

## **NEONATAL ASPHYXIA AND ITS MANAGEMENT**

**EFFIE G. LIABUNYA**

Maternal and Child Health Department, Kamuzu College of Nursing,  
University of Malawi, P/Bag 2, Lilongwe, MALAWI

### **ABSTRACT**

This paper highlights the pathophysiology, signs and symptoms, risk factors, methods of detecting, and prevention of neonatal asphyxia. The management of asphyxia has been discussed. This information can assist those caring for asphyxiated neonates to provide the best possible care quickly.

### **INTRODUCTION**

To begin life as an individual, the neonate must make an abrupt transition from intrauterine to extrauterine life. The first moments of this transition are crucial in that, the baby must establish pulmonary ventilation in conjunction with circulatory changes. Major problems such as asphyxia are thus likely to occur during this period, particularly if there is abnormal transition.

Each newborn baby is subjected to some degree of asphyxia at birth, though some may not be pathologic in origin (Parer and Livingston, 1990). Failure to initiate breathing within 60 seconds of delivery is evident in 5.7% of all deliveries, and 13.6% of all newborn babies require some kind of resuscitation at birth (Roy and Bathers, 1990). Consequently, every newborn has the right to have resuscitation performed with a high level of competence (Bloom and Cropley, 1987).

The way an asphyxiated neonate is managed during the first minutes of life can have consequences over an entire life. If properly done, resuscitation can reduce neonatal morbidity and mortality rate. There is, therefore, a need to understand the pathophysiology, signs and symptoms, and identify the risk factors to asphyxia in order to manage it successfully.

### **PATHOPHYSIOLOGY**

The word asphyxia comes from Greek and means "pulseless". Nevertheless, asphyxia has the pathologic meaning of insufficient or absence of exchange of respiratory gases, leading to failure of tissues to receive or utilize oxygen.

Roy and Bathers (1990) outlined changes that occur during asphyxia. Firstly, there are a brief series of respiratory efforts lasting up to a minute during which the heart rate and blood pressure rise, the partial pressure of oxygen ( $\text{paO}_2$ ) falls and that of carbon dioxide ( $\text{PaCO}_2$ ) rises. This is followed by a period of apnoea. During this 'primary apnoea', the heart rate falls, the blood pressure rises further then steadily drops, and the  $\text{PaCO}_2$

and pH continues to rise. The baby is cyanotic, but may have some muscle tone and circulation is adequate. This phase was previously referred to as '*asphyxia livida*'.

The next phases are gasping and secondary apnoea. During the gasping phase, the baby takes irregular gasps of steadily decreasing frequency until the last gasp is taken. Following this, the baby goes into the phase of secondary apnoea, in which he/she will not spontaneously gasp. The heart rate and blood pressure continue to drop, there is circulatory collapse and the baby is in a state of shock. The skin is pale grey ('*asphyxia pallida*'), and the baby becomes limp. The PaO<sub>2</sub> is low, the PaCO<sub>2</sub> is increased, and pH may be below 7.0, due to combined respiratory and metabolic acidosis.

There are also a number of cardio-respiratory changes occurring during hypoxia or asphyxia. Initially there is selective vaso-constriction in some organs and vasodilation in others, so as to increase blood flow to 'priority' organs such as the brain, and the heart. When asphyxia becomes severe, there is intense vaso-constriction in all vascular beds, leading to reduction in blood flow to all organs and tissues.

The decrease in oxygen to tissues gives rise to a number of potential problems such as respiratory distress, meconium aspiration, heart muscle damage, kidney damage, disseminated intravascular, necrotizing enterocolitis and brain damage. When the brain is deprived of oxygen a series of biochemical and mechanical events occur. These include loss of spontaneous electrical activity, and rapid decrease of glucose and glycogen stores. The water content of the brain increases leading to cerebral edema, which further impairs blood flow. This impairment may have long term consequences such as mental retardation.

## SIGNS AND SYMPTOMS

Amiel-Tison and Ellison (quoted by Rowe, 1990) classified asphyxia into three stages. In the first stage the neonate is irritable, has abnormal muscle tone, poor head control, and exaggerated stretch reflexes. These symptoms usually disappear within first week of life and do not normally lead to long term impairment.

In the second stage the neonate shows signs of central nervous system depression. The baby is lethargic, or may be in a light comatose state, has diminished stretch reflexes, poor suckling and swallowing reflexes, is hypotonic and may have seizures.

In the third stage the baby is deeply comatose, is hypotonic and has a weak or absent stretch reflexes. Respiratory effort is weak, with periods of apnoea. The baby has frequent seizures that respond poorly to any interventions.

## RISK FACTORS AND THEIR PREVENTION

Most of the risk factors leading to neonatal asphyxia are treatable and can be prevented by anticipating the birth of an infant likely to develop asphyxia at birth. This can be

achieved by identifying those factors that would cause an abnormal transition. The adaptation at birth is often influenced by events that occur during pregnancy and more so, during labour and delivery.

Table 1. Neonatal asphyxia risk factors (Bloom and Cropley, 1987).

Antepartum	Intrapartum
Anaemia	Abnormal presentation
Antepartum haemorrhage	Elective or emergency cesarean section
History of previous stillbirth	Foul smelling meconium/amniotic fluid
Hydro-amnios	Foetal distress and prolapse of cord
Chronic pregnancy-induced Hypertension	Intrapartum haemorrhage ( <i>abruptio</i> and <i>placenta previa</i> )
Maternal diabetes	Narcotics administered to the mother within 2-4 hours of delivery
Maternal drug abuse	Premature labour
Maternal infections	Prolonged first/second stages of labour
Multiple gestation	Ruptured membranes longer than 24 hrs
Oligohydro-amnios	Uterine tetany
Post-term gestation	
Size-dates discrepancy	

These factors can predispose the baby to hypoxia or asphyxia at birth, hence the need for careful monitoring of fetus during pregnancy, labour and delivery. However, identification of fetuses at risk of asphyxia is often difficult. Only 20% of patients develop risk factors that initiate careful fetal monitoring and nearly 50% of fetal deaths will occur in the "low risk" group (Jacobs and Phibbs, 1989). Thus particular attention must be paid to all pregnancies. The process of labour and delivery is, in itself, traumatic.

Clinically, asphyxia causes the fetus to have tachycardia, loss of baseline variability, late decelerations or prolonged bradycardia. Therefore, fetal well being in labour can be assessed by using the tradition methods such as, auscultation of fetal heart using a fetal stethoscope, and/or checking for presence of meconium stained amniotic fluid, particularly in cephalic presentations, and observing for excessive fetal movements.

Recent developments in the assessment of fetal well being have included electronic fetal heart pattern monitoring by external or internal means. Analysis of fetal capillary blood pH and umbilical cord blood is also another method of assessing fetal asphyxia. Normal fetal blood pH ranges from 7.30 to 7.35. However, in labour, fetal blood pH greater than 7.25 is considered normal, while pH greater than 7.20 during first stage of labour is suggestive of asphyxia (Gilstrap *et al.*, 1988; Silverman and Suidan, 1985).

The Apgar score is the most commonly used clinical indicator to assess whether an infant has suffered asphyxia. The score was proposed by an American anesthesiologist Dr.

Virginia Apgar in 1953 (Kruppel and Drukker, 1986). The score grades five clinical parameters which are heart rate, respiration, skin colour, muscle tone, and reflex/activity. The individual scores of 0 - 2 for each parameter are accorded at one and five minutes and a total score of 0 - 10 is given at each time (Table 2).

Table 2. Apgar scoring system

Indicator	Characteristics at Apgar Scores of		
	0	1	2
Heart rate (beats/minute)	Absent	< 100	> 100
Respiratory effort	Absent	slow, irregular, weak cry	good, vigorous, cry
Skin colour	blue/pale	body pink, extremities pale	body and extremities pink
Muscle tone	flaccid, limp	some flexion of extremities	good flexion, active motion
Reflex activity/Irritability	no response	weak cry or grimace	vigorous cry, cough, sneeze

An Apgar score of 8 - 10 denotes no asphyxia, 5 - 7 mild asphyxia, 3 - 4 moderate asphyxia, and between 0 - 2 severe asphyxia (Gardner, 1989). Jacobs and Phibbs (1989) and Blackman (1989) argued that although an Apgar score is an excellent and useful tool in assessing the degree of asphyxia, it has serious limitations. For example, it can often be affected by maternal medications, gestational age, and possible human errors. These factors must, therefore, be considered when interpreting the Apgar score.

Using the acid-base status of neonate's blood has been advocated as a better reflection of the degree of asphyxia. This method is also questionable since some newborns may have a good Apgar score but the pH may be lower than 7.20, or have a low Apgar score but with a normal pH. Using both methods for same cases in defining and diagnosing asphyxia will give a more reliable assessment.

## MANAGEMENT OF ASPHYXIA

Once asphyxia is present its management is resuscitation initiated immediately without waiting for the one minute Apgar score. The goal is to provide an adequate airway and expansion of the lungs, decrease the pCO<sub>2</sub> and increase the pO<sub>2</sub> through ventilatory support, and support adequate cardiac output. Minimize oxygen consumption by reducing

heat loss through provision of a neutral thermal environment, (Rowe, 1990).

Immediately after delivery the baby should be placed under a radiant warmer, dried, and covered to prevent heat loss by evaporation. Secondly ensure a patent airway by positioning the baby on his/her back or side in a slight Trendenburt position with the neck slightly extended. If the baby has copious secretions turn head to one side to allow drainage of secretions. Suction the mouth and nose. If the baby does not initiate breathing soon after birth, provide tactile stimulation by gently flicking the soles of the feet or rubbing the baby's back.

The next step in the resuscitation process will depend on the evaluation of the baby's respiration effort, heart rate and the skin. In mild to moderate asphyxia, the baby is generally in primary apnoea. Circulation is unimpaired, and probably there is some muscle tone. The baby may usually respond to simple resuscitation measures such as superficial tactile stimulation. Administer free flow oxygen 100 % at 5 litres per minute, or 40 % when an Ambu bag is used. This may help initiate breathing or improve ventilation in those babies with apnoea or hypo-ventilation. It also increases the inspired oxygen concentration (Roy and Bathers, 1990). The rise and fall of the chest is observed for adequacy of ventilation. When ventilation is adequate, with each inspiration, bilateral breath sounds are audible on auscultation and the skin is pink.

If, however, colour and heart rate fail to respond to ventilatory efforts, the baby may be severely asphyxiated. In severe asphyxia, the infant is in secondary apnoea, shocked, and flaccid. The baby requires immediate vigorous expert resuscitation. This requires a team approach with electronic monitoring and intensive care. Establish positive pressure ventilation immediately via an endotracheal tube (ETT). If the midwife is not very skilful in inserting an endotracheal tube, use a bag and mask to ventilate with 100 % oxygen. The ventilation rate, oxygen flow rate and assessment of sufficient ventilation should be carried out every 15-30 seconds.

The second urgent resuscitative measure is the external cardiac massage (ECM). This should be initiated if the infant's heart rate is less than 60 beats per minute or inaudible. If the baby is not responding to adequate ventilation and cardiac massage, epinephrine (adrenaline chloride) 1:10,000 at a dose of 0.1 - 0.3 ml/kg should be administered rapidly intravenously. This helps to stimulate the heart and increases the strength and rate of cardiac contraction.

Volume expanders are also used to counteract the effect of hypovolemia by increasing vascular volume and improving tissue perfusion. Commonly used volume expanders include whole blood (O negative blood cross-matched with mother's blood), 5% albumin, normal saline or Ringers lactate. This is given at 10 ml/kg body weight intravenously over a period of about 5 - 10 minutes.

The next intervention will include correction of acidosis with sodium bicarbonate. This is necessary because it is likely that there may be an associated metabolic acidosis if there is

no quick response to adequate ventilation. The initial dose of sodium bicarbonate is 2 mEq/kg, of 0.5 mEq/ml concentration, which equals a 4.2% solution. This is given intravenously over a period of 2 minutes.

## CONCLUSION

Successful resuscitation with establishment of spontaneous respiration, normal heart rate, and correction of metabolic acidosis will prevent potential problems associated with asphyxia. Continued support to maintain adequate oxygenation is vital in preventing cerebral edema, which can lead to permanent brain damage and increased morbidity and mortality in the neonates.

## REFERENCES

- Blackman, J., (1989). The relationship between inadequate oxygenation of the rain at birth and developmental outcome. *Topics in Early Childhood Special Education (TECSE)*, 9 (1) : 1 - 13.
- Bloom, R. and Cropley, C. (1987). *Textbook for Neonatal Resuscitation*. Dallas, Texas. American Heart Association.
- Jacobs, D. and Phibbs, R. (1989). Prevention, recognition and prevention of perinatal asphyxia. *Clinics in Perinatology*, 16 (4) : 785 - 807.
- Gardner, M., (1989). *Handbook of Neonatal Intensive Care*. (2nd Edition), St. Louis, The Mosby Co.
- Gilstrap, L., Levano, K., Burris, J., Williams, H. and Little, B., (1988). Diagnosis of Birth Asphyxia on the basis of fetal pH, Apgar score and newborn cerebral dysfunction. *American Journal Obstetrics and Gynaecology* 160 (3) : 608 - 614.
- Mcghic, B. (1983). Neonatal Asphyxia. *Nursing Times*, 29 (270) : 34 - 38. (Not in Text)
- Parer, J., and Livingston, E. (1990). What is Fetal Distress? *American Journal of Obstetrics and Gynaecology*, 162 : 1421 - 1424.
- Roy, R., and Batharas, F. (1990). The Melbourne Chart: A logical guide to neonatal resuscitation. *Anaesthesia Intensive Care*, 18 (3) : 348 - 357.
- Rowe M. (1990). Asphyxiated Infants: Pathophysiologic Consequences, Parenting and Nursing Management. *Neonatal Network*, 4 : 7 - 10.
- Silverman F., Suidan J., and Wasserman J., (1985). The Apgar Score: Is it enough?, *Obstetrical Gynaecology* 66 : 331 - 336.