PRACTICAL PAEDIATRIC PROBLEMS

JAMES H. HUTCHISON

O.B.E., M.D.(Glas.), F.R.C.P.(Lond.),
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LLOYD-LUKE

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Professor of Child Health, University of Glasgow; Visiting Physician, Royal Hospital for Sick Children Glasgow; Consultant Paediatrician, The Queen Mother's Hospital, Glasgow

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This book is dedicated to

MY WIFE

PREFACE

THE invitation to write a book on practical paediatric problems is an almost irresistible challenge to one who has spent most of his professional life, as I have, in paediatric practice in hospital and in the homes of sick children. The fact that I have recently been immured in the "ivory tower" of a University Chair, if anything, heightens the challenge. The latter circumstance is, however, unrelated to the unexpected difficulty, encountered as the book took shape, of recognizing a practical paediatric problem. The experimental approach of today is quite likely to become the routine practice of tomorrow. Diagnostic and therapeutic methods which are looked upon as research tools in some hospitals have become matters of everyday routine in the large centres which introduced them. The present book is intended to be an account of clinical paediatrics as it has been experienced by a paediatrician in a large medical school with responsibility for both undergraduate and postgraduate teaching. There has always seemed to me to be a need for a textbook which in content lies somewhere between the usual undergraduate text (often of little value to the qualified physician) and the large work of reference by many authors (usually consulted only by the specialist). In this book I have tried to deal with the problems of the paediatrician and the family doctor in a fairly detailed fashion. Treatment in particular has been accorded sufficient space to be of value to the practising physician. References to original work and to books of a specialized nature have been included in every chapter as a guide to those who would wish to go more deeply into the subject.

It is obviously unavoidable that a textbook by a single author on a subject as vast as paediatrics must to some extent be selective; for the author must write only of what he knows. I have omitted from consideration diseases of the eyes, ears and skin of which others can write more authoritatively. Most textbooks of medicine deal with the common infectious fevers and I have, therefore, left them out in order to have more space for present-day paediatrics. My object, in short, has been to provide a manageable account of clinical paediatrics for undergraduates, for postgraduates specializing in paediatrics, and for general practitioners who find that a large proportion of their daily routine is concerned with the care of sick children. It is inevitable, and not necessarily disadvantageous, that a personal approach should be evident. None the less, the considerable space devoted to the neonatal period reflects the fact that 70 per cent of the total infant mortality in the United Kingdom now falls within the first twenty-eight days of life. Moreover, the major paediatric advances of recent years have been made

possible only by the evolution of new laboratory methods; for this reason the help to be obtained from the laboratory in many diseases has been discussed in some detail. The illustrations have been chosen with the sole object of amplifying the textual descriptions.

I count myself fortunate in having lived through a period of such rapid advances in medicine. I hope that in this book the reader will find a practical account of today's paediatrics, so different from that of thirty years ago when I qualified in medicine. It would be impossible for me to make suitable acknowledgements to the many colleagues, both senior and junior, who have helped to form the opinions and outlook of this book. I owe a special debt to Professor Stanley Graham and to the late Professor Noah Morris whose teaching and example set the feet of a young man upon the path of paediatrics. My colleague Dr. R. A. Shanks has read the manuscript of this book and I am grateful to him for many helpful suggestions. Mr. J. Devlin prepared the illustrations and gave me his expert advice. Finally, the work would have been impossible without the willing assistance of my secretaries Mrs. Margaret Stirling and Mrs. Dorothea Douglas.

I am indebted to the Editor of the *British Medical Journal* for permission to reproduce Fig. 65; to the Editor of the *Archives of Disease in Childhood* for permission to reproduce Fig. 62; and to the Editor of *Surgo* for permission to reproduce Fig. 57.

JAMES H. HUTCHISON

University Department of Child Health,
Royal Hospital for Sick Children,
Yorkhill,
Glasgow, C.3

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CHAPTER I

ASPHYXIA NEONATORUM

The term "asphyxia neonatorum" has long been used by clinicians to mean failure on the part of a newborn infant to establish spontaneous respiration. *Apnoea* would be a better name. It has not been customary to include in the term asphyxia neonatorum any subsequent failure to maintain respiration, although some of the causes of such failure as hyaline membrane disease, neonatal pneumonia and aspiration of meconium have been studied intensively by paediatricians and physiologists in recent years. The word "asphyxia" literally translated means absence of the pulse, but in its common usage today it means anoxia or anoxaemia. In the apnoeic newborn infant anoxia is associated with an excess accumulation in the blood of carbon dioxide. The clinician must have a sound understanding of the causes of asphyxia neonatorum, and of their effects, if his management of this extreme emergency is to be rational and effective. Indeed, an understanding of the aetiological factors will often enable him to foresee an emergency and so reach a better state of preparedness.

Aetiology

(A) **Foetal anoxia.**—In the great majority of cases the causes of asphyxia neonatorum have been operating before the birth of the baby. Unfortunately, there is at the present time no reliable method of measuring foetal anoxia. Slowing and irregularity of the foetal heart, or the intra-uterine passage of meconium are indications of foetal distress but they can, nevertheless, be absent even when the foetus is in imminent danger of death *in utero*. The probability of foetal anoxia can often, however, be deduced from the state of the mother before or during labour.

1. *Anoxic anoxia* results from an inadequate supply of oxygen. This may arise from a diminution of the oxygen tension in the mother's circulation, as in congestive cardiac failure, pneumonia, bronchitis and emphysema or during an eclamptic fit. A particularly frequent cause of maternal anoxia is the use of inhalational anaesthesia. Donald (1957) has stressed the disastrous effects of mismanaged anaesthesia, often complicated by inhalation of vomit, during labour. There is also great danger to the foetus if the mother's blood pressure is allowed to fall abruptly during spinal anaesthesia. On the other hand, the oxygen supply from mother to foetus is entirely dependent upon the efficiency of the placenta. Maternal toxaemia and hypertension lead to reduced

placental blood flow and this may exist for some time before the onset of labour (Morris *et al.*, 1955). Walker (1954) found evidence of a diminished transfer of oxygen due to placental insufficiency in post-maturity. The foetal life-line may also be cut when the placenta separates prematurely, as in placenta praevia or accidental haemorrhage. Finally, the foetus is in danger during a long and severe labour because his oxygen supply is reduced during every uterine contraction.

2. *Anaemic anoxia* exists when the foetal blood is so anaemic that it cannot carry sufficient oxygen to meet the needs of the tissues. This may arise in rhesus incompatibility or from foetal haemorrhage.

3. *Stagnant anoxia* can arise from compression, prolapse or knotting of the umbilical cord. Accidents to the cord can rarely be foreseen.

4. *Histotoxic anoxia* arises when the foetal tissues, especially the nervous system, are so poisoned by drugs or damaged by haemorrhage that the cells can no longer utilize the oxygen delivered to them. Every drug which has so far been devised for the relief of pain during labour is capable of depressing the foetal respiratory centre if unskilfully used. This is true of pethidine, trichloroethylene, and even more so of morphine, hyoscine and related drugs. Their dangers are particularly great during premature labours.

(B) **Natal and postnatal anoxia.**—It is important that the clinician should appreciate that the causes of foetal anoxia often produce the very circumstances in which further anoxia becomes inevitable during or immediately after birth. For example, foetal anoxia occurs in abruptio placentae which is a common cause of premature birth, and the respiratory centre of the premature infant is immature and functionally inadequate. It may, indeed, have been further depressed by foetal anoxia, drugs administered to the mother, or by raised intracranial pressure due to haemorrhage or cerebral oedema. Barcroft (1946) showed that when rendered anoxic the foetus makes vigorous gasping movements *in utero* so that his lungs may be deeply filled with irritating meconium. The premature infant may suffer the further disadvantage of a soft unduly pliable thoracic cage surrounded by a weak musculature, so that he is unable to produce the negative intrathoracic pressure required to expand his immature lungs. Infrequently asphyxia appears for the first time after birth due to some congenital abnormality which does not cause trouble during foetal life, such as a laryngeal web or a diaphragmatic hernia.

Pathology

Asphyxia produces congestive circulatory failure with over-filling of the chambers of the heart and pooling of the blood in the viscera (Morrison, 1961). The blood vessels are engorged. From the circulatory stasis and anoxic damage to the capillary walls petechial haemorrhages and oedema may result. If intra-uterine anoxia has been produced in

the premature infant from retroplacental haemorrhage, which increases the pressure in the intervillous space and so drives blood from the placental channels into the foetal circulation, widespread petechial haemorrhages may be found in the brain and viscera. The severity of these various changes varies in different organs. The basic lesions in the lungs tend to be congestion, accumulation of oedema fluid in the air spaces and interstitial spaces, primary atelectasis and patchy haemorrhages. There appears to be a close relationship, not yet clearly understood, between perinatal anoxia and the subsequent development of hyaline membranes in the alveolar ducts. A big defect in our knowledge concerns the mechanism whereby overloading of the pulmonary circulation is prevented in the normal newborn, and how the capillary bed of the lung is protected by the pulmonary arteries and arterioles. The development of intraventricular and subarachnoid haemorrhages, especially in premature infants, is well known. It is much more difficult to assess the importance and significance of cerebral oedema and petechial haemorrhages in the brain substance, especially in regard to their possible relationship to neurological disorders in later life. In the other organs, haemorrhages are the most common evidence of anoxia. Adrenal haemorrhages are uncommon but they are probably rarely compatible with survival. Furthermore, anoxia increases the severity of bleeding from traumatic lesions, for example, in subdural haemorrhage or subscapsular haematoma of the liver.

Clinical Features

The traditional descriptions of "asphyxia livida" and "asphyxia pallida" are still useful provided it is appreciated that the latter is only a more severe degree of the former and in which peripheral circulatory failure has supervened. Donald (1957) prefers the term "foetal shock".

The nature of the stimulus to breathe in the normal newborn infant has long been a subject of interest. Cutaneous stimuli, especially those from the area supplied by the fifth cranial nerves, seem to play an important role, but probably much less so than the low blood level of oxygen and high carbon dioxide level at the time of birth.* In fact, every healthy newborn is asphyxiated by normal adult standards. Asphyxia of a degree requiring special measures should be diagnosed if the period of apnoea following complete delivery of an infant from the mother lasts longer than one minute. In asphyxia livida the infant is apnoeic and cyanosed, but muscle tone is normal or increased, the heart-rate is over 100 per minute and regular in rhythm, good pulsation is palpable in the umbilical cord, and there may be flexion movements or grimacing

* It may be that the most important stimulus for the initiation of respiration is the hydrogen ion concentration (pH) in the blood at the moment of birth (Reardon *et al.* 1960).

on cutaneous stimulation. Recovery is heralded by periodic ineffectual gasps proceeding to more effective inspiratory efforts. These result in a clearing of the cyanosis. Finally, there is a welcome cry. The deadly asphyxia pallida may be present from birth or develop subsequent to a deteriorating state of asphyxia livida. The infant has a deathly grey pallor and flaccidity of muscles, the heart-rate is below 100 per minute, and may be irregular, pulsation in the umbilical cord is barely perceptible, and there is no response to stimulation. Recovery is preceded by an acceleration in the heart-rate and occasional gasps, but it is often not sustained so that death supervenes.

Assessment and Early Prognosis

In some hospitals special clinical methods of assessment of the degree of neonatal anoxia have been devised. Although these cannot accurately reflect blood chemical changes, their purpose is to give members of the staff some guide to those infants in whom continuing and possibly fatal respiratory difficulties may be expected. These infants can thereby be assured of more individual and careful supervision. Apgar *et al.* (1958) evolved a scoring system which evaluates five objective signs sixty seconds after complete delivery of the infant. These are skin colour, muscle tone, respiratory effort, heart-rate and response to stimulation. Each is given a score of 0–2. A total score of 10 indicates an infant in optimum condition. A mortality rate of 15 per cent may be expected when the score is 2 or less. However, Apgar's method permits of considerable subjective variation between different observers, and Auld *et al.* (1961) have found it unreliable as a method of prediction in individual infants. Miller and Calkins (1961) graded newborn infants by measuring the time (in minutes and seconds) which elapsed between the birth of the infant and the onset of spontaneous respirations, and subsequently recording the respiratory rates at frequent intervals for 48 hours. Infants who cleared their cyanosis in a few minutes and whose respiratory rates did not increase were classified in Grade I; when the cyanosis disappeared in a few minutes but the respiratory rates were high for an hour with a subsequent decline, the infants were placed in Grade II; when there was failure to lose the cyanosis or when the respiratory rate was increased by more than 15 per minute after the first hour the infants were placed in Grade III. In a group of infants born by Caesarean section and in a series of premature births all the neonatal deaths had been classified in Grade III. It is clearly impracticable to assess every newborn infant by these methods which demand experienced observers. However, this type of evaluation of infants known to be at special risk, such as those born prematurely, by Caesarean section or to diabetic mothers, would at the least ensure the adequate and frequent observation which they demand.

TREATMENT

When the birth of an asphyxiated infant can be foreseen as a reasonable probability from the condition of the mother before or during labour, the delivery should, if possible, be conducted in hospital where adequate facilities exist. The first essential in the management of an apnoeic infant is to avoid chilling. Indeed, no doctor should agree to

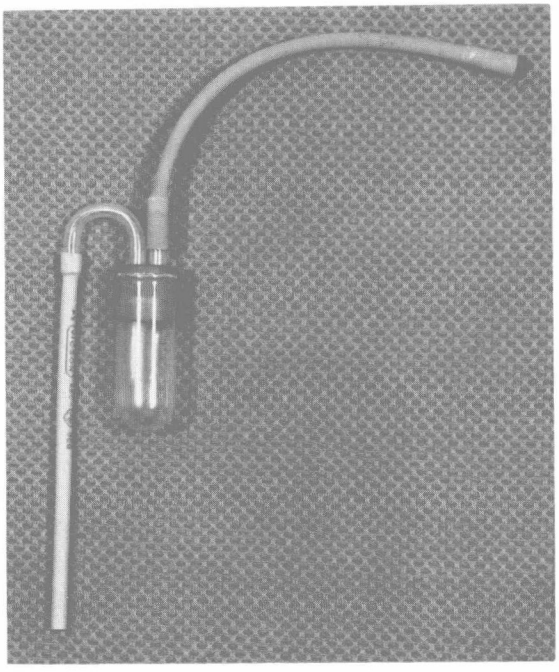

FIG. 1.—Mucus extractor with suitable trap and square-cut soft rubber end for infants.

conduct a confinement at home unless he can be assured of a room temperature not below 65° F. After complete delivery from the mother and section of the umbilical cord the infant should be received into a sterile towel which lies over warm non-woollen blankets. He should be placed with head at a lower level than his feet, on the attendant's lap or preferably on a resuscitation trolley or in a suitable cot. Drainage of fluid from the mouth and pharynx by gravity should be assisted by suction. If a mucus extractor is used the end for the infant's mouth must be of soft rubber with a square-cut open end, and there should be a trap between the infant's mouth and that of the operator (Fig. 1). In

hospital a mechanical or electric suction apparatus should be available. The nostrils should be sucked out as well as the pharynx. It is likely, in fact, that suction may do more good by stimulation than by removal of fluid from the respiratory tract. If the infant has not started to gasp within one minute pure oxygen must be given. This may be as a steady flow over the mouth and nose through a plastic filter funnel, or as intermittent short puffs from a small face-mask attached to the anaesthetic machine. The objectives thus far have been the avoidance of further shock by chilling, and the revival of the respiratory centre by getting oxygen to the brain after obtaining a patent airway. It is almost traditional to administer one of the analeptic group of drugs such as lobeline 3 mg. (1/20 gr.) or nikethamide 1 ml. The injection is best made into the umbilical vein which is then "milked" towards the infant. There is no doubt that these drugs, which act through the chemoreceptors of the carotid and aortic bodies, do initiate respiration in mildly asphyxiated infants. Unfortunately, they are ineffective in severely affected babies so that their real usefulness is open to question. Furthermore, in excessive doses they can be convulsant, and arterial thrombosis can follow accidental injection into an umbilical artery. We have formed the clinical impression that Vandid which can be given into the mouth or intravenously in doses of 25 mg. is a more effective drug and it appears to have a wide margin of safety.* Barrie *et al.* (1962) were only able to demonstrate consistent respiratory stimulation from nikethamide and Vandid. After spontaneous respiration has been established it is sound practice to raise the head above the level of the feet as respiration in the newborn is mainly diaphragmatic, and to aspirate the stomach contents with a sterile soft rubber catheter and 20 ml. syringe. This eliminates the danger of aspiration of vomitus into the lungs of an infant whose cough reflex is likely to be suppressed. Thereafter, if the infant's cyanosis is not completely cleared, he should be placed in an incubator or oxygen box of some type at an oxygen concentration of 35 to 40 per cent. Considerably higher concentrations may, in fact, be used safely for short periods in severely anoxic cases. On the other hand, mildly asphyxiated infants, who form the great majority, require no extra oxygen after respiration has become established.

The simple and conservative steps so far described will suffice to resuscitate the vast majority of apnoeic babies. There are, however, some severely asphyxiated infants who fail to breathe spontaneously in response to warmth, gentleness, pharyngeal suction, oxygen and analeptic drugs. Many of these are premature. An understandable lack of complacency about this kind of tragic ending to pregnancy has led to the development of some ingenious techniques of assisted respiration. None the less, they have been accompanied by some difficult

* Vandid is vanillic acid diethylamide.