Resuscitation of the newborn human infant is necessary when the vital processes which initiate and maintain adequate tissue perfusion with oxygenated blood fail. These vital processes may be impaired because of anatomical and functional immaturity or by a developmental anomaly of the organs involved. More often, however, respiratory and circulatory insufficiency is found in the normally formed mature infant following antepartum or intrapartum hypoxia, injury or narcosis.

Until recently, methods employed for the detection of foetal distress during the delivery process were inadequate. Intrapartum asphyxia was the commonest single cause of perinatal death and only 1 in 8 of such infants was born alive (Butler, 1963). With improved techniques now available for the detection of impending antepartum and intrapartum anoxia, it appears likely that an increasing number of such infants will be liveborn and in need of careful assessment, if not active resuscitation at birth.

In 45 per cent of teaching and Scottish hospitals a paediatrician is responsible for resuscitation of the newborn, but in more than 80 per cent of all hospitals in the United Kingdom where a paediatrician is not available the anaesthetist, usually a Registrar or S.H.O., performs neonatal resuscitation (Taylor, 1971). As junior anaesthetists are likely to remain responsible for such emergencies it is essential that their training should impart to them some understanding of the processes which initiate and maintain respiration in the newborn and be well prepared to give aid when something goes wrong. The main purpose of this article is to describe the relevant physiological events which occur when the foetus becomes the newborn, the major things which can go wrong, and when and how best to help the infant when things have gone or are going wrong after birth.

Although there are two basic vital processes to be considered, the circulation and respiration, some consideration must also be given to the central nervous and neuromuscular activity of the newborn infant, and to the metabolic processes which provide energy and maintain a reasonable acid base and electrolyte status.

THE PATHOPHYSIOLOGY OF FOETAL AND NEONATAL ASPHYXIA

Foetal circulation.

Maternal supplies of oxygen and other nutrient materials to the placenta are largely controlled by the maternal placental blood flow through the spiral arterioles to the intervillous space. The foetus regulates the supply of oxygen to its tissues by changes in blood pressure, heart rate and blood flow to organs, including the placenta. In other words the foetus is dependent solely on its circulation for blood-gas homeostasis (Dawes, 1971).

The foetal circulation and the changes which occur at birth have been well described for a number of animal species, but are largely inferred for man (Dawes, 1968). Nearly 60 per cent of the foetal sheep's cardiac output passes through the umbilical arteries to supply the placental tissues, whilst only 10 per cent flows through the lungs. Oxygenated blood from the placenta is distributed in part to the liver, but a large proportion passes through the ductus venosus to the inferior vena cava and directly from there through the foramen ovale to the left atrium (fig. 1). One-third of the combined ventricular output passes through the ductus arteriosus. Gross cardiac abnormalities, which result, for example, in admixtures of blood at atrial and ventricular level, complete absence of a ventricle or of the aortic or pulmonary valves have little if any effect on foetal survival. Even the complete absence of one umbilical artery, without

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RESUSCITATION OF THE NEWBORN

**Foetal circulation.**

In man at birth a substantial volume of blood, up to 200 ml, may be present within the placental and umbilical vessels. A series of studies has shown that there are several important changes in the basic physiological status of the infant, dependent on the time at which the cord is clamped after birth and on the relationship to the onset of respiration. If the cord is clamped before the onset of respiration, in the absence of asphyxia, the infant has a lower blood volume (Gunther, 1957), lower respiratory rate (Oh, Lind and Gessner, 1966), higher lung compliance associated with a greater functional residual capacity (Oh et al., 1967), lower pulmonary arterial pressures and higher Pa\textsubscript{o}_2 and lower Pa\textsubscript{o}_2 than with late clamped infants (Oh et al., 1966). When the cord is clamped later there is an increased blood volume, arterial pressure and haematocrit with consequent increase in blood viscosity (Usher, Shephard and Lind, 1963; Burnard, 1966).

It is difficult to prevent a normally delivered infant from receiving a placental blood transfusion, unless there is unnecessary haste in cord clamping. The infant delivered by Caesarean section may not receive a placental transfusion if held above the mother's abdominal wall whilst the cord is clamped. Incontrovertible evidence of benefit or harm from early or delayed cord clamping is not yet available. Unless there is an emergency situation caused by placental separation, maternal haemorrhage or severe intrapartum asphyxia of the foetus, there seems no great merit in rushing to separate the infant from his placenta.

During intra-uterine life pulmonary arterial pressure exceeds aortic pressure, but after birth, as the ductus arteriosus constricts, pulmonary arterial pressure comes to be determined more closely by the pulmonary vascular resistance, so that the pressure falls gradually. The pulmonary blood vessels constrict vigorously in response to hypoxia, hypercapnia or metabolic acidosis (Campbell et al., 1966a, 1967).

In the immediate newborn period the transitional state of the circulation between that of the foetus and the older infant has been responsible for some of the difficulties experienced in the interpretation of the clinical findings in newly
born infants, particular when the infants are hypoxic. From figure 1 it can be seen that the newborn might shunt blood from left to right through the foramen ovale or from right to left through a patent ductus arteriosus when hypoxia or hypercarbia produces increased pulmonary vascular resistance. Cyanosis, in association with cardiac murmurs in the newborn infant, may thus be due to hypoxia and a transitional circulation rather than cyanotic congenital heart disease.

The foramen ovale functionally closes within a few days of birth, but there may be normally a small right-to-left shunt at this level during the first week of life (Lind and Wegelius, 1954; Prec and Cassels, 1955; Condorelli and Ungari, 1960), firstly because of the decreased inferior vena cava flow when the umbilical vein is occluded and secondly because of the large increase in pulmonary venous return with lung expansion. These haemodynamic changes hold the foramen closed, or almost closed, but anatomical closure may take many months or years (Scammon and Morris, 1918).

The closure of the ductus arteriosus, which in foetal life has almost the calibre of the aorta, is a remarkable phenomenon and its initial closure is the result of an active vasoconstriction as was suggested by Virchow (1856). Raising the oxygen tension of the blood or fluid perfusing the ductus channel causes the smooth muscle of the ductus arteriosus to contract (Assali et al., 1963; Kovalcik, 1963). In foetal lambs, however, a similar response occurs when the PaO₂ falls to 13 mm Hg, but probably this effect is due to the release of noradrenaline from the adrenals (Born et al., 1956).

Lung expansion with gas or air produces a large increase in pulmonary blood flow from 10–12 per cent up to nearly all of the cardiac output. This increased flow is associated with a decline in pulmonary vascular resistance, and when the pulmonary arterial pressure falls below that in the aorta increasing amounts of blood may pass back through the ductus arteriosus into the lungs. It is this flow that accounts for many of the soft inconsistent cardiac murmurs heard in the newborn infant during the first 10–12 hours after birth. Figure 2 demonstrates the changes in pressure, pulmonary blood flow and flow in the ductus arteriosus when the umbilical cord is tied and breathing begins (Dawes, 1958). In the human infant there is commonly a right-to-left shunt during the first hour of life. It is the balance between the left and right ventricular output against the altering systemic and pulmonary resistance which characterizes the circulatory patterns in the newborn period. Under normal circumstances the ductus constricts within 8–10 hours and is functionally closed by the end of the first week of life. Fibrous contraction with obliteration of the ductus to form the ligamentum arteriosum may take 2–3 months.

In prematurely delivered infants there may be delay in the functional closure of the ductus arteriosus and this may be aggravated in those infants in whom the respiratory distress syndrome develops (Burnard, 1959; Rudolf et al., 1961; Stahlman, 1966).
Resuscitation of the Newborn

Respiration.

Independent extra-uterine existence is not possible until there is an adequate alveolar surface area available for gas exchange. In man this stage of lung development is not reached until the 26/28th week of gestation when the foetus weighs about 1 kg. At about this time alveolar monocytes begin to be capable of secreting sufficient lipoprotein material into the alveolus (Reynolds and Strang, 1966). This material reduces surface tension within the alveolus so that when the alveolus is in the unexpanded state the increased surface tension associated with its reduced surface area does not prevent its expansion. Arteries, capillaries, veins and lymphatic vessels are as important to normal lung function as alveoli, and must adapt abruptly at birth to the sudden expansion of the lungs and increased pulmonary blood flow which are necessary to permit effective blood-gas exchange and removal of lung fluid. Anatomical studies would suggest that these vascular channels may be functionally immature until about 34 weeks' gestation. In particular, terminal and respiratory arteries remain narrow until 34 weeks' gestation (Kaufman, 1964).

Breathing movements in utero. Davis and Potter (1946) introduced radiopaque material into human amniotic cavities and showed that the material was present in the infant's lungs X-rayed after delivery. McLain (1964) repeated the experiment in 75 pregnant women and was unable to detect radiopaque material within the lungs of the foetuses X-rayed in utero. It appeared likely that the material was aspirated into the lungs during the delivery process. Long-term catheterization studies in foetal lambs, however, have shown that there is a rapid, irregular, rhythmic type of respiratory movement during the last third of pregnancy in utero unrelated to changes in arterial Po2, and with the production of appreciable quantities of lung fluid, 8 ml/hour (Dawes et al., 1971; Tchobrutsky, 1971, personal communication). The appearance of meconium and squamous cells in the lung periphery in infants dying in utero from asphyxia, indicates that substantial respiratory efforts can be made in response to asphyxia in utero.

There is, in utero, a substantial volume of fluid within the lung, most of it within the intra-alveolar spaces and tracheobronchial tree. Fluid is produced within the lungs and its chemical composition is entirely different from that of amniotic fluid and it is probably an ultrafiltrate of foetal plasma, modified by the secretions of the alveolar cells and tracheobronchial glands (Adams, Moss and Fagan, 1963; Adams, Fujiwara and Rowshan, 1963; Ross, 1963). Before air breathing can be established the fluid within the tracheobronchial tree must be removed. During a normal vaginal delivery the thorax is compressed during the delivery process. The rise in intrathoracic pressure (70 cm of water) expels a volume of fluid equivalent to an estimated one-third of the infant's functional residual capacity. This volume is replaced with air by the elastic recoil of the thoracic wall if the upper airways are patent (Karlberg, 1960). Infants delivered by Caesarean section do not "benefit" from this mechanism, and there must be an efficient removal of the remaining lung fluid. It appears likely that with the onset of respiratory movements and increased pulmonary blood flow, the excess fluid is removed by the pulmonary lymphatics (Boston et al., 1965).

The first breath. Normal, mature vaginally delivered infants make their first respiratory movement within a few seconds of complete delivery. The time interval between the appearance of the nose and the first breath is usually between 20-30 seconds (Westin et al., 1962). Within 90 seconds of complete delivery the majority have commenced rhythmic respirations (Wulf, 1959).

The strong initial inspiratory efforts and the subsequent rhythmic activities of the respiratory muscles are largely, if not entirely, dependent upon brain stem respiratory centres. In spite of a very large effort by many physiologists there is as yet no agreement as to the factors which initiate and maintain respiration. Table I summarizes the factors which can, under a variety of circumstances, initiate or influence the patterns of neonatal respirations, but none of these individual factors is absolutely essential. For example, some infants commence breathing normally when their blood-gas status is normal, whilst others do not make any respiratory movement until they have become moderately asphyxiated. There are a number of factors which influence the ability of the respiratory centres to respond to each or any of these stimuli:
Stimuli which may contribute to the initiation and maintenance of respiration in the newborn assuming intact nerve pathways between receptors and functional respiratory centres and from these centres through the spinal cord and lower motor neurones to the respiratory muscles.

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thermal Pain</td>
<td>Loss of heat at about 600 cal per minute; cold stimulus</td>
</tr>
<tr>
<td>Pressure</td>
<td>Skin, muscle and tendon receptors</td>
</tr>
<tr>
<td>Tactile</td>
<td>Gravity—decreased pressure from that in utero</td>
</tr>
<tr>
<td></td>
<td>Increased intrathoracic pressures during delivery</td>
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<tr>
<td></td>
<td>Increased intracranial pressures during delivery</td>
</tr>
<tr>
<td></td>
<td>Changes in intrathoracic and intracranial pressure</td>
</tr>
<tr>
<td></td>
<td>Trigeminal area particularly important</td>
</tr>
<tr>
<td>Auditory Visual</td>
<td>Hering-Breuer reflexes</td>
</tr>
<tr>
<td></td>
<td>Head's reflex</td>
</tr>
<tr>
<td>Cord clamping</td>
<td>Stimulated by alterations in posture during and after delivery</td>
</tr>
<tr>
<td>Decreased PaO₂</td>
<td>Carotid baroreceptors</td>
</tr>
<tr>
<td>Increased PaO₂</td>
<td>Stretch receptors aorta and carotid</td>
</tr>
<tr>
<td>Increased [H⁺]</td>
<td>Aortic and carotid body chemoreceptors</td>
</tr>
</tbody>
</table>

for example, maternal anaesthetic and analgesic drugs, low environmental temperature (Westin et al., 1962), high environmental temperature (Dawes, 1968), gestational age (Bodegard and Schwieler, 1971), ante- and intrapartum asphyxia (Towbin, 1969a), intracranial or brain stem haemorrhage, vascular thrombosis and cerebral oedema with increased intracranial pressure (Towbin, 1969b).

Once breathing is established the pattern of respiration is primarily determined in the medulla. Pulmonary ventilation is regulated by the chemical state of the arterial blood, particularly by the Pco₂ and [H⁺] perfusing chemoreceptors in the medulla. Breathing may also be stimulated by a decrease in Po₂ acting on the chemoreceptors in the carotid bodies. This hypoxic drive may assume great importance if the medullary centres are insensitive.

### Pulmonary ventilation during regular respirations.

Normal, mature newborn infants achieve a minute volume of over 500 ml by breathing a tidal volume of 10–30 ml about 30–60 times per minute when lung compliance is 5 ml per centimetre of water.

The complexity of the respiratory processes in man has made the evaluation of various forms of resuscitation extremely difficult, and at the same time has made it difficult to evaluate the relevance of animal studies to the clinical situation. Nevertheless animal investigations of experimental asphyxiation of the newborn have clarified some aspects of the acute asphyxial process.

### Acute asphyxia.

Most newborn mammals respond to acute total asphyxia in a characteristic way (Davis and Moore, 1966; Dawes, 1968). Figure 3 shows the pattern of events following acute asphyxia in the newborn rhesus monkey immediately after birth, where after a period of hyperpnoea and struggling there is a period of initial or primary apnoea lasting from 1 to 1½ minutes. Regular respirations follow the initial apnoea and after a terminal acceleration in rate, but diminution in depth, cease about 8 minutes after birth. The apnoeic period which follows the last spontaneous gasp is called secondary or terminal apnoea.

Damage to the central nervous system of animals can occur even before the end of the gasping stage (Windle, 1966), but is invariably present after 12½ minutes of acute asphyxia in the rhesus monkey. The pattern of events in acute asphyxia and the degree of brain stem damage can be influenced by a great many factors, and the value of various resuscitative measures can be assessed by their influence on the asphyxiated animal. For example, alkali and...
RESUSCITATION OF THE NEWBORN

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ARTERIAL

\[ P_{O_2} \ 25 \ 5 < 2 \]

\[ P_{CO_2} \ 45 \ 100 \ 150 \ 200 \ 40 \]

\[ pH \ 7.3 \ 7.0 \ 6.8 \ 6.75 \ 7.1 \]

Gases/min

6

4

2

0

PRIMARY APNOEA

LAST GASP

ONSET OF GASping

SECONDARY OR TERMINAL APNOEA

Beats/min

200

150

100

50

0

HEART RATE

mm Hg

60

40

20

0

BLOOD PRESSURE

TIME FROM ONSET OF ASPHYXIA (min)

FIG. 3

Schematic diagram of changes in rhesus monkeys during asphyxia and on resuscitation by positive pressure ventilation. Brain damage was assessed by histological examination some weeks or months later (Dawes, 1968).

glucose given intravenously during asphyxia improve survival and decrease brain damage (Adamsons et al., 1963, 1964); hyperbaric oxygenation is less effective than intermittent positive pressure ventilation (Campbell et al., 1966b); hypothermia which was thought to have some beneficial effect (Miller and Miller, 1965) was subsequently shown to be ineffective (Daniel et al., 1966a); barbiturates protect the central nervous system from asphyxial damage and although not recommended as a therapy for asphyxia there is less worry about the possible harmful effect of barbiturates given during early labour (Cockburn et al., 1969); analeptics like nikethamide can cause severe hypotension and be harmful in asphyxia (Daniel et al., 1966b).

Acute asphyxial experiments in animals, however, may not be relevant to prolonged or recurrent hypoxaemia in man. Labour is a much longer process in man and disorders of placental function may cause intra-uterine hypoxia which is an important aetiological factor in the cerebral damage which may follow complicated delivery (Gruenwald, 1963). Placental failure and intra-uterine asphyxia will also result in many metabolic changes within the foetus and these changes will reduce the newborn infant's ability to withstand any undue stress during the delivery process or the immediate newborn period. In particular, reserves of glycogen within cardiac muscle and liver are low (Shelley, 1964) and any additional hypoxaemia can readily exhaust these limited energy supplies. Oxygen lack, acidosis and hypercapnia produce not only the circulatory changes described earlier, but also widespread electrolyte changes. Anaerobic metabolism is wasteful of the limited energy reserves, results in a metabolic acidosis and causes disruption of cell membrane integrity. However, there seems little doubt that the newborn human infant is more resistant to severe hypoxia than the adult (Avery, 1963) and can tolerate variations in plasma hydrogen ion concentration and Pco₂ which would be fatal to the adult. The reasons for such resistance are not clear, but in part it is due to the ability of the infant to maintain the circulation in the face of severe hypoxaemia, acidosis and hyperkalaemia.

Tissue survival during asphyxia depends on a number of factors. Less mature tissue, tissue which is relatively inactive, tissue containing substrate for anaerobic degradation within the cells or tissue with a good circulation which effectively removes hydrogen ion and potassium will survive hypoxia better than tissues without these advantages. These are probably the reasons why infant tissues, e.g. cardiac muscle and brain, show increased resistance to hypoxaemia.

Biochemical changes in brain from hypoxia and ischaemia.

The brain has very limited energy reserves and normally relies, for practically the whole of its energy supply, on glucose and oxygen transported by the blood. Arrest of cerebral circulation results in a rapid fall in brain glucose with a rise in tissue lactate as glycolysis commences.
Concentrations of ATP are maintained for a while at the expense of phosphocreatine, but as the concentrations of high energy phosphate fall, cellular function ceases. Active transport will fail, and sodium will enter cells as the "sodium pump" mechanism decelerates and potassium and amino acids will leak out. Inorganic phosphate and ammonia will accumulate, there will be increased formation of γ-aminobutyric acid (GABA) through decarboxylation of glutamate, and the necessary synthetic processes of the cell, e.g. resynthesis of acetylcholine, will cease.

Impairment of cerebral function, as indicated by e.g., in hypoxic rabbits occurs before there is much depletion of high energy phosphate (Olsen and Klein, 1947). These, and other observations, suggest that the synthesis of acetylcholine is more sensitive than the "sodium pump" to the effect of anoxia (Richter, 1967).

Circulation.

Figure 3 shows that in acute asphyxia there is an initial acceleration of heart rate followed by an abrupt fall associated with primary apnoea. The heart rate remains low until resuscitation is given, when there is an equally abrupt increase in heart rate which precedes by several minutes the onset of spontaneous gasping. A persisting low heart rate, e.g. less than 100 beats/min, may rarely be due to congenital heart block or vagal overactivity, but is almost always due to hypoxia and is the most reliable indication of the need for active resuscitation. Absence of the heart sounds on auscultation would mean that an infant was almost certainly in secondary apnoea and probably implies that the infant has been asphyxiated to a stage beyond the last spontaneous gasp. Here, however, we run into the difficulty of trying to relate a human situation to acute asphyxia in animals.

Blood pressure during asphyxia shows an initial rise which may in part account for the marked umbilical arterial pulsation seen in the moderately asphyxiated infant. Severe and prolonged asphyxia causes a fall in blood pressure. In practice it is seldom possible to measure arterial blood pressure, but an e.c.g. tracing showing prolongation of the Q-T interval with an inverted T wave and variable degrees of heart block might indicate a falling pressure.

Assessments of colour are not often helpful, but the very pale infant is likely to have peripheral circulatory failure.

Respiration.

After heart rate, observation of the respiratory movements is the most helpful of the vital signs. If respiratory movements are vigorous from birth the normal infant quickly settles to a steady respiratory rate of about 40 b.p.m. within 5 minutes. Less mature infants or infants with respiratory depression have a marked variation in respiratory pattern akin to Cheyne-Stokes breathing and rates will fluctuate wildly sometimes for days after birth. Inspiratory movements are largely abdominal in the newborn, but when retractions of the xyphoid and lower intercostal muscles develop this often indicates the onset of the respiratory distress syndrome of the newborn.

Rapid respirations (over 60 b.p.m.) which
cause respiratory alkalosis, in the absence of a coexistent metabolic acidosis and infection, are almost certainly due to intracranial haemorrhage.

Neuromuscular responses.
Assessments of muscle tone and reflex irritability in the mature infant give some indication of central nervous system depression, but must be interpreted with care, for example, in the prematurely born or mongol infant, in the infant born to a mother with myasthenia gravis or following maternal antepartum treatment with magnesium, and also in the rare instances of amyotonia congenita and poliomyelitis. Major causes of poor muscle tone, failure to respond to stimuli and ineffective respiratory movements are hypoxia, cerebral haemorrhage or oedema, narcosis or damage to the cervical spinal cord.

The Apgar score.
A clinical score based on heart rate, respiratory effort, muscle tone, responses to stimuli and skin colour was derived by Apgar (1953) and has been found valuable in the assessment of the newly born (Apgar and James, 1962; Gupta and Tizard, 1967). The Apgar score (table II) is usually made at 1 minute after birth and repeated again at 5 minutes. A time interval of 60 seconds after birth was chosen by Apgar because at this time most infants in her large series had achieved their lowest score.

Whereas the 1-minute score gives some indication of the need for active resuscitation, correlates well with biochemical assessment of acidosis and is inversely proportional to the neonatal death rate (James et al., 1958), the 5-minute score has been shown to have some correlation with subsequent brain damage (Drage and Berendes, 1966).

Procedure for assessment.
Assessments of the infant are best made by someone other than the person conducting the delivery. While the delivery is taking place the person responsible for the assessment and resuscitation should obtain information about the mother's general health, her health during pregnancy, the duration of the pregnancy and other relevant information such as the dosage and timing of analgesics given to the mother.

As the head is born during a vertex presentation the delivery attendant or assistant will wipe or aspirate excess mucus and debris from the nares and oropharynx. A clock with a large second hand should be started after complete delivery of the infant.

After birth the clinical examination and assessment of the infant's adaptation to its new environment is best performed with the infant in a good light on a warm flat surface, tilted head down at an angle of 15–20 degrees to the horizontal at a convenient working height. A quick overall examination should be made to exclude gross congenital malformation. With the infant lying supine and loosely wrapped in a warm, dry, sterile covering or positioned under a properly designed radiant heat cradle, the time of the first gasp, first cry and onset of sustained respiration should be noted and any asymmetry of chest or abnormality of abdominal movements observed.

If there was evidence during delivery of severe foetal distress or if the infant is hypotonic and

<table>
<thead>
<tr>
<th>Sign</th>
<th>0</th>
<th>1</th>
<th>2</th>
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<tbody>
<tr>
<td>Heart rate</td>
<td>Absent</td>
<td>Slow (below 100)</td>
<td>Over 100</td>
</tr>
<tr>
<td>Respiratory effort</td>
<td>Absent</td>
<td>Weak cry; hypoventilation</td>
<td>Good; strong cry</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Limp</td>
<td>Some flexion of extremities</td>
<td>Active motion; extremities well flexed</td>
</tr>
<tr>
<td>Reflex irritability (response to stimulation of sole of foot)</td>
<td>No response</td>
<td>Grimace</td>
<td>Cry</td>
</tr>
<tr>
<td>Colour</td>
<td>Blue; pale</td>
<td>Body pink; extremities blue</td>
<td>Completely pink</td>
</tr>
</tbody>
</table>

Sixty seconds after complete birth of the infant (disregarding the cord and placenta) the five objective signs are evaluated and each given a score of 0, 1 or 2. A total score of 10 indicates an infant in the best possible condition.
makes no respiratory movements during the brief initial examination, the heart should be listened to immediately. Needless to say one would not wait until 1 minute after birth and an Apgar score had been completed before giving aid to an infant who was not breathing and had no audible apex beat. Absence of heart sounds in a viable infant is an absolute indication for cardiac massage and ventilation.

If, after completing the 1-minute Apgar score, the infant is breathing well and there is no excess of mucus in the oropharynx the infant should be turned into the head-up position and the 5-minute Apgar score completed.

THE MANAGEMENT OF NEONATAL ASPHYXIA

Ambient temperature.

During assessment and resuscitation, particular attention must be given to the prevention of heat loss by the infant. Rapid heat loss from the wet infant stimulates heat production (Hardman, Hey and Hull, 1969) and the heat-producing adipose tissue will compete with other tissues for cardiac output and limited oxygen supply, to the detriment of the infant (Heim and Hull, 1966). As much of the infant as possible, particularly the head, should be covered with a warm, dry covering or the environmental temperature surrounding the infant should be maintained by an external source of heat. Care must be taken in the use of undiffused radiant heat sources which can badly burn the skin of any infant in peripheral circulatory failure. Servo-controlled radiant warmth might prove the best way to maintain infant body temperature during resuscitation.

Mucus extraction.

During the initial minute of observation, gentle nasal and pharyngeal suction with a soft catheter which has a single end hole and a mucus trap is recommended. Although blockage to upper airways with mucus may lead to respiratory difficulties, the value of mucus extraction lies as much in its stimulus to respiration and in the opportunity it affords to check for anatomical abnormalities which might cause respiratory difficulties.

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The Normal Infant.

Apgar score 7–10.

The majority of infants fall into this category and require no resuscitation at 1 minute after birth. However, they must be assessed again at 5 minutes. It is not unusual to record a normal 1-minute score, particularly in the infant born to a mother given opiates during labour, and to find the infant apnoeic and blue minutes later from failure to maintain regular respirations.

Moderately Depressed Infants.

Apgar score 3–6.

At 1 minute and in the absence of upper airway obstruction, these infants should have oxygen or oxygen-enriched (40–50 per cent) air blown on to the face through a mask. If this cold stimulus and oxygen does not improve the infant’s condition it is unlikely that other stimuli will initiate respiration, but there is no contraindication to giving vitamin K (1 mg) by intramuscular injection at this stage. An Ambu bag attached to a face mask through which pressures of 30 cm of water are applied from 1 to 2 seconds whilst the mask is held tightly over the nose and mouth, will result in gas entering the trachea and respiratory bronchioles and will on occasion prove helpful; however, high inflationary pressures will fill the stomach with gas and should not be used. If there is no improvement in the ensuing 60 seconds with these measures it is necessary to examine the larynx directly with a neonatal laryngoscope and to aspirate any debris. Very often this is a sufficient stimulus to respiration, but if not, an endotracheal tube should be inserted and intermittent positive pressure ventilation performed.

Severely Depressed Infants.

Apgar score 0–2.

These infants require immediate help. An endotracheal tube should be inserted, after pharyngeal suction, under direct vision and the lungs inflated. Thankfully the usual response to this treatment is a sudden increase in the intensity and rate of the heart sounds, the infant rapidly becomes pink, respiratory efforts start and muscle tone returns. Slowing of the heart rate, despite artificial ventilation, is an ominous
sign and an indication for cardiac massage. This is carried out by compressing the heart between the chest wall and vertebral column. The index and middle fingers are placed to the left of the lower sternal border, the chest wall is depressed approximately 2 cm then released 4-5 times every 3-4 seconds. A very effective arterial pulsation will result. Inflate the lungs once every 4 seconds then repeat the cardiac massage. After 30 seconds stop briefly and listen for signs of improvement in quality and rate of heart sounds. Repeat the procedure if necessary, but stop as soon as a heart rate above 100 beats/min is achieved. During this procedure sodium bicarbonate 8.4 per cent (5 ml) and dextrose 10 per cent (5 ml) can be injected into the umbilical vein, and this may be repeated 5-10 minutes later. Ventilation may be discontinued and the tube removed once rhythmic respiration is established.

**Technique of intubation and intermittent positive pressure ventilation.**

With the infant supine and the neck slightly extended pass the laryngoscope blade over the dorsum of the tongue until the epiglottis is seen. Clear the area of mucus with gentle suction and insert the tip of the blade just over the epiglottis and pull the laryngoscope gently forward. It is not always easy to catch the epiglottis with the blade tip as shown in figure 4, but an adequate view is possible with the tip of the blade in the vallecula anterior to the epiglottis. The blade tip is unlikely to cause any harm by pressing in the vallecular region. Gentle pressure over the anterior aspect of the neck with the little finger of the hand holding the laryngoscope, or with an assistant’s finger, will bring the glottis into view (fig. 4). The glottis of the newborn infant appears as a mound with a small central hole or anteroposterior slit. Vocal cords are rarely seen. Although frothy fluid may be actively draining from the glottic orifice, there is generally no need to aspirate. Once the infant is intubated and positive pressure applied, the fluid is readily resorbed into the pulmonary lymphatics. Hold the laryngoscope in the left hand and pass the tip of the endotracheal tube about 2 cm through the glottis. Keep the tube in this position with the right hand and withdraw the laryngoscope. A water manometer (Barrie, 1963), mechanical ventilator, Ambu bag or the resuscitator’s cheeks can then be attached to the tube and a few puffs given. The usual safe inflation pressure recommended for the newborn’s lungs is 30 cm of water, but there are some infants who require higher pressures. This can only be judged by assessing chest movement. Most infants respond adequately to 30 cm of water pressure. If mouth-to-tube breathing is necessary, use only the cheeks to push air or oxygen-enriched air into the tube because it is not difficult to achieve pressures of 100 cm of water by blowing. A ventilatory rate of between 40-50 per minute, with inflation lasting approximately 1 second at 30 cm of water pressure, gives effective ventilation (Hull, 1971). Tidal volume varies between 10 and 30 ml.

Most newborn nurseries have a supply of 100 per cent oxygen. Very few have air or oxygen-enriched air (30-60 per cent oxygen). Exposure to 100 per cent dry oxygen for the few minutes involved in resuscitation does not appear to carry any risk of oxygen toxicity. A drawback to the
use of 100 per cent oxygen is that when the pure oxygen is resorbed from the alveoli, complete alveolar collapse occurs. Where ventilation has to be prolonged the gas requires to be humidified and the oxygen concentration maintained at that level which keeps the arterial $P_{O_2}$ above the ductus between 60 and 100 mm Hg.

If both sides of the chest move satisfactorily and continued ventilation is necessary, a large safety-pin may be pushed through the edge of the endotracheal tube at the lip margin. The pin is then closed and strapped at each end to the cheeks. It is then safe to let go of the endotracheal tube and it is unlikely to disappear down the oesophagus (Dickson and Fraser, 1967). Should chest movement be asymmetrical, withdraw the endotracheal tube a little and try again. Even with a tube designed to prevent its insertion too far, it is very easy to pass this tube into the right main bronchus and ventilate only the right lower lobe. Figure 5 shows complete collapse of the right middle and upper lobes, together with the whole of the left lung, following ventilation with pure oxygen when the endotracheal tube was in the right lower lobe bronchus. The second X-ray was taken less than 10 minutes after the first with the endotracheal tube removed.

**Fig. 5**

(A) Insertion of an endotracheal tube through the right main bronchus can result in overdistension of the right lower lobe with collapse of the right upper lobe and left lung.

(B) Withdrawal of the endotracheal tube allows complete re-expansion. The repeat chest X-ray with oesophagogastric tube in position was made less than ten minutes after (A).

**EQUIPMENT**

Resuscitation apparatus.

The equipment illustrated in figure 6 has been found adequate for most resuscitative measures in the immediate newborn period. There is a separate intravenous cut-down umbilical catheter pack and thoracocentesis tray.

A 15 f.g. Aberdeen mucus extractor is still the most convenient and simple instrument for removing pharyngeal debris and is safe when used with direct vision, but must not be thrust blindly into the pharynx or larynx.

The 5 f.g. soft polyvinyl catheter can be used to check the patency of the nasal airways and to test for oesophageal atresia if thought necessary. The same catheter may be used to aspirate meconium or other material from inside the endotracheal tube.

A 12 f.g. disposable polyvinyl endotracheal tube (Warne) is suitable for resuscitation, but where prolonged ventilation is necessary, a Jackson Rees nasotracheal tube (2.5–3.0 mm) is convenient and is positioned using Magill forceps. There are many laryngoscope designs available. The Magill infants blade has proved satisfactory. A spare bulb and battery, if not a spare laryngoscope, are absolutely essential.
A 10 f.g. disposable trocar and catheter may be necessary at times. It is inserted through an intercostal skin incision at a position best decided on the erect chest X-ray film and connected to an underwater seal drain in the management of a tension pneumothorax.

Ancillary therapies.

Tracheal intubation with IPPR is the most effective method of resuscitation. Of the other methods available, face mask or mouth-to-mouth respiration and hyperbaric oxygen are of value when facilities for intubation are absent. Placing the infant in a hyperbaric chamber and increasing the pressure to 3 atmospheres takes longer than endotracheal intubation. It does not effectively remove carbon dioxide from the infant and makes cardiac massage, mucus aspiration or catheterization of the umbilical vessels difficult. Analeptic agents have little or no value and may cause harm when given to asphyxiated infants. Nalorphine 0.5 mg is of value when given intra-

![Resuscitation tray](https://academic.oup.com/bja/article-abstract/43/9/886/276599)

<table>
<thead>
<tr>
<th>Item</th>
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</thead>
<tbody>
<tr>
<td>Face masks</td>
</tr>
<tr>
<td>Ventilation bag</td>
</tr>
<tr>
<td>Y-adapter to connect water manometer to endotracheal tube</td>
</tr>
<tr>
<td>Infant oral airway</td>
</tr>
<tr>
<td>Mayo's forceps</td>
</tr>
<tr>
<td>Nasotracheal tube (2.5/3.0 mm)</td>
</tr>
<tr>
<td>Endotracheal tube (12 f.g.)</td>
</tr>
<tr>
<td>Infant laryngoscope with spare batteries and bulb</td>
</tr>
<tr>
<td>Suction catheters (5 f.g.)</td>
</tr>
<tr>
<td>Mucus aspirator</td>
</tr>
<tr>
<td>Syringes with needles</td>
</tr>
<tr>
<td>Nalorphine (1 mg/ml)</td>
</tr>
<tr>
<td>5 ml NaHCO₃ (5%/8.4%)</td>
</tr>
<tr>
<td>5 ml dextrose (10%)</td>
</tr>
<tr>
<td>Adhesive tape</td>
</tr>
<tr>
<td>Safety-pin</td>
</tr>
<tr>
<td>Scissors</td>
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The syringe, needles, suction catheters, mucus extractor, forceps and face masks are normally in prepack sterile packets and have been removed for demonstration.
venously to counteract the effects of maternal sedation with morphine, diamorphine or pethidine. Sodium bicarbonate 8.4 per cent (5 ml) given with 5 ml 10 per cent dextrose and repeated in 5–10 minutes when necessary improves survival in severely acidotic infants and has caused no obvious harm or gross alteration in plasma osmolalities or sodium concentrations when used in our nurseries over the past 18 months. There are many infants who appear to show dramatic clinical improvement related to the infusion. Further aliquots of bicarbonate are given only on the basis of biochemical assessment of metabolic acidosis. Berg, Mulling and Saling (1969) recommend the infusion of 4 ml/kg body weight of a solution containing 2 ml 40 per cent tris-hydroxy-methyl-aminomethane (THAM) in 10 ml 10 per cent glucose via the umbilical vein. Because of the risks of cardiac arrhythmia and respiratory arrest, it has been our practice to reserve THAM for infants on ventilator treatment of respiratory failure and then to use it only when the plasma sodium exceeds 150 m.eqiv/l.

Dexamethasone 1 mg by intravenous injection may be of value in cases of severe hypoxia. The rationale for its use is:

1. to reduce intracranial pressure;
2. to decrease capillary fragility; and
3. to reduce pulmonary inflammatory reaction to aspirated meconium.

No published trial of dexamethasone therapy in asphyxiated human infants is available, but it has been widely used to reduce the cerebral oedema of head injuries in older children and adults, and merits a careful trial. Treatment would need to be continued for several days with 6-hourly dexamethasone 1 mg by intramuscular injection.

During the few days after birth the infant badly asphyxiated at birth may lose control of thermo-regulation and plasma osmolality. There are often gross swings in plasma electrolyte concentrations with hyponatraemia, hyperkalaemia, hypocalcaemia, hypo– and hyperglycaemia secondary to cellular damage. Correction of these electrolyte disturbances may reduce the infant's tendency to convulse and certainly makes the neurological findings easier to interpret.

Infants with bilateral choanal atresia or with the receding chin and glossoptosis of the Pierre-Robin syndrome have respiratory difficulties, best relieved by an oral airway or in the latter disorder by placing the infant in the prone, head-down position.

Congenital laryngeal or tracheal stenosis or atresia are rare, but must be considered in any infant gasping but without signs of air entry into the chest. Laryngeal webs found by laryngoscopy can be penetrated. Usually this group of conditions is best treated by tracheostomy. Unfortunately with these congenital malformations the lungs are abnormal (pulmonary hypoplasia) and the mortality rate is very high (Smith and Bain, 1965).

Bilateral pulmonary hypoplasia is usually associated with absence of amniotic fluid and is secondary to bilateral renal agenesis, lower urinary tract obstruction or prolonged leakage of liquor. Attempts to expand such lungs often produce a pneumothorax. The squashed appearance of the facies is similar in all three disorders (Bain, Smith and Gauld, 1964).

Diaphragmatic herniae are relatively common—1 in 2,000 deliveries (Butler and Claireaux, 1962) and apart from mechanical difficulties produced by the abdominal contents within the chest, the lungs are hypoplastic. Survival usually depends on the state of the lungs as well as the skill of surgeon and anaesthetist. Infants with a hernia on the left side are more readily diagnosed as the apex of the heart is on the right and the abdomen appears scaphoid. The differential diagnosis between diaphragmatic defect, pneumothorax and dextrocardia with cyanotic congenital heart disease is difficult and is best confirmed by prompt X-ray examination. Immediate management is by intermittent positive pressure ventilation through an endotracheal tube. To prevent distension of the gut within the chest, a large-bore open-ended tube is passed into the stomach. Early surgery gives the best chance of survival.

Isolated unilateral pulmonary agenesis does not usually give rise to respiratory difficulties in the newborn period.

Intratatal pneumonias may be suspected if there is a history of prolonged rupture of the
membranes or if the amniotic fluid smells offensive.

Spontaneous pneumothorax, pneumomediastinum, lobar emphysema, atelectasis and meconium aspiration are diagnoses to be considered and evaluated on the basis of history, clinical and X-ray examination.

Pulmonary oedema, pleural effusions and pulmonary haemorrhage are found not uncommonly in the infant with severe haemolytic anaemia.

A variety of rare bone and cartilage disorders involving the chest wall and vertebral bodies can cause neonatal asphyxia (Jeune et al., 1954; Barnes, Hull and Symons, 1969; Wilson, Crispin and Carter, 1969).

Signs of upper airway obstruction can be produced by a variety of cysts, tumours, goitres and vascular anomalies. Most of the respiratory difficulties can be overcome by endotracheal or nasotracheal intubation, but on occasion tracheostomy is required.

With intracranial haemorrhage or spinal cord injury infants may exhibit weak and irregular respiratory movements. Their responses are similar to an older patient with head injury, and their management should not be dissimilar to that of the older patient.

Severely anaemic infants, particularly after acute blood loss, may also appear extremely pale and have ineffectual and irregular respiratory movements. The blood loss may be obvious following umbilical cord haemorrhage or it may be masked and appear as maternal bleeding from an antepartum haemorrhage. Infants with rupture of abdominal viscera or with massive intra-abdominal haemorrhage and gastric perforation may all present the same clinical appearance. Haematocrit or haemoglobin estimations should be repeated hourly on several occasions, but if on clinical grounds there is severe hypotension, blood transfusion with universal donor blood should be given to raise the venous and arterial pressures to a normal value. If blood loss cannot be estimated, a volume of 20 ml partially packed red blood cells per kg body weight may be given immediately.

WHEN TO START AND WHEN TO STOP
These questions cannot have a simple answer. The object of resuscitation is to prevent cellular damage in an infant who may be potentially normal, and to prevent severe abnormality occurring in an infant who has already sustained a moderate degree of cellular damage. But what of the infant born with gross damage? Can we identify him? Should we identify him?

In the presence of intra-uterine hypoxaemia foetal heart monitoring throughout delivery will measure the exact duration of bradycardia or of cardiac electrical inactivity before resuscitation is started, but even the presence of a normal e.c.g. tracing does not signify effective cardiac output or adequate foetal cerebral blood flow. If there is any doubt about the need to resuscitate an infant there is sufficient evidence to advise a trial of active resuscitation, including cardiac massage (Gallagher and Neligan, 1962). This should certainly be tried where the foetal heart has been heard within 10–15 minutes of delivery. If there is no response, either in e.c.g. or heart sounds after 10–15 minutes there is little point in continuing. In otherwise normal infants with no vital signs present at birth “masterly inactivity” is inexcusable; at least until we have learned a great deal more.

A more difficult decision is when to stop ventilating the infant who fails to sustain regular respirations but who maintains a good colour and regular heart beat during ventilation and after correction of metabolic acidosis with bicarbonate. It is easy to say ventilate for 1 hour or 2 hours and then stop, but it is not so easy when you are immediately responsible. One can always give individual instances where infants resuscitated for longer than 2 hours have been apparently normal 5 or 6 years later.

Electroencephalograms do not help, because during hypoxaemic episodes the tracing can be flat, but during ventilation the tracing can return to normal. If such an infant is placed on a mechanical ventilator for several days the tracing can appear normal for a few days and then if brain damage has been severe, either a bizarre epileptic pattern or a flat tracing may develop. After a few days the electromyograph of a severely asphyxiated infant can show a denervation pattern.

The problem is very similar to that posed by the adult with respiratory failure after a severe head injury, or asphyxia, or after a cardiac arrest, and just as difficult to resolve.
When there is any doubt as to what to do in this situation it is better to continue ventilation until the opinion of an experienced neonatal paediatrician or paediatric neurologist can be obtained.

Whether or not any asphyxiated infant will sustain a permanent neurological deficit must depend on which of the several aetiological factors predominate and the speed and effectiveness of resuscitation. For any particular infant the Apgar score is of limited prognostic value, but if the 5-minute Apgar score is low, careful observation of developmental progress will be required for the first few years of life.

It is difficult to obtain good information on resuscitative procedures unless details of these are recorded by an observer or on monitoring and recording equipment. As soon as the resuscitative measures are complete, as full a record as possible should be included with the infant’s case sheet for future reference.

Tremendous improvements have been made in the management of newborn infants with respiratory difficulties during recent years. These improvements have arisen particularly as a result of a greater understanding of foetal and neonatal physiology. The responses of immature organs and tissues have been critically examined in the experimental animal and measures which prevent or alleviate the harmful effects of asphyxia in animals are now being applied to the human situation. There are so many factors to be considered in the pathogenesis of mental and physical handicap that the relative importance of neonatal asphyxia is difficult to isolate from the other possible causes. To this end, careful recording of the infant’s responses to any resuscitative measures must be made and correlated with the eventual well-being or otherwise of the child.

REFERENCES


— — — — (1954). Polvchondrodystrophie ayec blocage thora- 


NORTH OF ENGLAND SOCIETY OF ANAESTHETISTS

Programme 1971–72

1971
FRIDAY, OCTOBER 8. President’s Night.
FRIDAY, NOVEMBER 12. Dr Gordon Pledger, Department of Medical Administration, Oxford Regional Hospital Board: “Problems in Anaesthesia—a Medical Administrator’s View”.
FRIDAY, DECEMBER 10. Dr J. E. Riding, Department of Anaesthesia, United Liverpool Hospitals: “A Never Ending Tale”.

1972
FRIDAY, MAY 12. Professor E. A. Cooper, University Department of Anaesthesia, Newcastle upon Tyne: “In and Out”.

There will not be an ordinary meeting of the Society in March 1972 but arrangements are in hand with the University Department of Anaesthesia for a one-day Symposium on “Resuscitation and Intensive Care” on Saturday, March 18, 1972.

Meetings are held in the New Lecture Theatre, R.V.I., Newcastle upon Tyne at 8 p.m. Buffet suppers will be held as previously in the board room from 6.30 p.m. and coffee will be available in the ante-room to the lecture theatre from 7.30 p.m. onwards.

All communications should be addressed to the Honorary Secretary: Dr W. Ryder, Department of Anaesthesia, Royal Victoria Infirmary, Newcastle upon Tyne, NE1 4LP.