

SUMMARY

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Perinatal asphyxia is the failure of a newborn infant to establish adequate ventilation at birth with subsequent hypoxaemia, respiratory and metabolic acidosis, cardiovascular deterioration with hypotension and bradycardia, and central nervous system depression.

In the great majority of cases the causes of neonatal asphyxia have been operating before birth. Commonly asphyxia may be caused by other conditions operating during the birth process itself. Infrequently it may appear for the first time after birth.

Slowing and irregularity of the fetal heart rate or the intrauterine passage of meconium are warning signs of fetal distress. The two most commonly employed techniques for monitoring fetal well being during pregnancy and labour are continuous electronic monitoring of the fetal heart rate, and fetal blood sampling.

Only a small number of infants are severely asphyxiated at birth without any prior warning of fetal distress.

At birth, the clinical state of the baby depends upon the duration and severity of the anoxia before, during and immediately after delivery.

In acute asphyxia there is a slight initial increase in heart rate followed by a sudden brady-cardia during primary apnoea.

In severe apnea, the baby appears dead; it is either livid or pallid. All superficial and deep reflexes are absent and there are signs of circulatory failure.

The scoring system devised by the late virginia Apgar has been widely used to quantitate the severity of birth asphyxia. Routine scoring at 1 minute and 5 minutes after delivery has become the usual practice.

Asphyxia is generally regarded as the major cause of perinatal death and of disability in later life. Less severe degrees of anoxia may lead to damage of the nervous system with nerve cell necrosis and result in later spasticity or mental defect.

Multiple organ systems can sustain damage secondary to the hypoxia and circulatory insufficiency of perinatal asphyxia. Most noteworthy is hypoxic-ischemic encephalopathy.

The treatment of anoxia will vary with the cause. In all forms, however, warmth, oxygen and gentle handling are necessary.

The first treatment is to clear the upper airway. The next step must depend upon the condition of the baby as evaluated by the Apgar scoring.

The main object is to keep the severely asphyxiated baby artificially ventilated and with clear airways, until he can ventilate himself naturally. If spontaneous respirations are delayed, administer intermittent positive pressure breathing with a bag and mask. Continue bagging until heart rate >100 , color is pink, and spontaneous respirations have begun. If heart rate <60 , intubate and begin cardiac massage.

If heart rate is not >100 despite 2 minutes of adequate ventilation with 100 per cent O_2 and cardiac massage, insert umbilical venous catheter and administer drugs.

Cardiac massage, umbilical venous catheters, and drugs are all useless unless a secure airway and adequate ventilation with 100 per cent oxygen are first achieved.

The prognosis following recovery from a period of acute asphyxia seems to be good, but the prognosis following chronic asphyxia is much more unpredictable.

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