PAEDIATRIC EMERGENCIES

A Practical Guide to
Acute Paediatrics

Tom Lissauer, MB, MRCP

PAEDIATRIC EMERGENCIES

A Practical Guide to
Acute Paediatrics

Tom Lissauer, MB, MRCP

Research Fellow, Perinatal Research Unit
and Honorary Paediatric Senior Registrar,
St Mary's Hospital Medical School,
London, W2

Published, in association with
UPDATE PUBLICATIONS LTD., by

MTP PRESS LIMITED · LANCASTER · ENGLAND
International Medical Publishers

Published, in association with
Update Publications Ltd., by
MTP Press Limited
Falcon House
Lancaster, England

British Library Cataloguing in Publication Data

Lissauer, Tom
Paediatric emergencies.
1. Pediatric emergencies
I. Title
618.92'0025 RJ370

ISBN 0-85200-436-2

Printed by Butler & Tanner Ltd, Frome and London

Tom Lissauer qualified from Cambridge University and University College Hospital, London, in 1973. After working as a senior house officer at The Hospital for Sick Children, Great Ormond Street, London and the Neonatal Unit at University College Hospital he spent a year in Boston at the Children's Hospital Medical Center as a Senior Resident. Since then, he has worked as Paediatric Senior Registrar at Northwick Park Hospital and Clinical Research Centre, Harrow, and at St Mary's Hospital, London. He is currently doing research in neonatal paediatrics at St Mary's Hospital Medical School, London. He is married to another paediatrician and has two young children.

Preface

The aim of this book is to provide a practical guide to help junior doctors to manage the important acute paediatric problems they are likely to encounter. The emphasis has been placed on the diagnostic problems and management when the child first presents. The approach taken is largely pragmatic, in contrast with the more theoretical approach of undergraduate teaching. As many doctors in general paediatrics are also required to perform neonatal resuscitation, a chapter on this topic has been included, but no attempt has been made to cover the specialized field of neonatal intensive care.

Several of the chapters have been published in a series of articles in *Hospital Update*. They have been thoroughly revised and many new chapters added.

It would have been impossible for me to have written this book without the help and encouragement of my wife, Dr Ann Goldman. She has read the book at each stage of its gestation and made many constructive suggestions and improvements. I am also grateful to Dr Paul Hutchins who has helped me considerably. Dr Doug Jones has provided helpful advice on the anaesthetic aspects and practical procedures and contributed the section on the insertion of central venous catheters. Many other colleagues have read sections of the book and I should like to thank Drs Ruby Schwartz, Terry Stacey, Andy Whitelaw, Rodney Rivers, John Warner, Sue Rigden, Susannah Hart, Mike Liberman and Bernard Valman. The staff of the

pdate Group especially the Managing Editor Mrs Anne Patterson, .ne Staff Editor Sharon Kingman and Illustrator Peter Gardiner have shown me much kindness and patience. Mr Phil Johnstone of MTP Press has edited the book most efficiently. I am most grateful to Mrs Ruth White who has typed the manuscript so willingly and competently.

Photographs

I would like to thank those who have so generously allowed me to use their photographs. Their names are listed below.
Chapter 3 Figure 4: Dr R. Snook; Chapter 4 Figure 2: Health Education Council; Chapter 4 Figure 3: Department of Diagnostic Virology, St Mary's Hospital, London, W2; Chapter 5 Figure 1: Dr H. A. Davies; Chapter 8 Figure 1: Dr A. Whitelaw; Chapter 8 Figure 3: Dr Hillas-Smith; Chapter 8 Figure 4: Dr K. Rogers; Chapter 8 Figures 8, 9: Dr M. M. Liberman; Chapter 10 Figures 2, 3: Dr Vic Larcher; Chapter 14 Figures 5, 6: Radiology Department, The Children's Hospital Medical Center, Boston; Chapter 15 Figures 1, 5, 6: Dr Peter Jaffe; Chapter 15 Figures 3, 4: Dr Paul Hutchins; Chapter 15 Figure 7: Professor T. E. Oppe; Chapter 16: *Family Group* appears courtesy of the Tate Gallery, London; Chapter 17 Figures 9a and b: Dr Paul Hutchins. Kari Hutchins kindly helped drawing Figure 2 in Chapter 3.

Contents

CHAPTER 1

Neonatal resuscitation

The perinatal mortality varies considerably between westernized countries (Figure 1). Although there has been a steady reduction, lives are still undoubtedly lost and brain damage sustained because babies are still being delivered in hospitals where competent neonatal resuscitation is not always available.

Ideally, all medical members of the paediatric and obstetric staff, anaesthetists, midwives and nurses working in neonatal units should be trained and skilled in the techniques of neonatal resuscitation. Frequent changes in staff necessitate repeated lectures and demonstrations, as well as the attendance of experienced staff at deliveries until new members are confident to conduct resuscitation.

Apnoea

The sequence of events following acute total asphyxia has been extensively studied in rhesus monkeys (Dawes, 1968). There is an initial period when the animal takes rapid shallow gasps. This lasts less than a minute and is followed by a period of apnoea known as primary apnoea. After one or two minutes the animal starts to gasp again. These gasps increase in depth and frequency but then become shallow and less frequent until the last gasp. Thereafter there is no further spontaneous respiratory effort. This period of secondary or terminal apnoea ends in circulatory failure and death. In the rhesus monkey, the sequence of events up to the last gasp takes about eight minutes (Figure 2).

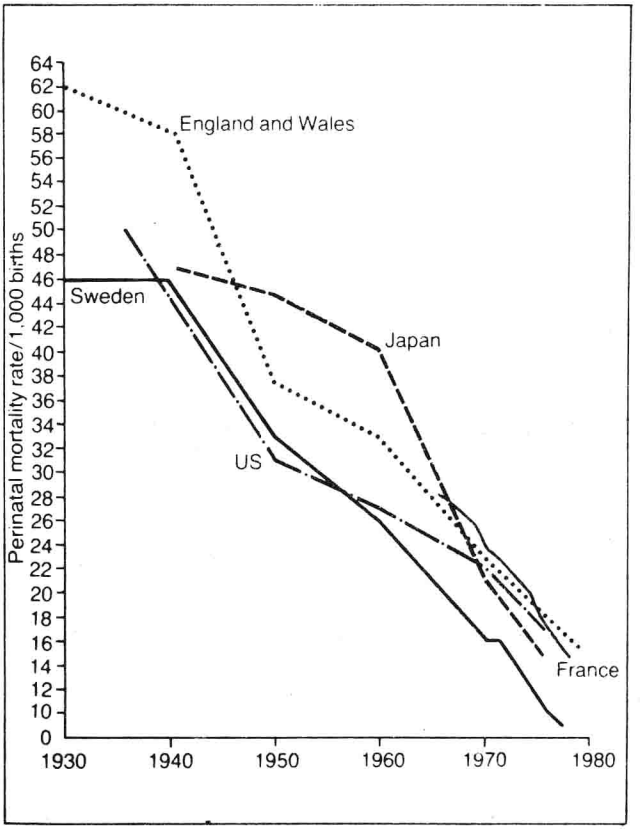

Figure 1 *Perinatal mortality in various countries*

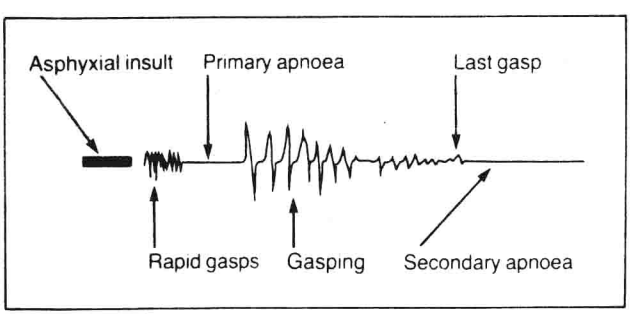

Figure 2 *Sequence of respiratory events following total asphyxia at birth in a rhesus monkey*

Primary apnoea

The human baby usually experiences intermittent partial hypoxia during labour rather than acute total asphyxia. However, the sequence of respiratory events seen in animal experiments is relevant in part to the resuscitation of the asphyxiated neonate. If there has been only minimal or moderate asphyxia the neonate may be born in primary apnoea. These babies look blue, have a heart rate greater than 100 beats per minute, and although their muscular tone is reduced there is flexion of their limbs and they make reflex responses to aspiration of their nostrils. If given appropriate stimulation regular respirations will soon follow.

Secondary apnoea

If there has been more severe asphyxia the neonate may be born in secondary apnoea. These babies look white, have a heart rate less than 100 beats per minute, and are flaccid. They do not make any reflex response to suction, and will only be successfully resuscitated if positive pressure ventilation is given. The contrasting clinical features of primary and secondary apnoea are listed in Table 1.

Table 1 Contrasting clinical features of primary and secondary apnoea

	Primary apnoea	*Secondary apnoea*
Heart rate	> 100	< 100
Colour of trunk	Blue	White
Reflex response to stimulation	Gasps or coughs	None
Posture	Flexion of limbs	Flaccid
Blood pressure	Normal or raised	Hypotension

In baby monkeys, the longer the delay in initiating resuscitation after the last gasp, the longer the time until the first spontaneous gasp. For every minute's delay in resuscitation after the last gasp, the next gasp is delayed by two minutes. The dramatic fall in heart rate following prolonged total asphyxia is shown in Figure 3. The baby monkey rapidly becomes severely acidotic from combined respiratory and metabolic acidosis. The immediate rapid elevation of Pco_2 from hypoventilation causes a respiratory acidosis, and the change from aerobic to anaerobic glycolysis causes a metabolic

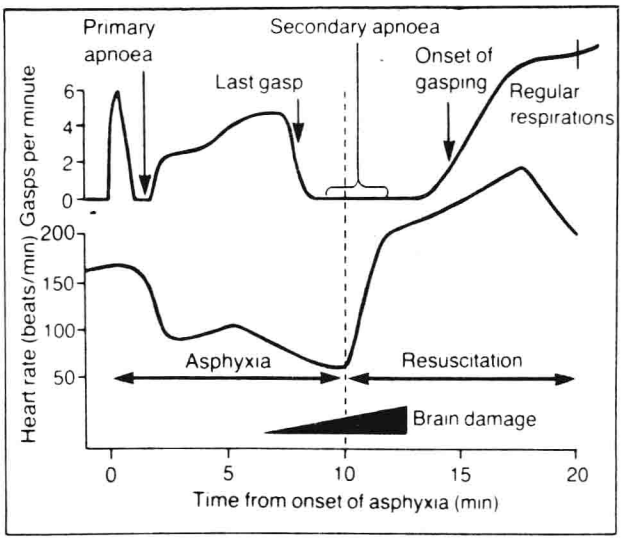

Figure 3 *Response to resuscitation in a rhesus monkey*

acidosis. After 10 minutes of total asphyxia, the pH drops from 7.3 to 6.8, the Pco_2 increases from 6 kPa (45 mmHg) to 20 kPa (150 mmHg) and the Po_2 falls from 3.4 kPa (25 mmHg) to virtually zero. Brain damage begins after approximately eight minutes of total asphyxia and is maximal after 12 to 13 minutes.

These findings emphasize the importance of rapid resuscitation of all severely asphyxiated infants. The rapid increase in the heart rate following resuscitation during secondary apnoea is also shown in Figure 3. If the heart rate is not accelerating within 30 seconds of adequate ventilation, this suggests that severe hypoxia has been sustained.

Anticipating and preparing for resuscitation

During fetal life, the lungs are filled with fluid and the circulation modified so that arterial blood from the placenta supplies the fetus with oxygenated blood. Following the transition to normal breathing at birth, the circulation has to adapt to extrauterine life, and this is triggered by tactile and thermal stimuli, as well as central chemosen-

sory mechanisms. Some of the reasons why a baby may fail to establish normal respiration soon after birth are listed in Table 2.

Table 2 Some causes of failure to establish normal breathing at birth

Central
Respiratory depression:
Prolonged fetal asphyxia in labour
Sedatives/analgesic drugs given to mother
Trauma to brain stem:
Haemorrhage
Herniation
Peripheral
Congenital malformations of respiratory tract:
Pulmonary hypoplasia (often with renal agenesis, characteristic facies with prominent epicanthic folds, flattened nose and low-set ears and maternal oligohydramnios—Potter's syndrome)
Diaphragmatic hernia
Airway obstruction

All high risk deliveries should, where possible, take place in hospitals experienced in the care of sick neonates. Regular checks should be made on fetal well-being during pregnancy and continuous fetal heart monitoring and fetal blood sampling may be employed to detect fetal distress early during labour. In many instances the need for neonatal resuscitation can be anticipated and the paediatrician called in advance (Table 3), but these criteria fail to predict the need for resuscitation in some cases, and it is imperative that a trained doctor is immediately available at all times.

Before the delivery of a baby likely to require resuscitation, the paediatrician should check that all the equipment is available and working (Figures 4 and 5 and Table 4). Details of the obstetric history, labour and, in particular, any drugs recently given to the mother should be obtained while waiting for the delivery. This can also provide an opportunity to introduce oneself to the mother and explain that one is there to look after the baby. It is always necessary to have some assistance, and for particularly high-risk infants and multiple births, the presence of an additional paediatrician is valuable.

Table 3 High risk deliveries

Caesarian section
Breech
Forceps delivery (except low forceps)
Multiple births
Large doses of analgesic/sedative drugs given late in labour
Fetal distress
Thick meconium in liquor
Abruptio placentae, placenta praevia
Preterm
Intrauterine growth retardation
Rhesus isoimmunization
Diabetes
Severe toxaemia
Rupture of membranes > 24 hours

Figure 4 A resuscitaire incorporating an overhead heater and oxygen supply with pressure-gauge and suction. This new born baby's nose and mouth are being aspirated, prior to him being dried, wrapped in a warm towel and handed to his mother to hold

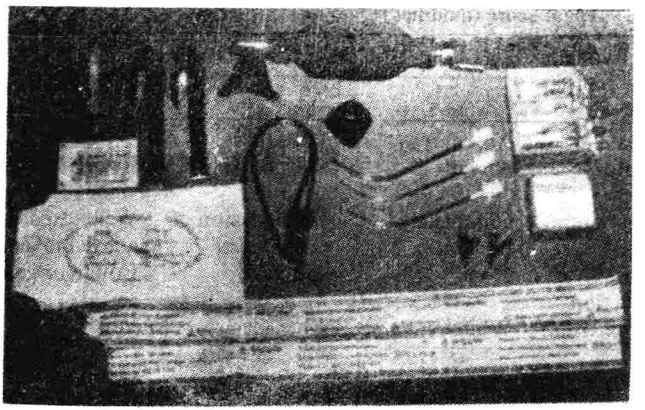

Figure 5 *Additional equipment required for neonatal resuscitation*

Table 4 Check list before delivery

1. Radiant warmer on
2. Oxygen supply and connections
3. Self-inflating bag and mask
4. Oral mucus extractor and fine suction catheters
5. Laryngoscopes with straight blades for term and preterm infants; check the light bulbs
6. Endotracheal tubes and introducer
7. Drugs: naloxone, adrenaline, dextrose, calcium chloride, sodium bicarbonate
8. Umbilical artery catheters

Apgar score

The condition of the neonate is commonly assessed using the Apgar score shown in Table 5 (Apgar, 1953). Heart rate and respiratory effort are by far the most important of the five clinical features measured, and it is these which determine the subsequent course of action. Apgar scores are routinely determined at one and five minutes. The one minute score is usually the minimum score, while the five minute score gives an impression of the infant's progress. A breakdown of how the score was derived as well as the total should always be recorded.

Table 5 Apgar score (modified)

	Score		
	0	*1*	*2*
Heart rate	Absent	< 100	> 100
Respiratory effort	Absent	Gasping or irregular	Regular or crying vigorously
Muscle tone	Completely flaccid	Some flexion of extremities	Well flexed with active movements
Response to nasal suction	None	Grimace	Cough or gasp
Colour of trunk	White	Blue	Pink

High risk deliveries

All newborn babies have fluid in their mouth and pharynx, which is a mixture of amniotic fluid, lung liquid and blood. Immediately after the head has been delivered the pharynx should be gently aspirated with a mucus extractor or soft suction catheter. Care should be taken not to touch the back of the pharynx as this may produce reflex bradycardia or apnoea from vagal stimulation. The clock above the resuscitaire should be started when the baby has been delivered. The infant should be placed on the resuscitaire and any remaining fluid in the mouth and nostrils gently and quickly aspirated. He should then be rapidly dried and assessed. On average, infants make their first respiratory effort within six seconds of birth, and most within 20 seconds. Rhythmic respirations have usually started by 30 seconds, but if no respiratory effort has been made by that time, active resuscitation should be started. The heart rate can be monitored either with a stethoscope on the left anterior chest wall or by palpation of the pulsation of the umbilical cord.

Most newborn babies will not need any resuscitation (Apgar score 8 to 10) and after they have been dried can be returned to their mothers.

Resuscitation

The appropriate resuscitation procedure will mainly be determined by the respiratory pattern and heart rate (Figure 6). Alternatively,

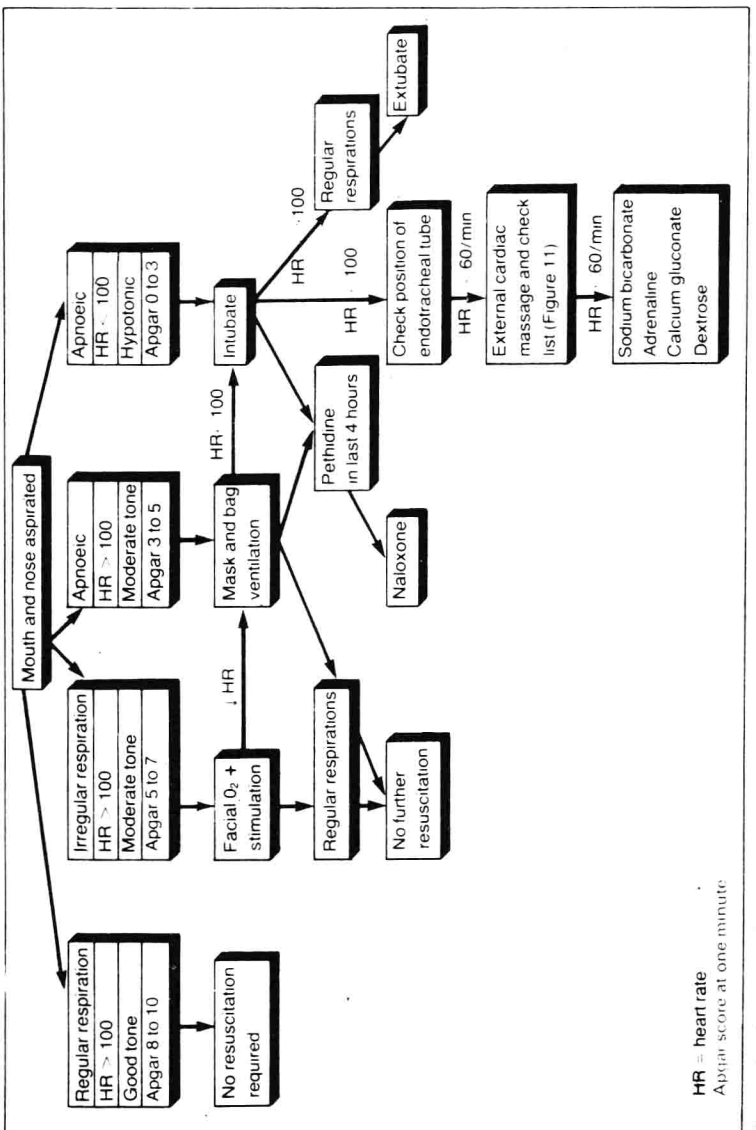

***Figure 6** Sequence for resuscitation*

the Apgar score at 1 minute can be used as a guide. In general those with an Apgar score of 5 to 7 will respond to stimulation and oxygen, those with an Apgar score 3 to 4 will respond to bag and mask ventilation, while those with an Apgar score of 0 to 2 need immediate endotracheal intubation. Measuring the Apgar score at 1 minute has the disadvantage that it tends to obscure the importance of continuous assessment of the heart rate, respirations and overall condition of the baby.

Irregular respiration

The delayed onset of regular respirations in this group of infants is usually secondary to the analgesics or sedatives given to the mother, or caused by mild to moderate hypoxia. Gasping often occurs in response to pharyngeal suction, and oxygen administered over the face will not only provide oxygen-enriched air but the flow of cold gas over the face will also act as an additional stimulus to breathing.

Apnoea

If the baby is apnoeic but the heart rate is over 100 per minute, ventilation should be started with a suitably sized mask and self-inflating bag applied over the mouth and nose, with the head slightly extended. Ventilation should be maintained at a rate of 30 to 40 breaths per minute and the chest wall should be seen to be moving. If satisfactory oxygenation has been achieved, the heart rate will usually accelerate and the colour rapidly improve. Once the baby is breathing regularly without assistance, resuscitation can be stopped. It is important to keep the baby warm and dry throughout the procedure.

Ventilation by bag and mask is relatively simple to perform, can be instituted rapidly and will usually initiate a reflex gasp without the trauma of intubation. Its main disadvantages are that it may be difficult to achieve effective ventilation and care needs to be taken to ensure that the head is correctly positioned and that there is a satisfactory seal between the mask and the face. In addition, the abdomen may become distended, and this may further embarrass establishing adequate respiration. If the heart rate does not improve rapidly or the breathing become regular, endotracheal intubation should be carried out.