12
Convulsion	2

Table II shows the complications found in 76 of these asphyxiated infants. Metabolic disorders notably hypoglycaemia, were most frequently encountered. Pneumothorax and infection were possibly due to the resuscitative procedure rather than asphyxia *per se*. The fractured femur was related to difficult delivery. The anaemia was due to inadvertent blood loss during umbilical vessel catheterisation.

Table III illustrates the frequency of asphyxia associated with different modes of deliveries. There were 2,843 spontaneous vaginal deliveries; 67 (2.4 per cent) of these were asphyxiated and 14 (0.5 per cent) had Apgar score of 3 or less. Of the 330 infants delivered by caesarean section, 99 (30 per cent) were asphyxiated and 15 (4.5 per cent) were severely depressed. Twenty-one of the 138 breech deliveries were asphyxiated and 9 (6.5 per cent) had Apgar score of 3 or less.

TABLE II

Complications in 75 Asphyxiated Infants

Complication	*No. of cases*	*Per cent of Total*
Hypoglycaemia	28	37.4
Anaemia	14	18.7
Jaundice (Bilirubin > 15mg%)	10	13.3
Hypocalcaemia	7	9.4
Infection	4	5.4
Pneumothorax	3	0.4
Hyponatraemia	3	0.4
Necrotising enterocolitis	2	2.6
Intra-cranial haemorrhage	2	2.6
Hypernatraemia	1	1.3
Fracture of femur	1	1.3
Total	75	100.0

TABLE III

Mode of Delivery and Percentage Frequency of Asphyxia

Mode of delivery	*Percentage of asphyxia (Apgar ⩽6)*	*Percentage of severe asphyxia (Apgar 1–3)*
Caesarean section	30.3	4.5
Breech	21.0	6.5
Forceps	14.7	2.3
Vacuum	11.5	2.1
Spontaneous vertex delivery	2.4	0.5

Other factors associated with asphyxia are summarised in Table IV. Table V illustrates the method of resuscitation of the 226 asphyxiated infants. Forty of the 46 infants (87 per cent) with severe asphyxia had endotracheal intubation while only 35 (19 per cent) of the 180 with mild to moderate asphyxia were intubated.

Death within the first 72 hours of life was related to low Apgar score at 5 minutes. Thus eleven of the 26 infants (42 per cent) with a five-minute

TABLE IV

Obstetric factors associated with Asphyxia in 266 Infants

Factor	*No. of Cases*
Primiparity	74
Grand multiparity	51
Cephalopelvic disproportion	29
Breech	29
Foetal distress	29
Pre-eclampsia and eclampsia	25
Uterine dysfunction	22
Multiple births	19
Anterpartum haemorrhage	12
Prolonged rupture of membranes	9
Cord complication	5
Others	14

TABLE V

Apgar Score and Methods of Resuscitation of 226 Asphyxiated Infants

		Method of resuscitation		
Apgar score	*No. of infants*	*None*	*Oxygen given by mask*	*Endotracheal intubation*
1–3	46	0	6	40(87)*
4–6	180	26	119	35(19.4)*
Total	226	26	125	75

*Numbers in brackets represent percentage of asphyxiated babies that required endotracheal intubation.

Apgar score of 6 or less died within the first 72 hours of life, whereas only two (15.4 per cent) asphyxiated infants with 5 minutes Apgar score above 6 died within the same period.

Ten (38 per cent) of the 26 infants who died from asphyxia were subjected to necropsy. The important findings are shown in Table VI. Pulmonary pathology was responsible for asphyxia in all the small low birthweight infants, while in the normal birthweight infants the common factor was an extra pulmonary pathology incompatible with extra-uterine life.

TABLE VI

Necropsy findings in Ten Infants with Respiratory Distress Syndrome

Case No.	*Birthweight (Kg)*	*Gestational age (wks)*	*Necropsy findings*
1	0.8	27	Atelectatic lungs
2	1.0	30	Pulmonary haemorrhage
3	1.05	30	Pulmonary haemorrhage
4	1.05	28	Atelectatic lungs
5	1.1	28	Atelectatic lungs
6	1.2	35	Pulmonary haemorrhage
7	3.1	38	Congenital heart disease
8	3.2	40	Pneumothorax
9	3.55	39	Tentorial tear and intracranial haemorrhage
10	3.55	40	Right-sided diaphragmatic hernia with hypoplastic lung

Discussion

The incidence of asphyxia as reported by various workers varies widely and depends largely on the quality of the obstetric and neonatal care. Brown, Parvis and Forfar (1974) reported an incidence of 54 per 1,000 live births at the Simpson Memorial Maternity Pavillion, Edinburgh. Versluys and Casson (1975) found an incidence of 62 per 1,000 at the Kilimanjaro Christian Medical Centre in Northern Tanzania. Although a comparable rate of 63 per 1,000 live births was found in the present study, the Tanzanian neonatal mortality rate of 15 per 1,000 was twice as high as that found in our series.

Thirty per cent of the infants delivered by caesarean section were depressed as compared to 21 per cent delivered by breech. However, breech delivery was more commonly associated with severe asphyxia (6.5 per cent). The reason may be due to poor case selection for breech delivery and/or inexperience on the part of the operator. Most of the infants delivered by caesarean section were emergency deliveries that were associated with asphyxiating conditions

such as prolonged labour, placenta praevia and malpresentations (transverse lie and breech).

An early indicator of foetal distress is the abnormality of the foetal heart rate. In the present study, foetal distress was detected clinically in only 13 per cent of the asphyxiated infants. This compares poorly with the 43 per cent rate of diagnosis reported by Franklin, *et al.* (1964). This finding suggests inadequate monitoring of patients during labour thus resulting in failure to recognise features of foetal distress.

During the study period when interest in neonatal resuscitation was intensified, neonatal mortality due to asphyxia declined by approximately 30 per cent, thus lending support to the evidence that artificial ventilation and intravenous administration of alkali together with dextrose solution, decrease the incidence of cardiovascular collapse as well as increase the survival rate and reduce brain damage (Adamson, *et al.*, 1963; Adamson, *et al.*, 1964; Holowach-Thurston, Haubart, and Jones, 1973).

Asphyxia rapidly depletes the liver of its glycogen stores; hence depressed infants become more vulnerable to hypoglycaemia (Hull, 1971). Twenty-eight infants in our series were hypoglycaemic. It is therefore suggested that blood glucose level be determined on all asphyxiated infants and intravenous dextrose solution be administered during resuscitation in the delivery room.

The present study has shown that asphyxia is an important cause of neonatal mortality at UBTH. It accounted for 28.6 per cent of the neonatal deaths during the study period as compared to 58 per cent prior to the period (1973–74) when paediatricians and paediatric resident staff were not always present at the delivery of high risk infants. Because of the serious neurological deficits consequent to perinatal asphyxia (Hadberg, Hadberg and Olow 1975), asphyxia should be viewed as an acute neonatal emergency that calls for the intimate co-operation between the paediatricians, obstetricians and the anaesthetists.

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