Care of the Newborn in Developing Countries

G. J. Ebrahim

Macmillan Tropical Community Health Manuals

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Macmillan Tropical Community Health Manuals
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CARE OF THE NEWBORN IN DEVELOPING COUNTRIES

G. J. EBRAHIM

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To my wife

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Preface

This book first appeared under the title 'The Newborn in Tropical Africa'. On the advice of many colleagues, I have expanded the text to discuss the general principles of newborn care in the context of the special circumstances of the developing countries.

The book grew out of notes for reference and personal use made by me, first when serving as a single-handed paediatrician in Dar-es-Salaam, and lately as tutor to the WHO/UNICEF sponsored Course for Senior Teachers of Child Health. In the latter capacity I was able to study perinatal problems in the rural areas of several countries. Even though the diseases of the neonatal period are not markedly different in the developing countries, the approach to a particular problem and methods of care may be different – as in the case of the low birth weight baby or in infant feeding, to take just two examples.

Teaching of medical students and auxiliaries is a special challenge. Many of them will have to work in remote areas with very little equipment and poor facilities. Hence the emphasis in teaching has to be on local needs, simple diagnostic reasoning and practical regimes of management and therapy. Teachers from several institutions of medical training have sent helpful comments and suggestions which have been incorporated in the book. In addition many colleagues working on their own in remote rural areas with little access to medical libraries have written to say that they found the previous edition useful. It is my hope that the present edition will continue to serve this useful purpose not only in countries of tropical Africa but also on a wider scale in other parts of the developing world.

Several individuals have given help in the preparation of the book; to all of them I am grateful, and especially to Dr. Alan Murdock, of the Children's Hospital of Eastern Ontario, and to Dr. Edison Altamirano of Universidad Central del Ecuador. My wife has edited the text and corrected flaws in the manuscript in the various stages of its preparation. Without such help and lively criticism the text would not be very readable.

If the book achieves its objective of helping to standardise the care of the newborn in developing countries, all of us who have been involved in its preparation will feel that our labours have been rewarded.

Care of the Newborn in Developing Countries is one of five volumes intended for health centre personnel. Each volume is aimed at specific members of the health team, and *Care of the Newborn* is intended for the midwife and the 'doctor' in the team.

G.J. EBRAHIM

Chapter 1

Problems of the Newborn

The problems of the newborn in the developing countries have not yet been clearly defined. The statistics of teaching hospitals show consistent improvement over the years[1] and thus illustrate the benefits of a well-organised obstetric-paediatric service to the community. In all large cities where the teaching hospitals are situated, the populations served are often mobile, and so comparable community figures from one year to another are difficult to obtain. In Dar-es-Salaam it is estimated that about 75 per cent of all deliveries take place in hospital as compared with the overall national figure of 20 to 25 per cent,[2,3] and similar figures are also true for other developing countries. This indicates the very large number of births occurring in rural areas under traditional methods of midwifery, without any antenatal care. The associated dangers of low birth weight, birth trauma, intrapartum anoxia, infection and many others are all too familiar.

In the large maternity hospitals, with all available facilities, the perinatal mortality is in the region of 50 to 60 per thousand. In the rural areas it must be much higher. Many victims of traditional midwifery and poor neonatal care are not brought to the knowledge of the medical profession and remain unrecorded. In the rural Punjab perinatal mortality has been reported to be 73.5 per thousand live births.

The perinatal mortality, however high, constitutes only the tip of the iceberg; there will be a much larger proportion of newborns who do not die but are maimed or handicapped. It is therefore an urgent necessity to develop health and social services in rural areas to prevent this damage to human potential.

It is encouraging to know that in many rural areas there is now an ever increasing demand for Western type of medicine – in Tanzania, for example, 122.09 thousand antenatal attendances were recorded in 1965 as against 68.6 thousand in 1961 and 6.7 thousand in 1951. In comparison, confinements under medical supervision have been slow to increase. In 1961 the number of deliveries conducted by rural clinics and health centres was 22,643; this figure rose to 45,354 in 1964.[4] It is

hoped that when popular use of antenatal and obstetric services has been maintained over a number of years, more data will accumulate on the social, medical and administrative problems of the parturient mother and her newborn (figure 1.1).

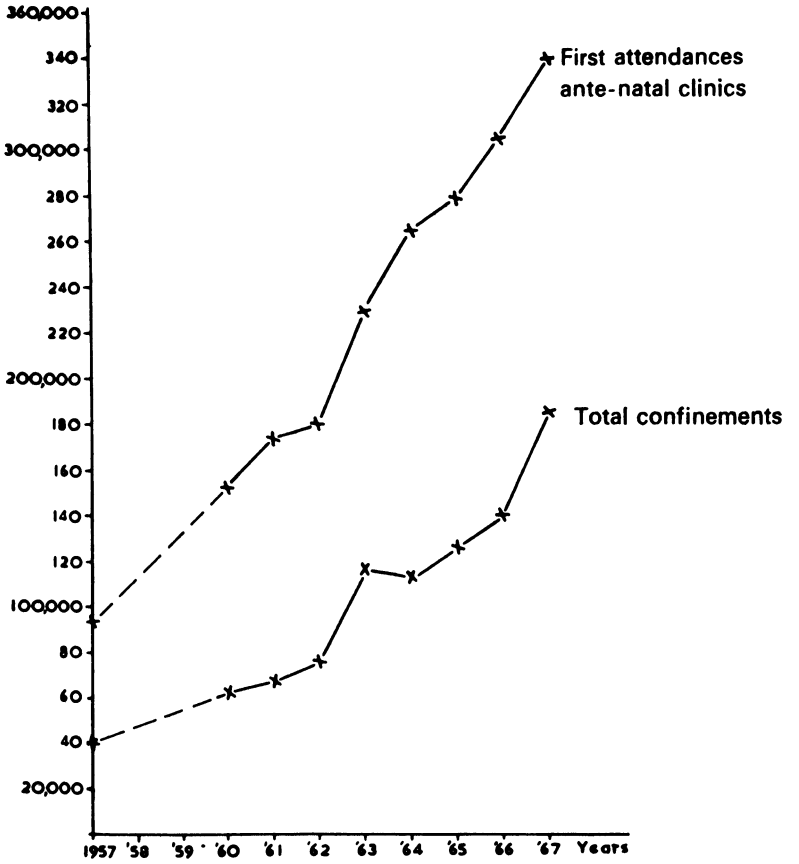


FIG 1.1 Acceptance of institutional midwifery (Tanganyika)

Several biological problems of the newborn are still being defined and the minimum in perinatal mortality is yet to be achieved even in more sophisticated communities. With persistent effort, however, further reduction in present mortality rates can certainly be attained; for example, in England and Wales, perinatal mortality has dropped from 38.5 per thousand in 1948 to 35.1 per thousand in 1958 until it

reached 30.8 per thousand in 1962, and 21 per thousand in 1973. And yet these figures are high when compared to those of the Scandinavian countries for the same years. The perinatal mortality survey in England in 1958 demonstrated how mortality can be reduced by better discernment of patients needing hospital care, by early recognition of disease and treatment and by good standards of antenatal care.

Large scale acceptance of hospital deliveries in urban areas is a healthy sign; however, in rural areas people are still reluctant to have institutional deliveries, with the result that in many health centres empty maternity beds are to be found. In certain cases this may be because people wish to follow traditional customs and practices associated with childbirth, which may not be possible in an institution. In all instances, therefore, it is important to inquire about local customs concerning disposal of the placenta, cord care and disposal of the cord stump, feeding of the postpartum mother and pre-lacteal feeding of babies; where possible, some relaxation of the rules at the health centre may be allowed so as to make institutional delivery more acceptable.

The advantage of institutional deliveries with availability of skilled supervision and associated prenatal and postnatal care do not need to be stressed. On the other hand, growth of services should take place to satisfy demand, otherwise the existing facilities tend to be swamped. It is to be deplored that this is the trend in most urban areas so that antenatal clinics are overcrowded, most mothers are discharged from hospital within 24 hours after delivery, and often the deliveries have to be conducted in overcrowded labour rooms by overworked staff. The standard of care is bound to fall under such conditions and, as a result, one often sees cord sepsis and neonatal tetanus in babies who have had the benefits of hospital deliveries.

Most babies, on discharge from hospital, are taken to live in crowded homes amidst unhygienic environments. The training of the mother in the care and handling of her baby is therefore extremely important, especially so in the case of the low birth weight baby or the first-born. The mother observes the way babies are handled by nurses and doctors in hospital and learns. If her stay in hospital is drastically cut, this important aspect of health education is lost.

At home two aspects of care need special mention: (a) avoidance of infection especially respiratory and through the cord stump, and (b) maintenance of nutrition.

Establishment and maintenance of lactation is important in the tropical mother, to such an extent that any baby that is not breast-fed has virtually no chance of survival. For most mothers in the tropics, breast feeding is not a problem but in urban areas, where the effects of commercial advertising and sophistication are catching up with mothers, bottle feeding is increasingly becoming a problem.

The approach to the problems of the newborn should be in the fields of:

(1) Medical care.
(2) Administration.
(3) Training.

MEDICAL CARE

There is already a popular demand for antenatal care. Facilities should be widespread and a minimum of six antenatal visits during pregnancy should be the aim. This aspect of prenatal paediatrics should be understood by the mother as well as the health worker. The quality of care is important and in areas where existing services are in danger of being swamped, consideration should be given to expanding them.

Regular antenatal visits will enable the health worker to determine the obstetric, medical or social criteria on which to decide whether a patient should be delivered at the health centre or referred to hospital.

Besides antenatal care, a good standard of health throughout childhood, adolescence and adult life is necessary for successful reproduction. The physical health of the mother, her life experiences of illness, of nutrition and immunisation, her education, the social and cultural conditions in which she has been brought up – all influence the perinatal mortality and morbidity. Therefore the care of the mother should be a continuing process beginning from childhood and well founded in a good maternal and child health programme.

ADMINISTRATION

The health of the mother is influenced by what happens to the family as a whole. The family in turn is affected by the physical, social and environmental conditions of the community within which it lives. Most of the problems of mothers and their babies are community problems that can be solved by ensuring adequate health coverage in a whole area.

In health planning, it is not possible to separate child care from maternal care. Therefore, liaison should exist between health workers in these fields. This is easier to achieve at a health centre than in a big urban hospital where a large number of workers may be involved. Regular inter-departmental visits and consultations on administrative matters help to create a spirit of team work.

TRAINING

Care of the newborn should form an integral part of obstetric training for physicians, nurses and health auxiliaries. To be realistic, such train-

ing needs to include a study of existing problems and their practical solutions, such as practical methods of caring for the low birth weight baby, or the common forms of neonatal sepsis and their causes.

In many countries it is now the policy to integrate the traditional midwives into the national health service. They are given simple training at the local hospital or health centre in hygiene and asepsis and also in the discernment of high risk pregnancies. For example, in a district in rural Punjab, deaths from neonatal tetanus were 20 per thousand live births, but after implementing a programme of training for traditional birth attendants in clean cord handling techniques and immunisation of the pregnant mother with tetanus toxoid the mortality was reduced by two-thirds in a period of six months. In the Sudan, training programmes for traditional birth attendants have been in existence for more than fifty years and such attendants conduct the bulk of deliveries in the rural areas with measurable impact on perinatal mortality.

Chapter 2

Perinatal Mortality

The perinatal mortality rate, together with the maternal mortality rate, serves as a good index of the effectiveness of a maternity service. It has two components: the stillbirth rate which is calculated per 1,000 live births, and the neonatal death rate which is also calculated per 1,000 live births. Such a gauge for the health problems of the newborn has the added advantage of a team approach involving the obstetrician, the paediatrician and the pathologist.

It is reasonable to assume that high perinatal mortality is prevalent in many developing countries, especially in rural areas (see figure 2.1). Marriages tend to occur early in most peasant societies. Among the Wazaramo of Tanzania, marriage takes place soon after the onset of menarche; in the rural areas of Hyderabad, India, the average age at marriage is 13 years. Thus a large number of women embark on their reproductive career at an early age, often before their own growth is complete. The average age of the mother at the time of first delivery is 16 years in rural Hyderabad and 17 years in rural Punjab. Maternal depletion due to uninterrupted cycles of pregnancy and lactation, chronic ill-health, lack of maternal care and skilled supervision during childbirth added to the unhygienic environment of most homes where births take place, provide the background against which this high mortality occurs. In urban areas, where maternity services have been established, a consistent improvement in the mortality figures can be seen (table 2.1).

Stillbirths (late foetal deaths) make the main contribution to perinatal mortality. As a matter of fact, the reduction of perinatal mortality in some western countries in recent years is largely due to reduction in stillbirths (table 2.2).

Perinatal mortality is influenced by the personal and social characteristics of the mother as well as the quality of medical and obstetric care she receives. In the British perinatal mortality survey[6] it was possible to identify two broad categories. In one group death could have been avoided by good obstetric care, and so development of obstetric

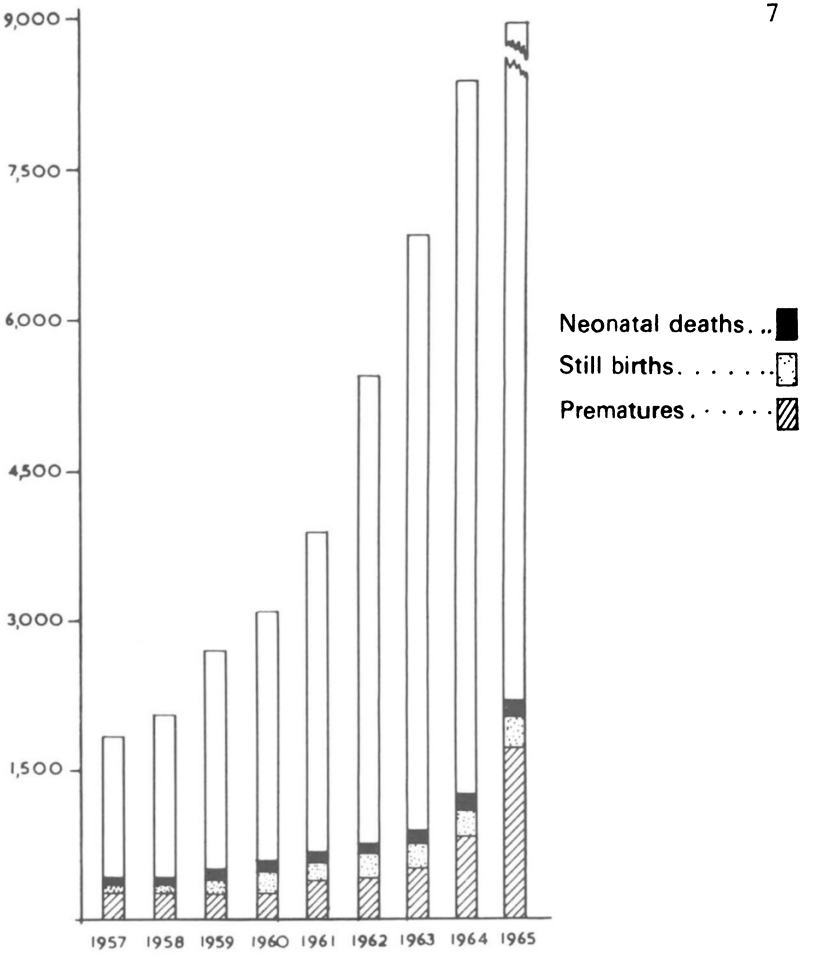


FIG. 2.1. Perinatal mortality in Dar-es-Salaam

services and improving the standards of care will be one way of reducing perinatal mortality. In the second larger group, perinatal mortality was found related to personal, social and nutritional factors affecting the mothers. The reproductive physiology of the mother is influenced not only by the events in the current pregnancy but also by her life experience of nutrition and infection and by her achievement of full growth potential.

The more specific causes of stillbirth or early neonatal death may exert their influence antepartum or during the process of childbirth.

TABLE 2.1 Reduction of perinatal mortality in East Africa

	Total deliveries	*Still birth rate per 1,000 live births*	*Neonatal death rate per 1,000 live births*
Mulago Hospital, Kampala			
1956/1957/1958	14,499	73.77	46.30
1962	4,886	52	28
1965	11,713	30	20
1969	14,464	26.1	24.3
Ocean Road Hospital, Dar-es-Salaam[5]			
1957	1,637	50	27.2
1961	3,707	42.8	27.0
1965	10,778	29.4	14.6

TABLE 2.2 Reduction of perinatal mortality in Western Europe

Country	*Perinatal deaths*		*Late foetal deaths*		*Deaths in 1st week*	
	1956	*1966*	*1956*	*1966*	*1956*	*1966*
England and Wales	37.6	26.7	23.4	15.6	14.1	11.1
Sweden	28.8	19.0	17.0	10.0	11.7	8.8
Italy	45.4	36.2	26.6	19.7	18.8	17.1

Analysis of such causes helps to identify the 'at-risk' pregnancy well in advance so that a plan of management or referral can be devised. It is also likely that for every stillborn baby there are several who are born with severe handicaps which could have been prevented.

STILLBIRTHS

In a study of 152 consecutive stillbirths over a period of six months, the commonest associated factors in the antenatal period were:

(1) Lack of antenatal care in 17 (11.2 per cent).
(2) Antepartum haemorrhage in 16 (10.6 per cent).
(3) Maternal anaemia of less than 7.5 g per cent in 17 (11.2 per cent).
(4) Toxaemia of pregnancy in 14 (9.24 per cent).

Perinatal studies in several countries have demonstrated the importance of antenatal care. This stands out as the single most important factor in promoting perinatal health. The British study showed that of all the factors contributing to perinatal mortality, lack of antenatal care was the most powerful – it is responsible for five times the overall mortality

rate in the survey. This is also the experience in the developing countries and it is now the usual practice to treat the 'unbooked' case as an emergency. For example, in Tirupati, India, perinatal mortality in 'unbooked' mothers was 15 per cent and the stillbirth rate was three times that due to abnormal delivery.

Low birth weight is a major contributing factor. In Kampala, 40 per cent of stillbirths weighed less than 2,500 g. In the above study of 152 consecutive stillbirths 39 of the babies (25.7 per cent) weighed 2,040 g or less, 82 (54.1 per cent) weighed more than this and in 31 (20.2 per cent) the weight was not known. The intrauterine growth of the foetus and the subsequent weight of the baby at birth are closely related to the outcome of pregnancy. In Western Europe, perinatal mortality is in general 30 to 35 times higher in infants weighing 2,500 g or less at birth.

Another important factor is congenital abnormality. Out of 152 stillbirths, 11 babies (7.26 per cent) showed a congenital abnormality, the most common abnormality being anencephaly (in 8 cases).

The common obstetric factors associated with stillbirths were:

(1) Cord complications (prolapse or presentation) in 15 (9.9 per cent).
(2) Obstructed labour in 9 (5.6 per cent).
(3) Prolonged second stage in 4 (2.64 per cent).
(4) Placenta praevia in 2 (1.32 per cent).

Variations in the relative importance of these factors are seen from one place to another depending upon anthropometric and socio-economic characteristics of the communities concerned. For example, ruptured uterus was the main maternal contributory factor seen in the Baganda[1], and toxaemia of pregnancy was rare.[1,8]

In the absence of post-mortem findings, only a clinical diagnosis is possible. The British perinatal mortality survey has shown the importance of anoxia; in more than half the stillbirths surveyed, anoxia alone (48.2 per cent) or in association with cerebral birth trauma (7.8 per cent) was an important cause of death.[6]

NEONATAL DEATHS

Seventy-five per cent of all early neonatal deaths (in the first week of life) are seen in the low birth weight baby. The other major causes of early neonatal deaths are:

(1) Difficulty in establishing and/or maintaining respiration.
(2) Cerebral birth trauma.
(3) Congenital abnormalities.
(4) Infections.

After the first week of life an important factor in the survival of the baby is the availability of breast milk. A baby born in a rural area of the developing world with no access to breast milk has virtually no chance of survival. In this respect, the establishment of successful lactation is as crucial as antenatal care and supervised labour.

After the first week of life, infection, both pulmonary and extra-pulmonary, is an important cause of death. Force feeding by hand, resulting in inhalation pneumonia, traditional methods of cord care giving rise to a high incidence of neonatal tetanus, and the overcrowding and lack of hygiene in many homes producing respiratory or other sepsis, are the major contributory factors to high mortality in the late neonatal period.

Chapter 3

Medical Disorders in Pregnancy

The effects of maternal illness on the foetus vary according to the duration and the severity of the illness. The effects of several infective illnesses have been considered in the previous chapter.

The purpose here is to consider the effects on the foetus of some of the common disorders of pregnant women. These are:

(1) Toxaemia of pregnancy.
(2) Late pregnancy haemorrhage.
(3) Anaemia.
(4) Diabetes.
(5) Malaria.

The adverse effects of many of these illnesses are due to interference in the placental circulation so that the supply of nutrients and oxygen to the foetus is affected. Thus, antepartum haemorrhage can have lethal consequences for the foetus, and toxaemia is frequently associated with intrauterine growth retardation and hypoglycaemia in the early newborn period. The data below show the overall effects of each of these conditions on the health of the newborn.

TOXAEMIA OF PREGNANCY

The incidence of toxaemia varies a great deal from one geographical region to another; it is the most common disorder of pregnancy in the coastal region of Tanzania but is comparatively rare in Uganda. The reasons for this variation in incidence, whether genetic, dietetic or geographic, are still obscure.

Toxaemia is defined along the following lines:

(1) *Pre-eclamptic toxaemia* — presence of any two of the following three symptoms
  (a) Blood pressure of 140/90 mm Hg and above.
  (b) Albuminuria.
  (c) Ankle oedema.
(2) *Eclampsia* — toxaemia as above plus convulsions.

It has been found that foetal mortality is about 24 per cent higher in mothers with toxaemia of pregnancy. In some studies, the foetal prognosis has been related more to the duration than to the severity of the symptoms of toxaemia.[16]

Birth weight

In a study of 368 cases of toxaemia [17] who delivered in hospital, the mean birth weight was 2,550 g as compared with the overall mean birth weight of 2,950 g. It is possible that interference with the placental circulation in toxaemia produces foetal growth retardation.

Foetal outcome

In this study, 73 pregnancies (19.8 per cent) did not come to fruition. The losses were as follows:

Stillbirths – 44 (60 per cent).
Neonatal deaths – 21 (28.7 per cent).
Abortions – 8 (10.9 per cent).

The highest incidence of stillbirths was found with accidental haemorrhage, eclampsia and severe pre-eclamptic toxaemia in the mother.

Good antenatal care stands out as the single most important contributory factor to the wellbeing of the newborn in this condition. Table 3.1 compares the foetal outcome between booked and unbooked cases of toxaemia of pregnancy.

TABLE 3.1 Foetal outcome in booked and unbooked cases of toxaemia

Mother	*No. of babies born*	*Total survived*	*Died*		*Abortion*	*Loss %*
			S.B.	*N.N.D.*		
Unbooked	19	12	4	1	2	36.84
Booked	349	281	38 (+3*)	18 (+2*)	2	19.4

* pairs of twins S.B. = stillbirths N.N.D. = neonatal deaths

It is likely that in the absence of regular antenatal supervision the disease process remains unchecked and may progress to a lethal outcome.

LATE PREGNANCY HAEMORRHAGE

This complication of pregnancy is associated with poor foetal growth and survival, especially in the case of accidental haemorrhage. Out of

12,727 deliveries conducted at the Ocean Road Hospital, Dar-es-Salaam, there were 60 mothers (0.5 per cent) with late pregnancy haemorrhage. These were subdivided as follows:

Accidental haemorrhage 28
Placenta praevia 22
Others 10

The type of delivery and foetal outcome in each group are set out in table 3.2.

TABLE 3.2 Foetal outcome according to method of delivery

	Total no. of mothers	*Method of delivery*			*Deaths*		*Low birth weight*
		S.V.D.	*C.S.*	*Other*	*S.B.*	*N.N.D.*	
Accidental haemorrhage	28	21	2	5	18	2	11
Placenta praevia	22	3	17	2	2	4	12
Others	10	9	1	–	5	1	4

S.V.D. = spontaneous vertex delivery C.S. = caesarean section

ANAEMIA

Maternal anaemia is associated with an increased risk of low birth weight and perinatal mortality. In one study in East Africa [18] it was found that among mothers whose haemoglobin was 7.4 g per cent or less at the time of delivery, the incidence of low birth weight (below 2,500 g) was 42 per cent and the stillbirth rate was 147.1 per 1,000; in mothers with haemoglobins of 8.8 g per cent and above, the incidence of low birth weight was 12.7 per cent and the stillbirth rate was 51.0 per 1,000. Similarly in Malaysia it has been reported that in mothers with a haemoglobin of 6.5 g per cent or less, the incidence of low birth weight was 20 per cent compared with 7 per cent in non-anaemic pregnancies [10] and perinatal loss in the anaemic mothers was more than twice that in the case of non-anaemic ones.

Anaemia of pregnancy is common in many developing countries. Several prevalence studies indicate that from 15 to 50 per cent of pregnant women may have low haemoglobins. Hence early diagnosis and treatment of maternal anaemia is essential for the survival and well-being of the newborn.

DIABETES

Diabetes in pregnancy has long been associated with increased perinatal mortality. Careful management of the pregnant diabetic should begin

with the first antenatal visit. The outcome of pregnancy in such mothers depends largely upon how effective the control of diabetes has been from conception, and where possible such mothers should be kept under skilled supervision and care in the hospital.

Many normal women show glucose intolerance during pregnancy which returns to normal after the birth of the baby. The term 'gestational diabetes' has been used to denote this condition. A higher than average perinatal mortality has been reported in such mothers, especially when they are above the age of 25, and when there is a history of previous unexplained abortions, stillbirths, neonatal deaths, polyhydramnios or very large babies.

MALARIA

In malarious areas, infections of the placenta with malaria parasites is common. Where such infection occurs, the weight of the newborn is lower than that of babies born to mothers in whom the placenta is not infected.[12]

Relapsing fever is a common cause of low birth weight and stillbirths in certain regions, such as the dry central region of Tanzania. The causative organism is know to pass the placental barrier and can be detected in the blood stream of the newborn.

Chapter 4

Cerebral Birth Injury

It has been said that although less than 10 per cent of babies are lost in the perinatal period, it is only one-tenth of the survivors who reach a university level of education. It is possible that birth injury may damage the brain only minimally so that babies are born well below their potential of intellectual attainment, though not obviously defective. Hence, when considering the hazards of the birth process, survival only should not be the aim, but 'intact survival' needs to be emphasised, which means survival with undiminished potential for intellectual growth and attainment.

Birth injury may be caused by one of three factors:

(1) Late pregnancy haemorrhage.
(2) Anoxia.
(3) Direct trauma.

Categories (2) and (3) overlap in the causative mechanisms, so clinical aspects and treatment are considered together.

In the British perinatal mortality survey[6] cerebral birth injury has been estimated to contribute to stillbirths at the rate of 0.3 per thousand births; its contribution to overall perinatal mortality is estimated at one per thousand live births. Various factors in the antenatal and the intrapartum periods were found to be associated with cerebral birth injury, such as prolonged labour, precipitate deliveries or breech presentation. Primipara need to be watched since about half the cases of deaths due to birth injury were amongst first-borns.

Breech delivery is associated with maximum risk. Even in the best circumstances, the risk factor in breech is almost seven times that in vertex presentation, three times that in forceps delivery, and twice that of caesarean section. Several authors have commented that the most common type of trauma in breech delivery is injury to muscle and soft tissue of the back and lower extremities. Such extensive muscle trauma may be the cause of death in the same way as in crush syndrome or in disseminated intravascular coagulation.

Next to delivery by breech, neonatal asphyxia is another important cause of death and brain injury in the newborn. Babies who suffer a delay in the onset of respiration by five minutes or more stand a high risk of such injury.

Several studies of foetal physiology during birth have indicated that even in normal labour brief episodes of foetal hypoxia are not uncommon. Such episodes occur during powerful contractions of the uterus followed by recovery during relaxation. More prolonged asphyxia occurs whenever there is interference with the maternal or foetal side of the circulation in the placenta. Thus, abnormal uterine activity, maternal hypotension, separation or infarction of the placenta or cord complications during birth can all cause foetal hypoxia.

A high incidence of foetal distress in labour has been recognised in several illnesses like diabetes, chronic hypertension, chronic renal disease, toxaemia of pregnancy, prolonged rupture of membranes, and in those mothers who have a poor obstetric history. Many of these conditions are known to compromise uterine blood flow in the mother; but it is not unusual for foetal distress and even death to occur in what appears to be a completely normal labour. In such cases, the foetal heart rate is usually a reliable sign of imminent trouble. If the rate persistently deviates from a range of 120 to 160 per minute with or without irregularity of rhythm, it is an indication of foetal distress. Many such babies show biochemical and clinical evidence of asphyxia at birth. In this respect, bradycardia is always a bad sign. For example, a threefold increase in perinatal mortality has been recorded in cases with foetal bradycardia persisting for more than half an hour prior to the birth of the infant.

There are three variables involved in the causation of cerebral birth injury:

Patient and the community (or tribe) she comes from:

(a) Beliefs and attitudes of the community towards pregnant women.
(b) Whether institutional delivery is accepted.
(c) Traditional medicines given to women in labour.
(d) General nutritional status of the community:
    (i) if malnutrition is common there may be stunting of growth.
    (ii) the type of pelvis.
    (iii) maternal depletion syndromes, if any.

The birth process – Abnormal presentations, multiple pregnancy and parity together with factors already mentioned like prolonged labour, precipitate delivery and breech presentation contribute to the incidence of cerebral birth injury.

Birth attendant – In countries where an estimated 15 per cent to 20 per cent births occur in institutions, most deliveries in rural areas are conducted by traditional midwives. If to this factor are added lack of antenatal supervision, chronic ill-health and inadequate nutrition, it is not surprising that there is a high incidence of cerebral birth injury in such communities.

Amongst newborns with a clinical diagnosis of cerebral irritation, the majority recover, so that the exact pathological changes underlying the clinical picture are not known. In most cases the basis of the 'injury' is anoxia and cerebral oedema.

In the management of cerebral birth injury it is important to remember that anoxia co-exists and the effect of one potentiates the other. Therefore, oxygen is the prime necessity of all newborns in whom cerebral injury is suspected. Inj. vitamin K. 2.5 mg should be administered soon after birth because trauma and anoxia have occurred in an individual with a potential bleeding tendency. Early feeding with 5 per cent glucose, given by naso-gastric tube if necessary, may be an advantage. As soon as mother's milk is available, expressed breast milk is substituted for glucose solution. Sedation is useful and helps to conserve body strength by preventing excessive crying and restlessness. Chloral hydrate (15 mg to start and 30 mg two to three times daily) is useful for the purpose.

In the early diagnosis of cerebral birth injury a high degree of suspicion is essential, especially where the above-mentioned factors of pregnancy and labour have been present. Besides this, poor Apgar scores one minute and five minutes after delivery, cyanosis at birth, failure to cry at birth, and failure to suckle are important diagnostic points. The baby may change colour intermittently, may show an anxious wakeful expression, vomit and be unable to suckle. The cry may be weak or very shrill and different from the cry of the normal baby. In some cases the fontanelle may appear full, there may be rapid eye or tongue movement or rigidity of limbs.

Babies who continue to show signs of cerebral irritation for more than three days after birth may be left with permanent damage. Similarly, a combination of persistence of fits, apnoeic attacks and abnormal neurologic symptoms carries a bad prognosis.

Two types of birth trauma need special mention.

(1) *Cephal-haematoma* – This could be large enough to produce exsanguination in some cases. Again when large cephal-haematomas are being absorbed jaundice may occur (see figure 4.1).

(2) *Subdural haemorrhage* – The history regarding the pregnancy and labour may not be very suggestive, but vomiting

which is persistent and failure to thrive should raise a suspicion of its presence. Rapid increase in the size of the head and the presence of retinal haemorrhages on fundoscopy will help to establish the diagnosis.

FIG. 4.1 A big cephal haematoma

Chapter 5

Prenatal Care

Good antenatal care is the unborn baby's best insurance for health. The first priority in perinatal medicine is to make such care widely available and to promote its acceptance, especially in rural areas. Both the number of antenatal visits and the quality of care provided are important. The ideal is to achieve one visit per month throughout pregnancy. If this target cannot be achieved, a minimum of five visits should be insisted upon, of which at least three should be in the last trimester. The quality of antenatal care is difficult to judge. Most antenatal clinics are conducted by auxiliaries with very little supervision. It has been suggested that the frequency with which blood pressure has been measured or haemoglobin has been estimated may be used as a criterion for judging the quality of care.

Unless the objectives are well defined they tend to get overlooked in a busy antenatal clinic. In general, the aim is to prepare the mother, both physically and psychologically, to give birth to a healthy baby and to be able to care for him. To this effect supervision of her health and nutrition are necessary in order to provide the optimum conditions for the growth of the foetus. All high-risk pregnancies should be identified early, so that an appropriate plan of management can be formulated well in advance. Regular health education classes should be held at the clinic or elsewhere in the community, to provide a basis for healthy living and to teach mothers how to care for and feed the young ones. These lessons can later be reinforced in the postpartum period or in the under-fives clinics.

NUTRITION IN PREGNANCY

A variety of physiological changes occur in pregnancy. These not only help to maintain homeostasis but also produce an environment suited for the adequate growth and development of the foetus. One of these changes is the alteration in maternal metabolism for building foetal tissues. In addition, the mother's body is also laying down energy stores to be utilised during lactation.

In a peasant society where large numbers exist on marginal nutrition, the state of nutrition of the expectant mother needs watching. The average weight gain during pregnancy in South India is 6.0 kg, compared with 11.7 kg in the U.K. and 17.0 kg in the U.S.A. In a group of 411 mothers studied in Tanzania the average weight gain was 9.1 kg and in a similar study in Uganda it was found to be 8.39 kg. In socially deprived groups and in times of food shortage it is not unusual to find that some mothers lose weight during pregnancy. Such mothers and those who weigh less than 40 kg after the 20th week of pregnancy tend to have babies with low birth weights and higher rates of perinatal mortality.

Studies in the physiology of the pregnant woman show that as pregnancy advances there is a growing need for protein and calories in the diet (see table 5.1).

TABLE 5.1 Mean daily increments of protein and fat[7]

	Week of pregnancy				
	0–10	*10–20*	*20–30*	*30–40*	*Cumulative total*
Protein (g)	0.63	2.5	4.64	5.36	910
Fat (g)	6.55	22.3	24.0	12.3	4,464

The average recommended requirements to meet the above needs are summarised in table 5.2.

TABLE 5.2 Recommended requirements for protein and fat

		Adult		*Pregnancy 6-9 months*	
	Body weight (kg)	*Protein (g)*	*Protein (g/kg)*	*Protein (g)*	*Protein (g/kg)*
F.A.O. (1957)	55	65	1.2	75	1.36
National Research Council U.S.A. (1958)	58	58	1.0	78	1.34
Nutrition Advisory Committee of India (1958)	45	45	1.0	100	2.22

In many rural communities the actual dietary intakes in pregnant women fall short of recommended allowances. It is customary for the pregnant woman to eat less so that the 'labour is easy'. Taboos exist against the eating of eggs and many flesh foods in several countries so that there is in fact a reduction in the intake of animal protein and calories during pregnancy; for example, dietary surveys in pregnant

women in South India, Tanzania and elsewhere have shown that only half the recommended allowances may be consumed.

As a result of inadequate nutrition the birth weights of babies born to mothers in the lower socio-economic groups are significantly low, with consequent increase in perinatal mortality. Supplementing the diets of pregnant women with calories and proteins results in heavier babies. Recent work in India has shown that supplementing the diets of pregnant women with 30-60 mg of iron and 200-500 mg of folic acid also leads to higher birth weights compared with control groups.

The adverse effects of nutritional deprivation in pregnancy on foetal growth have now been well documented in several studies. It is also true that when growth is arrested in experimental animals, that organ which is growing maximally at the time is affected the most. In the human, brain growth is maximal during the latter half of pregnancy and the first two years of life. It is therefore highly likely that prenatal malnutrition may predispose the baby to inadequate brain growth, particularly if postnatal malnutrition occurs as well.

Next to protein and calorie deficiency, anaemia is the most common nutritional disorder in pregnancy. Prevalence studies have shown that 15-50 per cent of women in Africa, 10-35 per cent of women in South America and more than 20 per cent of women in Asia have haemoglobins below 10 g per 100 ml.[8] A rapid succession of pregnancies may aggravate the pre-existing nutritional anaemia, resulting in maternal depletion. Table 5.3 gives the iron cost of a normal pregnancy.

TABLE 5.3 Loss of maternal iron in a normal pregnancy

Contributed to the foetus	200 mg – 372 mg
In the placenta and cord	34 mg – 170 mg
In blood lost during the delivery	100 mg – 250 mg
In milk (6 months' lactation)	100 mg – 180 mg
	434 mg – 972 mg
Iron conserved by 15 months' amenorrhoea	480 mg – 240 mg
Approximate net loss	0 mg – 700 mg

THE HIGH-RISK PREGNANCY

In such a pregnancy there are environmental factors causing hazards to the mother's health or to that of her infant. The foetus may not survive because of such health hazards, or he is likely to develop a physical, intellectual or personality disorder which may interfere with his growth and development. Such factors can be social, bio-medical, or both.

(1) *Social factors* – Perinatal risks are higher in mothers below

the age of 17 years or above 35, in first pregnancy and after the fifth, in lower socio-economic groups, in mothers whose heights are below 5 feet and in those with a previous history of abortion, stillbirth, neonatal death or complications of labour. Other factors like marital or family strife, polygamy and illegitimacy can also have a strong influence on the survival and health of the infant.

(2) *Medical factors* – These are (a) complication of pregnancy and (b) diease in the mother either before she became pregnant or during the course of pregnancy. The medical factors are discussed further in chapter 4.

NOXIOUS INFLUENCES

The placenta acts as an effective barrier against a wide variety of bacterial illnesses, but is not so efficient against several viral and chemical agents. Their effects on the foetus vary from being subtle and recognisable only after a prolonged period of observation to severe, as when they interfere with the development of organ systems and cause malformations. For example, cigarette smoking in pregnancy is known to increase late foetal and neonatal mortality by 28 per cent and reduces birth weight by up to 170 g. In the survivors, there are deficits of physical and mental growth. Thus, the children of mothers who smoke ten or more cigarettes a day have been found to be shorter and slower in learning at ages 7 and 11 years compared with matched controls.[9,10]

In animal experiments a number of factors have been shown to be associated with congenital malformations in the offspring. These include dietary lack or excess, physical and chemical injuries and infections. In humans, drugs and virus infections only have a proven association with congenital malformations.

Drugs

After the thalidomide disaster clinicians have been paying ever-increasing attention to the problem of drug administration in the pregnant woman. Several drugs have been implicated at one time or another, though unequivocal proof is not forthcoming in the case of many. Table 5.4 summarises available information on the effect of drugs on the foetus when administered to the mother during pregnancy.

Besides passing through the placental barrier and interfering with the development of the foetus, many drugs also influence placental function either by increasing or decreasing the blood supply or by interfering with placental biochemistry. Hence a cautious and conservative approach is necessary when prescribing medicines for a pregnant

woman. In this respect one should also take into consideration the effects of indigenous potions administered to women in labour. Some of them are soporific and, by depressing the respiration of the baby, cause difficulties with breathing at birth; others are oxytoxic and cause antepartum anoxia due to irregular contractions of the uterus.

TABLE 5.4 Possible effects of drugs on the foetus

Drug	*Defect produced*
Streptomycin	Neonatal deafness.
Tetracycline	Staining of deciduous teeth. (The drug combines with calcium and is deposited in tissues undergoing mineralisation, e.g. bones, teeth, etc.)
Sulphonamides	Coagulation disturbances in the newborn.
Phenobarbitone and Phenytoin	Neonatal haemorrhage.
Oral hypoglycaemic agents	Neonatal hypoglycaemia.
Reserpine	Stuffy nose, nasal discharge.
Quinine	In heavy doses can cause abortion. Deafness or thrombocytopaenia can also occur.
Antimitotics	Foetal death or malformations.
Thiazide diuretics	Thrombocytopaenia.
Progesterone, oestrogens or androgens	Masculinisation of the foetus
Pyridoxine	Pyridoxine-dependent fits.
Morphine	When given just before delivery, may cause respiratory disturbances.
Barbiturates and Phenytoin sodium	A small but significant risk of cleft lip or palate, diaphragmatic hernia and hypoplasia of distal phalanges.
Phenothiazines	Extra-pyramidal dysfunctions like tremor, increased muscle tone, etc.
Vitamin D	Infantile hypercalcaemia.

In addition to the above list of drugs contra-indicated during pregnancy, there are several which should be avoided near parturition because of possible effects on foetal physiology. These are the vitamin K analogues, long-acting sulpha drugs, nitrofurantoins, thiazide diuretics, anti-coagulants except heparin, the tetracyclines, chloramphenicol, streptomycin and hexamethonium.

Maternal infections

The association of maternal *rubella* with congenital abnormalities of the foetus is now well established. The estimates of risk for congenital defects are 50 per cent if the mother was infected in the first month of pregnancy, 30 per cent in the second month, 20 per cent in the third

month and 5 per cent in the fourth. Once foetal infection is established it persists throughout gestation and after birth and may continue to cause damage for several years. Moreover, an infected newborn is a carrier of the rubella virus and can be a source of infection in susceptibles who come into contact. Chronic viraemia is present up to 6 months after birth in babies who were infected in foetal life, and excretion of the virus has been shown to occur for the first 2-3 years of life.[11]

Many mothers suffer infections which are not clinically manifest. On the other hand, clinical symptoms or the rash may be dismissed as trivial and not reported. Hence antenatal diagnosis is rare in most developing countries except perhaps in the case of well-informed women in the upper social class. The most important stage in pathogenesis is maternal viraemia, which may occur up to seven days before the onset of the rash, so that by the time the rash has appeared the virus has already reached the placenta. Hence a high degree of awareness is necessary to arrive at the diagnosis in infected newborns. Babies with microcephaly, congenital heart defects, cataracts, retinopathy hepatosplenomegaly, hepatitis and thrombocytopaenia should be screened for possible rubella infection. Occassionally, an infected baby may not show the typical signs and symptoms of congenital rubella at birth. Deafness, vestibular dysfunction, seizures, delayed motor development and other such manifestations may not be evident until weeks, months or years after birth. Hence, if maternal rubella has been suspected during pregnancy, the baby should be checked at regular intervals. As a general rule, infants born to mothers with subclinical rubella carry a smaller risk of infection compared with those born to mothers with clinical disease.

Intrauterine infection with the *cytomegalovirus* (C.M.V.) is responsible for damage to the central nervous system. Symptoms vary from gross damage resulting in mental retardation to subtle dysfunction which is not evident until later childhood. The occurrence and severity of foetal infection depends upon the stage of pregnancy at which maternal infection is acquired. As in the case of rubella, most infected babies and especially those with severe disease are born to mothers with infections in the first half of pregnancy. In severe disease there is evidence of multiple organ involvement in addition to the central nervous system. Thus hepatosplenomegaly, jaundice, petechiae, microcephaly, cerebral calcification and chorio-retinitis all form part of the disease spectrum in the newborn.

Pregnant women with serological evidence of C.M.V. infection are known to excrete increasing amounts of the virus from the cervix as pregnancy advances, reaching rates of 11 to 28 per cent during the third trimester in some cases. Thus the new born infant may face a further risk of infection during birth.

Herpes simplex of the cervix, vagina or vulva in the mother is a potential source of infection in the newborn. Here, the baby most at risk is the one whose mother acquires the infection nearer term. In the absence of immunity in the baby, the virus spreads rapidly causing a destructive invasion of the tissues. Recovery from generalised neonatal herpes infection is rare and the survivors invariably suffer brain damage. Depending upon the tissues involved the clinical picture is one of meningo-encephalitis, pneumonia, liver and/or adrenal failure.

Toxoplasma gondii is a protozoa infecting the gut epithelium of the cat. Human infection occurs through close contact with cats or through eating raw or undercooked meat. It is estimated that at least one-third of the adult population contracts toxoplasmosis, largely asymptomatic, in most areas of the world. When infection is acquired *in utero,* severe consequences arise resulting in abortion, stillbirth or congenital defects like mental retardation and hydrocephalus. Less serious effects like uveitis and hepatosplenomegaly are commoner but may go unrecognised. Since congenital toxoplasmosis develops only if the mother acquired infection in pregnancy, the best method of prevention is to avoid contact with cats and eating undercooked meat.

Congenital syphilis is one of the most commonly diagnosed infections in the newborn in developing countries. The infected mother transmits the illness to the foetus through the placenta, and rarely through an abrasion of the skin or mucous membrane. About a quarter of the infected foetuses are aborted. Those who are born alive show various manifestations of the illness. The organs most commonly involved are the liver, skin and mucous membrane, bones and the central nervous system. The earliest symptom is 'snuffles' a term applied to a blocked nose due to secretions which are at first clear, later turning to sero-sanguineous and finally purulent. Skin rashes appear in the second week and are mainly around the mouth, the nose, and in the diaper region. Palms and soles may also be involved but usually they are diffusely red, thickened or wrinkled. Involvement of bone causes painful limbs which are moved very little, hence the term pseudo-paralysis. X-ray of the bones helps to make the diagnosis in most cases (figure 5.1). They show osteochondritis, periostitis, epiphyseal dislocation or a moth-eaten appearance. The diagnosis is confirmed by serology. In this respect it is important to remember that if the mother was treated in pregnancy her blood may still contain the reagin which causes false positive results in the baby. When in doubt serial tests should be done to observe the progressive rise or fall in titre. Penicillin by injection is the treatment of choice. A total dose of 50,000 units per kilogram of body weight should be administered over a period of one week.

Malarial infection of the placenta causes low birth weight and in endemic areas it is an important contributory factor to low birth weight.[1 2]

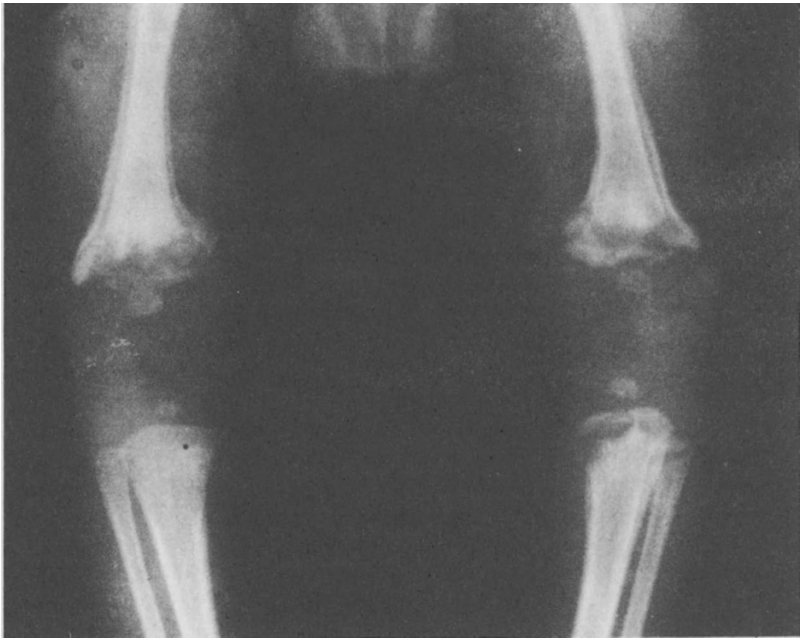


FIG. 5.1 Congenital syphilis.

The baby was brought to hospital for inability to move both lower limbs. Note the moth-eaten appearance of lower ends of both femurs and periostitis.

Infections of the placenta (chorio-amnionitis) are associated with premature labour and late foetal deaths. Many of these infections are due to ascending infections by bacteria in the lower genital tract or through the bloodstream of the mother.

As a result of various studies it is estimated that about 4.5 per cent of pregnant women have *bacteriuria.* Despite adequate therapy, birth weights of infants of such mothers are lower compared with those without any bacteriuria. If such infection is persistent or refractory to treatment the rate of foetal loss also tends to be high.

PROTECTION OF THE FOETUS

Since the aim of prenatal care is protection of the mother and the foetus, one ought to consider positive steps to achieve it. Immunisation of the mother against tetanus in the last trimester produces antibodies in her blood which reach the foetus in sufficient quantities to protect it from neonatal tetanus. In one study the incidence of neonatal tetanus was 0.57 per cent in the protected group as compared with 10.0 per

cent in the unprotected group.[13] In malarious areas routine chemoprophylaxis in pregnant women will not only reduce the incidence of pregnancy anaemia but also produce larger babies.[14] Similarly, early detection and treatment in all maternal illnesses, especially venereal disease, will help to reduce low birth weight and foetal wastage. This concept of positive action early in pregnancy so as to avoid illness in the baby has gone yet a step further in the treatment of Rh-negative mothers with anti-D globulin soon after delivery, to prevent sensitisation and thereby to protect her future children.[15]

Chapter 6

Routine Care of the Newborn

The time of birth is one of transition from intrauterine life to an independent existence and calls for many adjustments in the physiology of the baby. In foetal life, the placenta acts as an organ of gaseous exchange, as a source of nutrients and elimination of waste products, and also as an edocrine organ. Immediately after birth, the baby's own physiological systems must take over these functions for survival. The baby's cry at birth helps to expand his lungs and triggers normal respiration. At the same time pulmonary blood vessels open up, blood surges into the lungs, and the process of oxygenation of blood commences. Physiological shunts like the foramen ovale and the ductus arteriosus, which served the purpose of by-passing the pulmonary vasculature in foetal life, now begin to shut down and the circulation changes to the more adult type. In the same way, the baby's gastrointestinal tract begins to function in readiness for taking over the digestion and absorption of nutrients, and the kidneys assume their role of excretion of waste material.

Of these various changes, the expansion of the lungs at birth and the onset of regular respiration are crucial, since the changes in the baby's circulation are dependent on them. Moreover, the brain is dependent on a constant supply of oxygen and glucose and a drop in the blood levels of any of these can result in depression or permanent damage of brain function.

Intrapartum anoxia as a result of adverse factors operating on the foetus during pregnancy or at the time of birth is a recognised cause of death in the first 48 hours of life. The size and development of the infant at birth, his metabolic reserve, the type of delivery and the infant's capacity for adjustment to extrauterine life are all significant factors in his survival.

A large majority of newborns cry soon after delivery, are vigorous and suffer no complication; five to ten per cent may have difficulty with the establishment of respiration, and a few of these may need prolonged resuscitative measures. Various methods of assessing the vitality

of the newborn have been employed; the most commonly used is the Apgar score taken at birth and again after five minutes. In this method a score of 0 to 2 is given for each of five objective signs (see table 6.1). A score of 10 indicates an infant in the best possible condition. About 90 per cent of babies score between 7 and 10; a score below 5 needs prompt action.[20]

TABLE 6.1 Apgar scores

Sign	*0*	*1*	*2*
Heart rate	Absent	Below 100/min	Over 100/min
Respiratory effort	Absent	Slow, regular	Good cry
Muscle tone	Limp	Some flexion of extremities	Active motion
Reflex irritability	No response	Some motion	Cry
Colour	Blue or pale	Body pink, extremities blue	Completely pink

A simpler method of assessment takes into consideration only the time taken to establish respiration and accordingly the newborn is put into one of the following categories:

(1) Class A – cries at birth.
(2) Class B – delay in onset of respiration of two minutes.
(3) Class C – delay of 5 minutes.
(4) Class D – delay of 10 minutes or more.

CARE AT BIRTH

On leaving the intrauterine environment, the baby's basic needs are to establish respiration and subsequently adequate nutrition, to maintain normal body temperature, and to avoid contact with infection. The object of care at birth is to ensure that these needs are met and to assist the baby in making adjustments to extrauterine life. Many of the diseases of the neonatal period arise from failure of physiological adjustments, presence of congenital malformations, results of maternal illnesses and infections, effects of physical or biochemical injury in the perinatal period and from sepsis. Successful management depends upon creating an environment to which the baby can adjust with the least amount of stress, and upon providing protection from environmental hazards.

Immediately after delivery, the infant should be suspended head downwards to drain the fluid and blood from his mouth, nose and oropharynx. The mouth should be gently swabbed to remove excess mucus and the oropharynx cleared with a mucus extractor. The eyes

should be wiped with clean cottonwool, and the first (1 minute) Apgar score is taken.

There should be no hurry to cut the cord. As far as possible, one should wait until the cord has stopped pulsating; it should then be milked towards the baby. This manoeuvre gives the baby about 100 cc more blood and the iron contained in it will help to insure against anaemia later in infancy. It is also claimed that delayed clamping helps lung expansion by providing extra volume to fill up the lung capillaries, and that it reduces the incidence of respiratory distress syndrome in susceptible babies. Before clamping, the baby should be held at or below the level of the placenta, since reversal of flow has been demonstrated in the case of newborns held above the level of the placenta.

The cord is cut about 3 finger-breadths from the umbilicus and tied by means of a tape in two places with a reef knot or surgeon's knot. A slipped ligature is often a cause of severe haemorrhage from the cord. The cord stump is sprayed with nobecutane spray or painted with triple dye and left undressed.

The number of vessels in the umbilical cord should be noted. Congenital malformations are associated with the presence of a single umbilical artery. In one study it was estimated that 11 per cent of such babies die in their perinatal period, and 18 per cent of the survivors suffer a malformation. Amongst those showing no obvious anomalies, one in three harbour a lethal but unrecognised abnormality. There has been no demonstrable organ specificity amongst the various malformations described; any organ system may be affected.

A quick check for obvious congenital malformations should be made and a second (5 minute) Apgar score is taken before placing the baby in a pre-warmed cot to rest.

FIRST FEED

As soon as the baby has recovered from the effects of birth he can be put to the breast; most newborns will be ready for a feed in 2 to 6 hours. If the baby appears to be ill – if he is pale or blue in colour, or limp, or vomits excessively – initial feeding by naso-gastric tube may be necessary whilst the baby is kept under observation. Later, he may be put to the breast if he has recovered; if he is still not capable of sucking, feeds should be given by means of a naso-gastric tube.

MAINTENANCE OF BODY HEAT

The body surface area in the baby is three times that of the adult per unit of body weight. On the other hand, the layer of subcutaneous fat

in the baby is thinner compared to the adult, so the newborn loses heat at a rate four times that of an adult and can easily get chilled. At birth, the baby's body temperature is the same as the intrauterine temperature of the mother. But because he is wet and exposed, his temperature drops rapidly. In the normal infant, drop in body temperature is accompanied by increase in metabolic activity and oxygen consumption in order to produce more heat until an equilibrium point is reached where heat production is equal to heat loss. Rise of environmental temperature has a reverse effect. The more mature the baby the better is his response to environmental temperature change.

Dressing the baby improves resistance to heat loss. A vest, napkin and a long nightdress will reduce heat loss to about half of that in the naked state. If in addition the baby is also wrapped in a flannel sheet and covered with two layers of cotton blanket, heat loss will be reduced to a third. In such a 'cot-nursed' baby, there will be fewer fluctuations due to changes in the room temperature.

A draught-free environment of 24^0C (75^0F) is adequate for most cot-nursed babies with normal birth weights. Lighter babies need a higher environmental temperature. For example, babies weighing less than 1.5 kg need to be cot-nursed in an environmental temperature of 29^0C (85^0F).

In the tropics, where environmental temperatures tend to be high, incubators are not essential for the care of the smaller babies except in those rare instances where a baby has to be kept naked for observation. Even then it should be remembered that minor fluctuations in the temperature of the incubator can affect the body temperature of the naked baby. For example, variations of 2^0C in the incubator temperature can affect the body temperature of the naked baby, whereas room temperature has to fall to 19^0C or rise to 31^0C to affect a clothed baby under blankets in a cot. In the majority of instances, the small baby can be adequately maintained in a neutral body temperature by cot-nursing in a room with radiant sources of heat placed around the room. Occasionally, a source of warmth may need to be improvised under the mattress for some babies (see figure 6.1).

An environment that is too warm can be dangerous. Hence in a heated nursery or where an external source of warmth is being provided, the baby's temperature should be monitored at regular intervals of 3-4 hours.

THE NURSERY

All healthy newborns should leave the labour wards together with the mother. Early baby-mother contact is crucial in establishing close emotional bonds and also contributes to trouble-free lactation in the

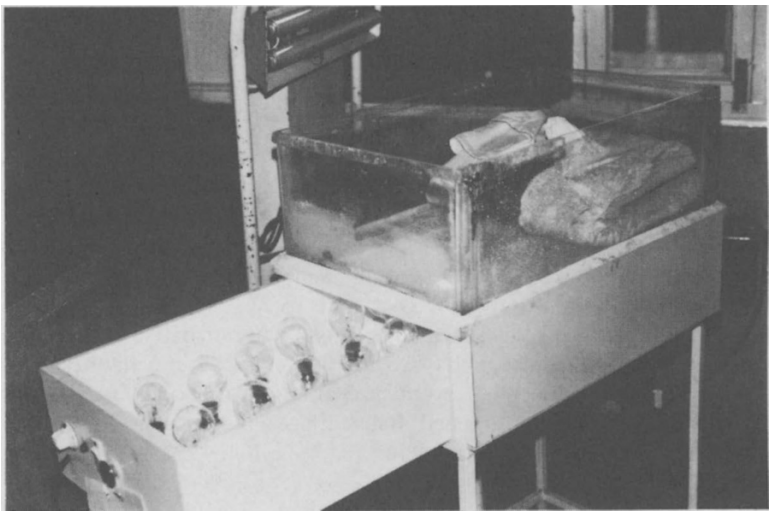


FIG. 6.1 A simple incubator developed by the Paediatric Department of the Postgraduate Institute in Chandigarh.

When the electric bulbs are switched on, the warm air rises through the mattress keeping the baby warm.

mother. Animal studies have indicated that there is a critical period of mother-baby contact for establishing close emotional relationships and any delay beyond such a critical period can lead to rejection or even aggression.

In the sick or the high-risk infant, prolonged observation in the nursery or special care unit may be unavoidable. In such cases opportunities should be created for frequent contacts between the mother and her baby and also for the mother to participate in the nursing and care of her baby.

The following groups of babies may be selected for special care or observation:

(1) Low birth weight babies.
(2) Abnormal presentations, e.g. breech or face presentations.
(3) Instrumental deliveries, e.g. vacuum or forceps, or caesarean sections.
(4) Illness in the mother, e.g. pre-eclamptic toxaemia, diabetes or mental confusion.
(5) Infections in the mother, e.g. tuberculosis or fever.
(6) Mothers who had no antenatal care (unbooked cases) and babies born before arrival in hospital (B.B.A.).
(7) Mothers with postpartum complications.

(8) Babies with signs and symptoms of disease in the early neonatal period.

These babies are likely to suffer from haemorrhagic disease of the newborn either because of an increased tendency to bleed (e.g. low birth weight) or because of a more traumatic delivery and anoxia, e.g. breech deliveries. They should be given Inj. vitamin K 1 mg at birth.

Breast feeding should be insisted upon for all babies admitted to the special care unit in order to avoid gastrointestinal infections. If a mother has no milk secretion, pooled expressed breast milk should be utilised for her baby.

The other danger to guard against in a nursery population is pyogenic infection. Umbilical and skin sepsis or eye infections are only too common. Many of these may be traced to an infected bath tub. The ritual of a daily bath is often time-consuming and potentially dangerous and is better avoided. Instead, at the time of admission to the nursery, each baby should be sponged with Savlon (1 in 200) solution and a daily routine of 'top-and-tail' with the same may be instituted. Other common sources of infection are the attendants in the nursery, the linen, blankets and other clothing with which the baby is in daily contact, and other infants (see also p. 86). A communal changing or treatment table is dangerous, and each baby should be attended to in his own cot.

A complete physical examination of the newborn admitted to the nursery should be carried out as soon as possible and within twenty-four hours of admission. All data regarding mother's age, parity, previous obstetric history and pregnancy should be recorded, together with relevant obstetric data. For the newborn this information serves the same purpose as a history of past illness does for the older child. The object of the physical examination is threefold:

(1) Has the mother given birth to a healthy baby free of any congenital abnormalities? Some abnormalities are gross and obvious on observation, others like congenital heart defect or anomalies of the gastrointestinal tract will need detailed physical examination and x-ray studies, whereas others such as hypertrophic pyloric stenosis may not be detected until the baby is a little older and develops further symptoms.

(2) Have the rigours of delivery incapacitated the baby in any way? There may be excessive moulding (figure 6.2), or a fracture, or soft tissue injury of varying degrees. On the other hand the symptoms may be subtle, like vomiting or failure to thrive, as in cases of subdural haematoma.

(3) Is there any other disease process peculiar to the newborn period? For example, atelectasis of the lung, hypogly-

FIG. 6.2 Excessive moulding

caemia, jaundice and effects of drugs administered to the mother during labour.

During the period of observation in the special care unit the following deviations from normal should be taken as warning signals:

(1) Not sucking well.
Vomiting.
Excessive drooling.
Weight loss.
Abdominal distension.
Diarrhoea.

(2) Convulsions.
Twitching.
Shrill cry.
Weak cry.
Lethargy.
Rigidity or hypotonia.
Moro reflex poor or absent or abnormal.

(3) Bleeding.
Petechiae.
Echymoses.
Heart rate below 100/min or over 140/min.
(4) Respiratory rate over 50/min.
Irregular respiration.
Expiratory grunt.
Retraction of chest or chin tug during breathing.
(5) Cyanosis.
Pallor.
Jaundice.
(6) Oedema.
Sclerema.
(7) Umbilical or skin infection.
Conjunctivitis.
Skin rash.

When the symptoms or signs are confined to one group it is likely that one system is involved; on the other hand, when they are present from several different groups, a generalised involvement or progressive deterioration should be considered.

During her stay in hospital the mother should be taught how to care for and handle her baby. As much guidance as possible in the art of mothercraft and simple hygiene should be given to the mother; she should also be told where to take her baby in case of any difficulty. Many mothers leave the maternity hospital not knowing what services and facilities exist for the newly-born baby, nor having received any instructions about home care. Medical facilities may be far from home, so that when any symptoms develop in the infant at home, mothers get bewildered and resort to folklore and traditional methods of treatment.

Chapter 7

The Normal Newborn

The 'normal' newborn has been described as a product of forty weeks' gestation, born to a young healthy mother after a spontaneous vertex delivery. The age of the mother, parity, birth order, maternal health during pregnancy, quality of antenatal care, type of delivery, instrumentation and drug administration during labour are some of the factors which determine the physical well-being and ability for extra-uterine adjustment in the newborn. In practice it has been found that the size of the newborn has an important bearing on his potential for growth and development and, by usage, has become the common criterion for classification of normal newborns. The gestational age is a reliable yard-stick for judging maturity especially when considered together with the birth weight; however, in most developing societies the length of the gestational period is difficult to assess with any accuracy and so in most cases the weight at birth may be the only available parameter for classification besides clinical observations.

The weight of the newborn varies from one locality to another depending upon a combination of genetic and environmental influences; in any one locality it will also be influenced by the social class of the family. The international classification of prematurity based on a birth weight of 2,500 g or less is not universally applicable and in many developing countries a lower limit will be more appropriate.[21]

Other useful anthropometric standards are those of body length and head circumference which are tabulated below for each weight group (see table 7.1):

In the first few days after birth most babies lose weight. This is due to loss of water by evaporation from the skin and by excretion in the urine. The weight loss may be as much as 10 to 15 per cent of birth weight especially in prematures who were born with oedema. By the fourth day of life weight loss is minimal and the baby may begin to gain in weight so that between the seventh and the tenth day the birth weight is regained. After this period, a healthy baby will gain weight at the rate of about 200 g per week.

TABLE 7.1 Standards of body length and head circumference for birth weight

Birth weight (g)	*Body length (cm)*	*Head circumference (cm)*
900	36	27
1000	40	28
1250	40.5	29
1500	41	29.5
1750	42	30
2000	42.5	31
2250	43	32
2500	45	32.5
2750	46	33
3000	47.5	34
3250	48	34.5
3500	49.5	35

If the initial loss of weight is excessive, or if the baby fails to gain weight adequately after the first week of life, a careful inquiry and examination are necessary to elicit the cause. In a breast-fed baby it may be difficult to estimate the amount of milk taken during a feed. The mother's breasts should be examined for adequate lactation and for defects like retracted or cracked nipples; she should be observed during feeding to ascertain that the baby takes enough. In case of doubt, test-feeding may help to indicate the approximate amount taken. Several such readings should be taken at different times of the day and their average will show the approximate amount of milk taken during a feed. The baby whose hunger has not been satisfied will continue to cry after a feed, will make searching movements with the mouth after the feed and will have hard, constipated stools.

Vomiting and diarrhoea may also be the cause for failure to gain weight; in breast-fed babies these complications are rare and, when they do occur, they are either due to a metabolic abnormality such as lactase deficiency or to faulty feeding technique and only occasionally due to gastrointestinal infections.

Urinary infections and sepsis in the newborn may cause vomiting and loss of weight; if no obvious cause for failure to thrive can be detected subdural haematoma as a likely cause should be considered.

The dysmature baby

Low birth weight may be due to shortened gestation. It is estimated that about a third of all babies classified as premature are born after a pregnancy lasting 38 weeks or less; in others the gestation period will be found to be normal and the low birth weight may be due to some form

of intrauterine growth retardation. Such babies are grouped together as 'small for dates'. A large majority cannot be easily distinguished from their premature counterparts; they are stunted both in length and in weight and may not show any clinical signs of undernourishment except for a tendency for hypoglycaemia. Some babies in this category, however, have a characteristic appearance. They are thin with an absence of subcutaneous fat; the skin is wrinkled and dry and may be peeling; there may be yellow staining on the vernix; the nails are long and the baby has an alert expression. To such babies the term 'dysmature' or 'post-mature' is occasionally given implying thereby that the intrauterine growth has outstripped placental function.[22]

PHYSICAL APPEARANCE OF THE HEALTHY NEWBORN

The Head

The head may undergo a variable degree of moulding during delivery and may assume a characteristic shape depending upon the presentation and the degree of flexion. There may also be overriding of one cranial bone over another. Both moulding and overriding indicate the stresses and pressure to which the cranium and its contents are subjected during labour. Excessive moulding may be associated with intracranial haemorrhage (see figure 6.1).

In the newborn the bones of the cranial vault are separated from each other by fibrous septa called sutures, and these give place to soft areas called fontanelles at the corners of the bones. From the clinical point of view the anterior fontanelle is the most important and remains open until the age of 18 months.

The hair on the scalp often falls off during the first few weeks; the resulting baldness may cause parental anxiety.

Eyes

At birth the eyes may have a lighter colour which changes later.

The newborn is able to differentiate between light and darkness. The pupillary light reflex is present indicating that the peripheral visual apparatus is functioning. Adequate neuromuscular co-ordination of the muscles of the eyeball is not achieved until several months after birth giving an impression of a squint; this is often a cause of anxiety in the parents and needs reassurance.

Mouth

Most mothers are worried about 'tongue-tie' (a short frenulum); be-

cause of this common fear, cutting the frenulum of the baby is a common practice in traditional medicine. The frenulum may *appear* to be short and give an impression of restricted tongue movements in the baby; true tongue-tie is very rare and in most cases an emphatic answer in the negative is necessary to reassure parents.

In the rare case of a baby being born with a tooth or two already present in the mouth, careful handling of the parents and repeated reassurances are necessary. Any deviation from the normal, however trivial to the sophisticated, is viewed with superstition in many rural communities.

Trunk

The abdomen may appear to be distended after a feed; this is normal and should be differentiated from the gross distension of Hirschsprung's disease or distension with bowel patterns in intestinal obstruction.

The cord begins to dry within twenty-four hours and will separate after five to ten days. It should be allowed to fall off spontaneously and not be pulled off lest haemorrhage occurs.

Development and size of the breast nodule has been used as a criterion for judging maturity by some workers.[23] It is not palpable before a gestational age of 33 weeks; by 36 weeks it is 3 mm in diameter when measured with a caliper; after 38 weeks it averages 5 mm to 7 mm.

Skin

At birth the skin is covered with a cheesy substance called vernix caseosa. It serves a protective function and no attempt should be made to wash it off when blood clots are being removed from the body surface after birth. Staining of the vernix, either by meconium or a yellow colour, is abnormal; except in breech presentations such staining indicates foetal distress, placental insufficiency or haemolytic disease of the newborn.

Desquamation of the skin may occur in the first few days after birth; it may be extensive in some babies and the skin may peel in large sheets. At this time, staphylococcal infections of the skin may take place, the organism gaining entry through minor abrasions and raw surfaces.

At birth the skin may not contain enough melanin, and many babies will appear fair-skinned. Gradual development of melanin normally occurs over a few weeks and the skin darkens.

Subcutaneous fat necrosis may be seen as circumscribed indurated lesions over the back, extremities and the hips. The overlying skin is red and oedematous. Spontaneous regression is the rule though liquefaction and drainage externally may occur in some.

BEHAVIOUR PATTERNS

Activity and sleep

In the first few weeks of life there are long periods of sleep, up to 3 hours, alternating with periods of wakefulness partly occupied by feeding. Neither sleep nor wakefulness is a uniform state.[24] In the early stage of sleep the baby is easily roused and may show small frequent movements of various parts of the body. After a variable period in this stage the baby passes into a stage of deeper sleep from which he cannot be easily roused.

During the period of wakefulness gross body movements occur. These are inco-ordinate and consist of alternate flexion and extension occurring reciprocally on the two sides. These movements are more pronounced in the supine position and less when the baby is being carried in the arms or in the prone position.

Feeding

The average baby takes about 90-100 g milk at every feed and will require to be fed 6 to 8 times per day depending upon the amount taken. The normal requirements for fluids and calories are about 75 ml and 50 calories per 500 g body weight respectively in twenty-four hours.

The act of feeding may set up a gastro-colic reflex and bowel movements occur during a feed. Also air is swallowed with milk during suckling and periodically the feed should be interrupted to 'break wind' by carrying the baby on the shoulder. Failure to do so will produce distress due to distension and will interfere with feeding.

In all newborns, the cardiac sphincter does not function efficiently and after every feed the stomach contents may be regurgitated. The quantity brought up varies from one baby to another and when excessive, it may become necessary to hold the baby in the erect position for long periods after each feed.

PERFORMANCE

A variety of reflexes are described in connection with the normal newborn such as Moro's reflex, foot withdrawal reflex (crossed extensor), grasp reflex, supracilliary tap reflex, etc. (see table 7.2). Most of these responses are mediated by the spinal cord or the lower centres of the brain; for example they can be demonstrated in the anencephalic infant where cerebral hemispheric function is lacking. In the normal baby, as cerebral function develops, voluntary control of movements and posture is acquired and these reflexes disappear at predictable times.

TABLE 7.2 Neonatal reflexes

Reflex	*Elicited by*	*Description*	*Appearance (age of gestation)*	*Remarks*
Moro reflex	Any loud noise or vibrations By flexing the head on the trunk and releasing suddenly	Both arms are thrown out and then adducted in an arc of embrace	28 to 30 weeks	Unilateral in Erb's palsy and in fractures of clavicle and humerus
Grasp reflex	Pressing firmly on the open palm or sole of foot	Flexion of the fingers or toes	32 weeks	A hand kept permanently 'fisted' is an early sign of cortical motor defect on that side
Supraciliary tap	Sudden tapping with the finger on the supraciliary region	A homolateral or bilateral blink	32 weeks	—
Crossed extensor	Stimulation of the sole of the foot in one leg held firmly in extension at the knee	The free leg shows flexion (withdrawal), adduction and extension	37 to 40 weeks	—

Abnormal persistence of the neonatal reflexes gives an early clue to cerebral motor defect.

In the low birth weight baby the presence of these reflexes can help judge the approximate gestational age. Thus, the sucking reflex is present from 24 weeks' gestation onwards and is strong at 32 weeks; the Moro reflex is complete by 28 weeks and the supraciliary reflex is constant at 32 weeks. Hence, if the Moro and supraciliary reflexes are both present in a baby of low birth weight, the baby may be able to suckle at the breast; if only the Moro reflex is present the baby will have to be carefully supervised during feeds, and if the Moro reflex cannot be elicited feeding by naso-gastric tube will be the only safe way.

No two babies behave alike, and in the description in table 7.2 of physical appearance and behaviour patterns, minor deviations may be noted in an individual baby. These may be within normal limits; on the other hand if persistent or if they cause maternal anxiety, such deviations should be observed carefully in case they may be an early indica-

These subtle differences from normal should be differentiated from gross symptoms which demand urgent attention. These danger signals include:

(1) Cyanosis.
(2) Pallor.
(3) Bleeding, malaena, echymoses.
(4) Jaundice.
(5) Convulsions.
(6) No meconium for 24 hours.
(7) No urine for 24 hours.
(8) Apnoeic spells and respiratory difficulties.
(9) Vomiting (especially bile-stained).
(10) Excessive drooling.
(11) Inability to feed.
(12) Sclerema and oedema.

Chapter 8

Acute Emergencies

Life-threatening situations may arise at different periods in neonatal life. Soon after birth an emergency may occur due to failure of establishment or maintenance of respiration; in the first few days of neonatal life emergencies are commonly due to congenital defects incompatible with extrauterine existence or due to low birth weight; emergencies in late neonatal life are either of metabolic origin or secondary to birth trauma or due to infections. In the newborn, prompt treatment of any disease process is important because deterioration can be very rapid and therefore alertness in noting any deviation from normal is necessary. A close watch should be kept for the appearance of the danger signals mentioned in the previous chapter; when they appear and are being investigated or treated, any increase in the intensity or the appearance of additional symptoms should be noted.

Failure to cry at birth is a common emergency and needs prompt resuscitative measures. In most cases it will be the midwife who has to resuscitate the baby and she should be conversant with the procedure.

RESUSCITATION OF THE NEWBORN

Certain obstetric situations are commonly associated with asphyxia of the newborn and therefore when they are present difficulties should be anticipated and a resuscitation tray should be at hand. These conditions are:

(1) Maternal factors such as:
   (a) Age over 35 years.
   (b) Grand multiparity.
   (c) Disease like diabetes or toxaemia of pregnancy.

(2) Obstetric factors such as:
   (a) Prolonged second stage of labour.
   (b) Prolonged rupture of membranes.
   (c) Prolapse of the cord or cord entanglements.
   (d) Difficulty with delivery of shoulders.

(3) Foetal distress as manifested by:
  (a) Meconium-stained liquor.
  (b) Tachycardia of more than 160 per minute or bradycardia of less than 100 per minute.
(4) Drugs or anaesthetics given to mother.

The basic principles of resuscitation are as follows:

(1) Establish and maintain an airway.
(2) Ensure oxygenation.
(3) Secure adequate ventilation.
(4) Correct acidosis.
(5) Support circulation.

Establishment of the airway

All mucus in the mouth should be cleared and the oropharynx should be sucked out with a mucus catheter. Positioning of the baby with the head low may help in the drainage of secretions.

In the collapsed patient the tongue tends to fall back and block the air passage; to prevent this an airway should be inserted.

Oxygen administration

Even with a feeble gasping respiration, the newborn can take in more oxygen than during vigorous artificial respirations. Therefore as soon as an adequate airway has been established, oxygen should be administered either by a funnel (flow rate four litres per minute) or by a nasal catheter (flow rate one litre per minute).

Oxygen is a life-saving agent and yet when administered in high concentration and pressure over a prolonged period it can give rise to undesirable side-effects. Retrolental fibroplasia is a well-known complication of high arterial oxygen tension in the newborn. Recently several lung complications have been described which occur as a direct result of 'lung burn' and are independent of arterial oxygen tension. Hence in babies who need intratracheal oxygen for prolonged periods the possibility of pulmonary toxicity and massive pulmonary haemorrhage should be borne in mind.

Stimulation of the respiratory centre by Nikethamide or other analeptics is not necessary and may even be dangerous.

Nalorphine, a narcotic antagonist, is only of use if respiratory depression is known to be due to morphine or its analogues given to the mother. Its indiscriminate use in all cases of asphyxia is dangerous because it may cause depression of the respiratory centre (*Dose*: 0.2 mg/kg intramuscularly.)

Adequate ventilation

Intermittent positive pressure by mouth-to-mouth breathing is an effective way of ventilating the lungs of a baby who has apnoea. Endotracheal intubation and breathing on the tube is much more effective but needs skill and should be resorted to if possible.

For mouth-to-mouth breathing, the baby's head is extended and the operator covers the baby's mouth and nose with his own mouth. Gentle pressure is applied on the larynx with one finger to push it gently back towards the spine to occlude the oesophagus. The operator uses only cheek muscles to blow air into the baby's lungs, at the rate of 12 to 16 breaths per minute, and in between allows sufficient pause for the baby's lungs to recoil spontaneously.

Correction of acidosis

During periods of asphyxia there is rapid accumulation of acid metabolites in the baby's tissues and a fall in blood pH. Hence all babies who have suffered from asphyxia and have required vigorous resuscitation should be given alkali in the form of intravenous injection of sodium bicarbonate 3 ml/kg of an 8.4 per cent solution.

Support of circulation

If there is no heart beat, external cardiac massage with rhythmic compression of the heart between the sternum and the vertebral spine at the back will assist the circulation of blood and carriage of oxygen to the tissues. (*Rate*: 80-100 per minute.)

The baby should be lying on a firm surface (figure 8.1). The operator places two fingers on the baby's sternum and presses the upper sternum towards the spine at the rate of one per second. There is always a danger of rupturing the liver if the pressure is too far down – for example, when the fingers are on the baby's xiphisternum.

During all these emergency procedures, the baby remains exposed and will suffer hypothermia. Hence all precautions should be taken to provide warmth and to maintain his body temperature.

When dealing with respiratory difficulties in the newborn it is important to remember that:

(1) Infants who have initially shown difficulty with establishing respiration will invariably show difficulty in maintaining respiration. Hence those who needed resuscitation at birth should be observed over a minimum period of twenty-four hours for cyanotic spells, apnoea, irregular respirations or tachypnoea.

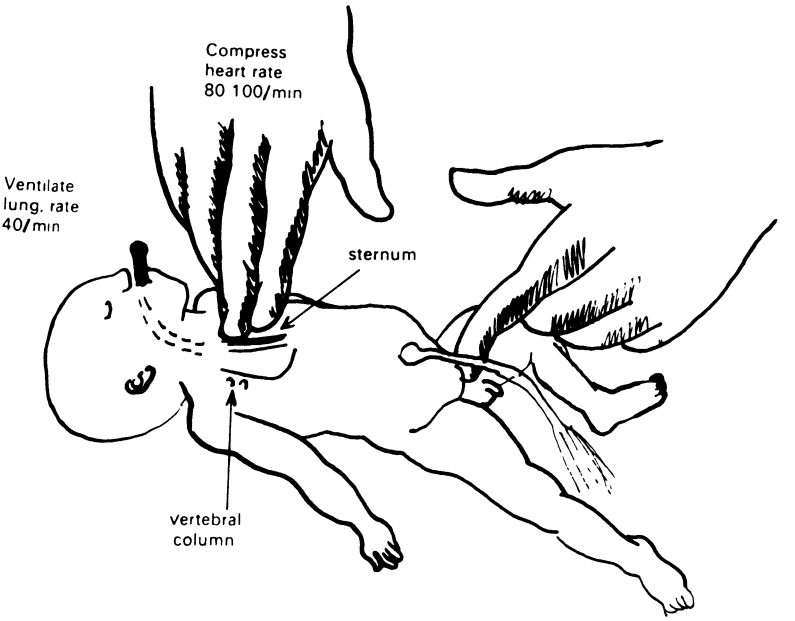


Fig. 8.1 Resuscitation of the newborn

In babies who have required vigorous resuscitative measures, lung complications like pneumothorax or tension cysts are common and should be excluded by clinical and radiological studies whenever further respiratory difficulties arise.

(2) Respiratory difficulties in the low birth weight baby are never benign and an early chest x-ray should be taken to help with a proper diagnosis.

(3) In the first hour of life, mild tachypnoea may occur; but if a baby of low birth weight shows a respiratory rate of over 50 per minute in the second hour of life, he should be watched for the development of the respiratory distress syndrome.

(4) The normal liquor amnii is harmless but if meconium has been passed *in utero* as in cases of foetal distress it may be inhaled during the gasping movements of the baby and causes severe chemical irritation of the tracheo-bronchial tree.

It has been noted that in the case of meconium-stained infants, more than half will have meconium in the trachea.

The viscid meconium blocks the airways and can cause patches of lung collapse or emphysema. In such babies the nasopharynx should be aspirated; next the vocal cords should be visualised and, if possible, the larynx and trachea should be aspirated as well.

SURGICAL EMERGENCIES

The average incidence of neonatal surgical emergencies is 2 per 1,000 live births. They fall into four main groups:

(1) Anomalies of the gastrointestinal tract resulting in intestinal obstruction.
(2) Respiratory emergencies.
(3) Developmental defects.
(4) Surgical aspects of neonatal sepsis.

Intestinal obstruction is one of the commonest conditions requiring surgical intervention in the newborn period. The final outcome in these babies depends a great deal upon the rapidity of the diagnosis and corrective surgery before complications can set in.

Certain warning signs should alert the obstetrician so that the baby can be put under observation:

(1) Polyhydramnios in pregnancy – it has been reported that in about 75 per cent of such babies there is obstruction of the upper small intestine.
(2) Presence of only two vessels in the umbilical cord instead of the usual three may be associated with major abnormalities.

Intestinal obstruction in the newborn presents three classic symptoms – vomiting, abdominal distension and failure to pass meconium within twenty-four hours after birth. Vomiting is the most reliable sign of obstruction. It usually starts on the first day and gets progressively worse. In a large proportion of cases, the vomitus contains bile as it is rare for the obstruction to be above the ampulla of Vater. Sometimes with high intestinal obstruction the baby may pass 'stools' which can vary from normal meconium to being mucoid or grey-green in colour.

In all babies displaying the above triad of symptoms, a stomach tube should be passed and gastric aspiration should be attempted. This procedure will also help to exclude oesophegeal atresia. If at the first emptying more than 20 ml of fluid is obtained, and especially if it is bile-stained, obstruction should be suspected and further studies undertaken as follows:

(1) Repeated physical examination for intestinal patterns on the abdominal wall, or distension of abdomen.

(2) Digital examination of the rectum (e.g. empty rectum in Hirschsprung's disease).
(3) Examination of the contents of the rectum.
(4) X-ray studies – plain views of the abdomen in the erect position, postero-anterior and lateral; also a plain view of the abdomen in the supine position (figures 8.2, 8.3, 8.4, 8.5, 8.6, 8.7).

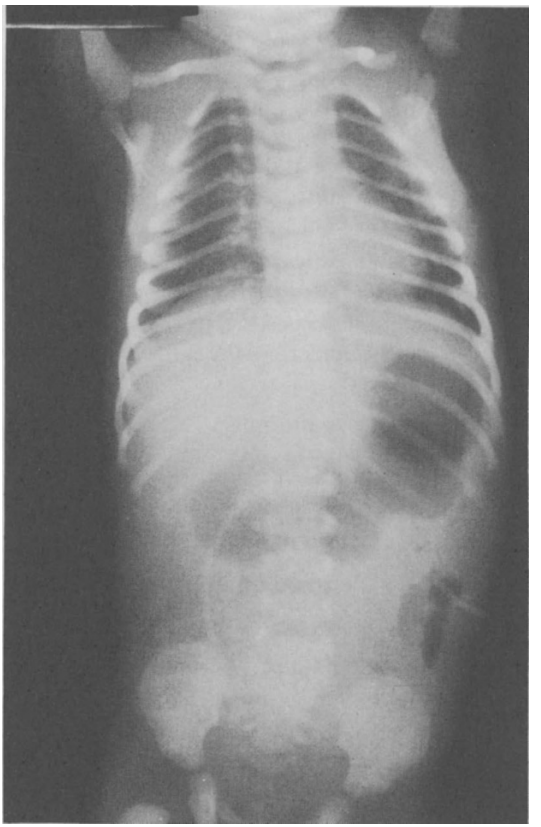


FIG. 8.2 Midgut volvulus

Baby had not passed meconium for 24 hours after birth, was vomiting and developed abdominal distension.
Note dilated stomach and duodenum. A little gas is present distally excluding atresia.

Air is present in the stomach of the newborn immediately after birth. It traverses the entire small bowel in 3 hours and most of the colon in 5

hours, and can be seen in the rectum in 6-8 hours after birth. For the diagnosis of intestinal obstruction in the newborn, air acts as an excellent contrast medium so that in most cases contrast studies are not needed. If it is at all necessary to carry out such studies (to confirm the diagnosis of oesophageal atresia, for example) lipiodol should be used instead of barium.

The baby with intestinal obstruction due to a congenital anomaly does not look ill and it is therefore necessary to investigate any vomiting in the first few days of life, especially if bile-stained. By the time the baby begins to look ill, pathological changes in the bowel are far advanced.

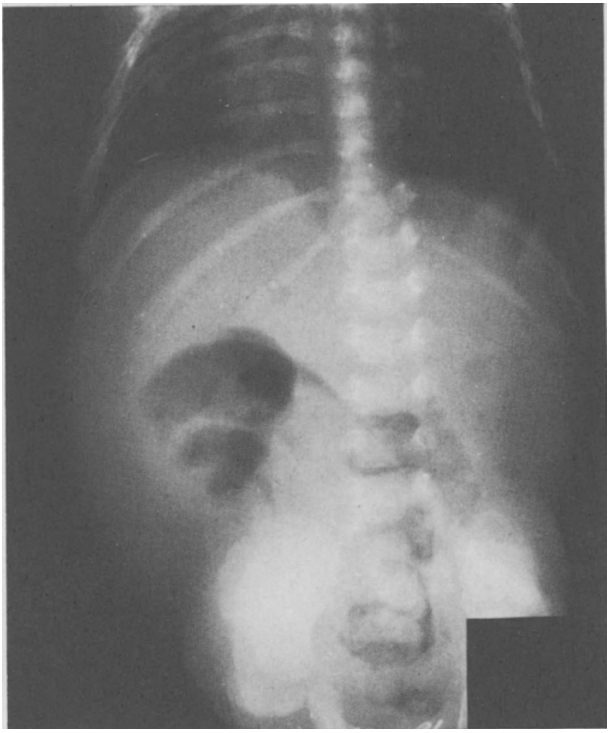


Fig. 8.3 Milk curd obstruction

Some dilated loops but no fluid levels.
Spontaneous improvement after bowel movement.

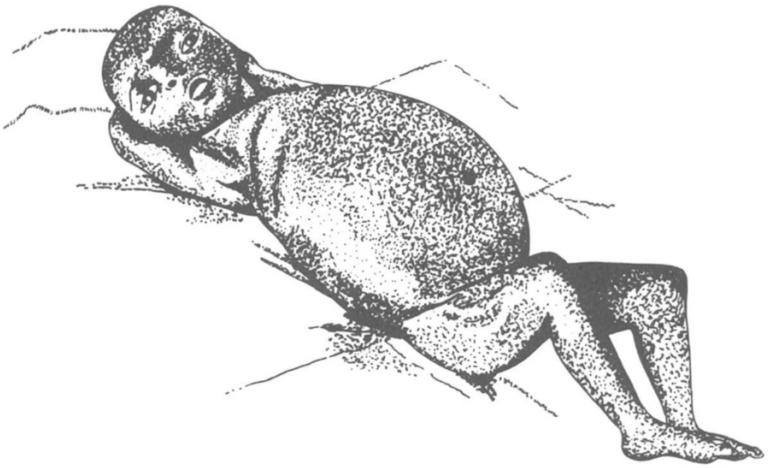


FIG. 8.4 Hirschsprung's disease

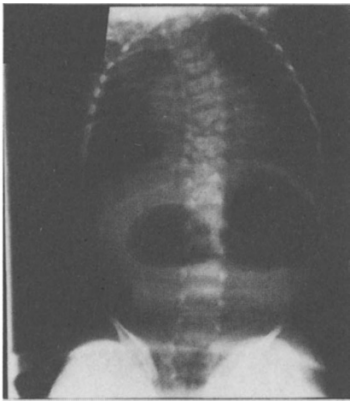


FIG. 8.5 Duodenal Atresia

Persistent vomiting after birth and absence of bowel movement for more than 24 hours with abdominal distension.
Note the 'double-bubble' appearance on plain x-ray.

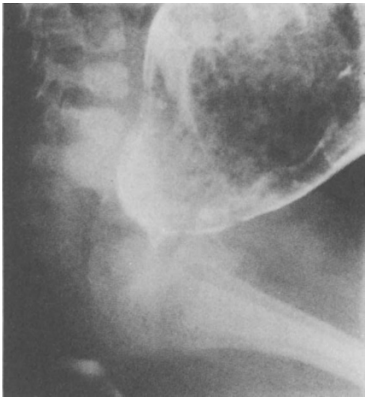


FIG. 8.6 Short segment Hirschsprung's disease.

Delay in diagnosis and treatment may lead to any of the following complications:

(1) Electrolyte imbalance.
(2) Aspiration pneumonia.

(3) Perforation of the gut due to enterocolitis.
(4) Strangulation and gangrene due to obliteration of blood supply.

Intestinal obstruction can occur in the absence of an organic lesion, for example by a plug of viscid meconium or disturbance of peristalsis as in sepsis or respiratory distress. Such functional obstruction responds well to conservative measures and treatment of the infection or the respiratory problem.

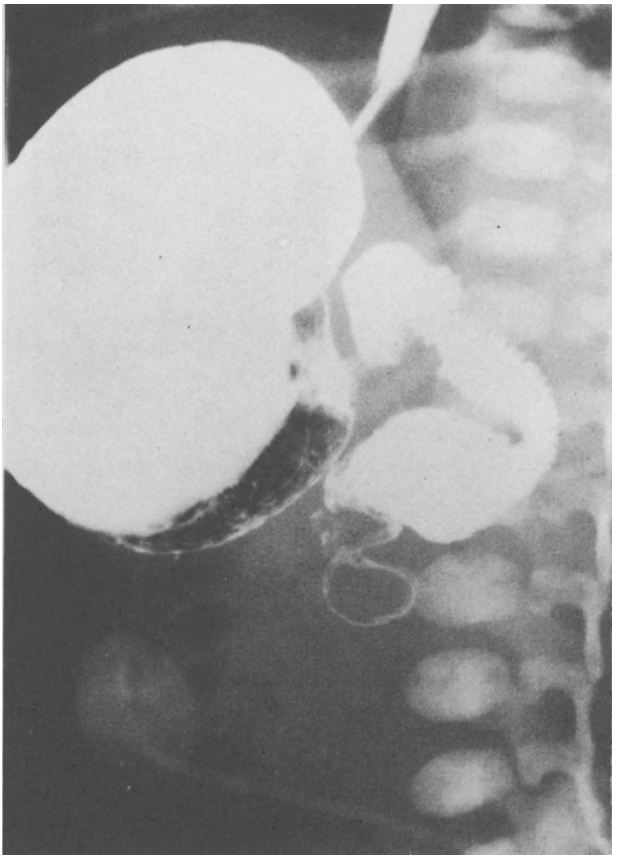


FIG. 8.7 Midgut volvulus
Dilated duodenal loop with the 'twisted ribbon' sign of volvulus.

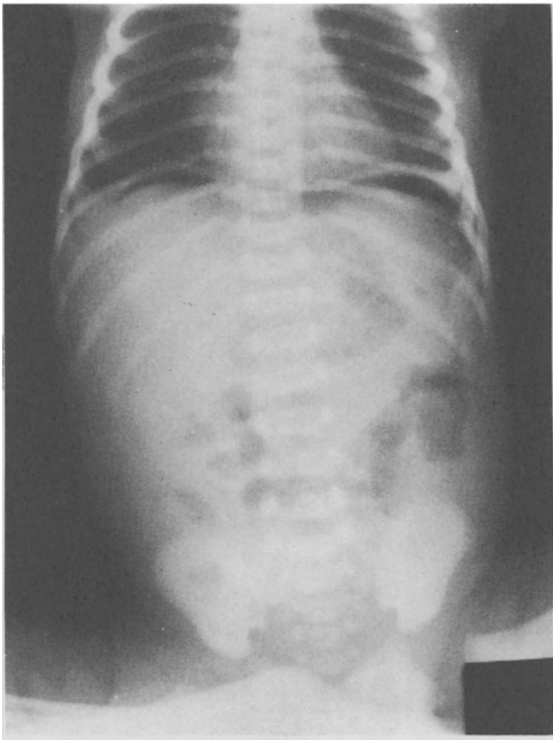


FIG. 8.8 Necrotising enterocolitis with gas under the diaphragm.

Neonatal necrotising enterocolitis (figure 8.8) presents intestinal obstruction and occurs particularly in premature infants or in those who have suffered from asphyxia, respiratory distress or apnoeic attacks in the immediate neonatal period. There are three possible factors in its aetiology – infection, intestinal ischaemia and stasis of gut contents. The mucous membrane of the terminal ileum and ascending colon undergoes haemorrhagic necrosis. It sloughs off in patches and in other areas there are submucosal gas-filled cysts. A plain x-ray of the abdomen will show pneumatosis intestinalis which is a characteristic diagnostic sign. In advanced cases, the x-ray shows free gas in the peritoneal cavity. Early cases are best treated conservatively with antibiotics, intravenous fluids and gastric suction. Surgery is necessary if signs of perforation and peritonitis are present.

Developmental defects in the newborn which require immediate

surgical intervention are exomphalos and open meningocele; surgery may be necessary in both defects to avoid infection setting in as a complication.

EMERGENCIES OF METABOLIC ORIGIN

Haemorrhagic disease of the newborn is occasionally seen (figure 8.9), displaying either echymoses, malaena or blood-stained vomitus. Such a haemorrhagic tendency usually occurs in babies of low birth weight, or those who have suffered anoxia at birth. Once established, there is a risk of damage to vital organs due to bleeding. Inj. vitamin K 5 mg should be administered on diagnosis, and repeated six hours later. Urgent blood transfusion may also become necessary. Bleeding from the cord stump is distressingly common, and at times fatal.

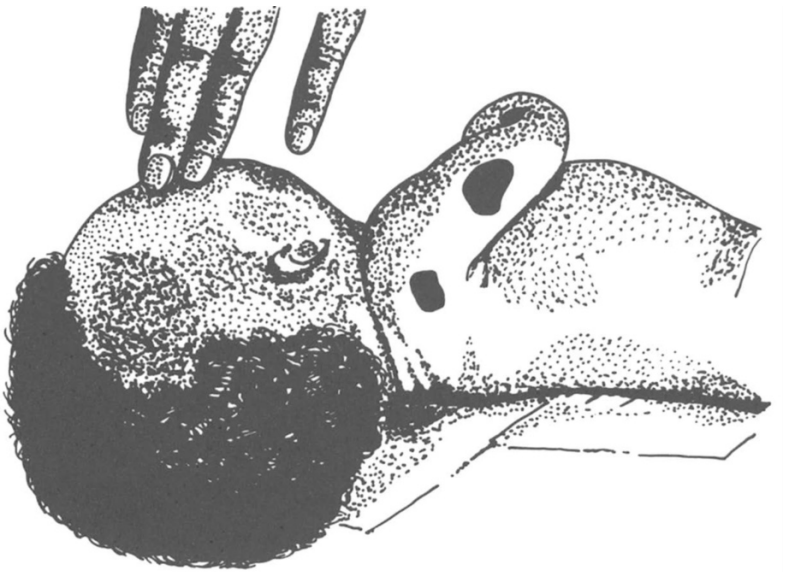


FIG. 8.9 Haemorrhagic disease of the newborn

Occasionally, the cause of dark stools may be maternal blood swallowed during delivery; similarly, blood from a cracked nipple may be swallowed and vomited. When in doubt regarding the cause of the blood, the alkaline denaturation test (Appendix III) will show whether the haemoglobin is of the adult or the foetal type. Foetal haemoglobin resists denaturation by alkali.

A serious and life-threatening coagulation defect occurs in sick or premature infants and is usually associated with abruptio placentae, severe intrauterine asphyxia, maternal toxaemia, hyaline membrane disease, infection with gram negative organisms and herpes simplex, shock and hypothermia. The haemorrhagic tendency is due to disseminated intravascular coagulation. The prothrombin time is normal but there is thrombocytopaenia and fibrinogen deficiency. Vitamin K is of no help in this condition and fresh heparinised blood is the best hope.

Convulsions in the newborn may be due to cerebral birth trauma, anoxic damage to brain, hypocalcaemic tetany or due to hypoglycaemia. According to their time of occurrence, fits in the newborn may be divided into two main groups – (i) those which occur within the first 48 hours after birth, and (ii) those which occur on the fifth or sixth day. Fits which occur in the first 48 hours are more likely to be associated with cerebral birth injury and usually carry a bad prognosis. When followed for a period of time, about a third to a half of such babies are known to die in the first few months of life, and amongst the survivors more than half show a neurological or intellectual deficit. In the case of fits occurring on the fifth or sixth day, there is no mortality and most babies show no sequelae. The most common causes in the latter group are hypocalcaemia and hypoglycaemia, which often exist together. Low levels of blood magnesium have also been associated with fits but the exact role of magnesium deficiency is not yet known.

In all instances it is important to control the convulsions as quickly as possible by means of Inj. Paraldehyde 0.2 ml/kg body weight or Inj. Phenobarbitone 3 mg-6 mg/kg body weight, while laboratory studies are undertaken to establish the cause of the convulsions. Pyridoxine-dependent convulsions have been known to be resistant to anticonvulsants. In resistant cases Pyridoxine 5 mg should be given by injection. Any delay in bringing convulsions under control may result in anoxic brain damage. Rare causes of neonatal fits are galactosaemia, maternal diabetes, and leucine sensitivity – all causes of hypoglycaemia – which should be considered if the routine laboratory investigations are normal.

Chapter 9

Respiratory Distress

Respiratory difficulties in the newborn can take different forms. The respiration may become irregular or gasping in character; there may be expiratory grunts or spells of apnoea. On the other hand, there can occur vigorous chest movements with inspiratory retraction of the chest wall.

In all newborns who are being observed for respiratory difficulty, the rate of breathing should be recorded at regular intervals. The trend of the respiratory rate in such babies is a useful guide for prognosis. A high mortality rate has been reported in those who show a steadily rising rate in the first few hours of life, especially if the heart rate is below 100 per minute.

In the healthy individual, the respiratory rate is usually the one that achieves optimal ventilation with a minimum of energy expenditure. One part of the work of respiration is to overcome the elastic recoil of the lung and the thorax when expanding them during inspiration; as the rate of respiration decreases this work increases. Another part of the work of respiration is to overcome airways resistance. This energy is proportional to the rate of airflow and will be greater when the respiratory rate is high.

Apnoea and tachypnoea are the two most common abnormalities of respiratory rate. Intermittent apnoeic episodes occur in prematures with immaturity of the central nervous system. Similarly, during recovery from the respiratory distress syndrome, apnoea and periodic breathing can occur from injury to the brain due to intracranial haemorrhage.

Respiratory distress in the form of increased rate, flaring of the nostrils, grunting and intercostal retraction is most commonly caused by the respiratory distress syndrome; meconium aspiration with or without pneumothorax, intrapartum asphyxia and congestive cardiac failure together account for most of the remainder. In an analysis of 100 consecutive cases of respiratory distress at the Ocean Road Hospital, Dar-es-Salaam, it was found that pneumonia and aspiration syn-

dromes are more frequent than the respiratory distress syndrome. The newborn's respiratory tract is sterile at birth, so that any lung infection may be assumed to be from infected secretions of the birth passages, infection reaching the liquor, or after mouth-to-mouth respiration. In these situations, prophylactic antibiotics should be administered to the newborn for at least three days. The common organisms obtained from throat and nasal swab cultures in babies with lung infections are *E. coli* and staphylococcus, and the antibiotic cover selected should be effective against these organisms.

In a study of respiratory problems of the newborn at the Hospital for Sick Children in Toronto[25] it was found that respiratory illness was the cause for admission in 21.7 per cent of cases. The most common cause of admission was the respiratory distress syndrome (7.2 per cent), followed by specific pulmonary conditions like aspiration, pneumothorax, pneumonia, etc. (5.5 per cent). Of the specific pulmonary conditions, half were caused by massive aspiration. The other diagnoses made were cardiac abnormalities (5.0 per cent) and extrapulmonary conditions (3.2 per cent).

Based on such observations made at several neonatal units, the common causes of respiratory distress in the first week of life are as shown in table 9.1.

TABLE 9.1 Causes of respiratory distress during the first week of life

	Disease	*Frequency*
(1)	Pulmonary conditions:	
	Respiratory distress syndrome	+++
	Massive aspiration	++
	Pneumonia	++
	Atelectasis	+
	Aspiration pneumonia	+
	Pulmonary haemorrhage	+
	Pneumothorax	+
	Congenital malformations of the upper respiratory tract	occasionally
(2)	Extrapulmonary conditions:	
	Congestive cardiac failure	++
	Oesophageal atresia	+
	Lesions of the central nervous system	+
	Diaphragmatic hernia	occasionally

In most instances diagnosis can be made by clinical observation, physical examination and simple radiological investigations. When respiratory difficulty is due to a cerebral lesion, the respiratory drive is

diminished. Irregular respiration or spells of apnoea should raise the suspicion of intracranial pathology. There may also be a bulging fontanelle or a cerebral cry.

Presence of the triad of tachycardia, tachpnoea and hepatomegaly occurs commonly in congestive cardiac failure.

If the baby brings up frothy mucus continuously, oesophageal atresia should be suspected and can be confirmed by the obstruction felt on attempting to pass a naso-gastric tube (figure 9.1).

Diaphragmatic hernia is more commonly on the left side and causes a shift of mediastinal structures to the right, with absence of air entry on the involved side of the chest.

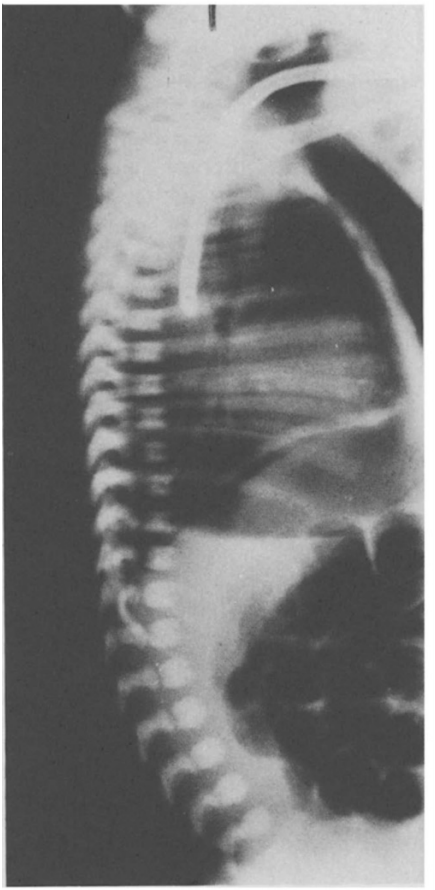


Fig. 9.1 Oesophageal atresia with catheter arrested at D 5-6 level

The respiratory distress syndrome occurs in pre-term babies, in those who suffered from antepartum and intrapartum anoxia, in babies of diabetic mothers and in those delivered by caesarean section. Prematurity is by far the commonest condition associated with it. The crucial factor in its aetiology is a deficiency of the lipo-protein lecithin which acts as a surfactant in the alveoli. The surfactant is formed in the alveolar cells and its main function is to lower surface tension at the air-liquid interface in the alveoli. Reduction of surface tension prevents collapse of the alveoli during expiration and facilitates the expansion of the lungs during inspiration. In the respiratory distress syndrome, absence of the surfactant causes the alveoli to collapse at the end of each expiration so that greater effort is required for inflating the lungs at each breath. This continuing effort for breathing soon leads to exhaustion. Moreover, because the air sacs remain collapsed, a large proportion of the blood coursing through the lungs remains unoxygenated, leading to further complications like hypoxaemia and acidosis. Both of these, in turn, interfere with the synthesis of the surfactant in the alveolar cells and thus a vicious circle builds up. Hypothermia has a similar effect. It has also been observed that in babies with the respiratory distress syndrome, mortality is much lower in those who were able to establish spontaneous respiration at birth compared with the ones who were severely asphyxiated.

A baby suffering from the respiratory distress syndrome needs intensive care with prompt resuscitation at birth, maintenance of body warmth, biochemical monitoring (especially for acidosis) and adequate ventilation of the lungs. Until recently the latter required intubation and intermittent positive pressure ventilation, which was not possible in most hospitals and health centres of the developing countries. It has now been shown that adequate ventilation can be achieved by applying a constant distending pressure to the lungs so that at the end of expiration the alveoli are held open by this distending pressure.[26] The distending pressure is applied through a hood which encloses the baby's head (the Gregory box), and more recently the concept has been adapted to provide continuous distending pressure through nasal prongs. These developments will help to simplify the management of the respiratory distress syndrome so that more and more health institutions in the developing countries will be able to provide adequate care for babies suffering from the syndrome.

Chapter 10

Feeding Difficulties

Mothers from rural communities usually have no difficulty with establishing lactation; amongst them breast feeding is the rule and attempts at 'teaching' may only serve to make them unduly anxious and suppress lactation. It is therefore best to interfere only when difficulties arise.

Several physiologic changes occur during pregnancy which prepare the mother for breast feeding. The body's metabolism alters so that stores of energy are laid down in the form of body fat. In the case of the average well-nourished woman, this increase in body fat is partly responsible for the gain in weight during pregnancy. The total increase in body fat is to the extent of 4 kg in such women, representing 3,500 calories. Most of it is used up in lactation. For example, it has been shown that even when diets are adequate, the nursing mother loses weight at the rate of 0.28 kg per week. Mothers whose diets are deficient accumulate only small amount of body stores of energy, or none at all. Many such mothers are known to lose their own body weight and become thinner over a period of prolonged lactation. Hence it is important to emphasise adequate food intake in all lactating mothers. Human milk has a low protein content, in the range of 1 per cent, and most of it can be found in a diet which provides adequate calories. Thus, even though protein is essential, food in the form of a mixed diet sufficient to satisfy hunger and to supply the energy needs of the body is usually adequate to meet the nutritional needs of lactation.

Physiological changes also occur in the breast tissue during pregnancy, to prepare the gland for secretion of milk. Under the influence of oestrogen, progesterone and the placental mammotrophic hormone, there is a growth of alveoli and lobules of the breasts, resulting in an increase in size of the glands. The lactational performance is closely related to the growth in size of the breasts. These changes are climaxed by the release of prolactin from the anterior pituitary at the time of birth. Prolactin acts by initiating the synthesis and secretion of milk. The act of suckling by the baby starts a reflex arc which results in the further secretion of prolactin from the anterior lobe and oxytocin from

the posterior lobe of the pituitary gland. Oxytocin promotes the ejection of milk from the acini and ducts by causing the contraction of involuntary muscle fibres in the breast. Complete emptying of the breasts at each feed by the baby is the single most important stimulus to maintain a regular flow of milk.

Mother's milk is the ideal food for the newly born. Experience in all developing countries has shown that most babies thrive well and show adequate growth in the first 4 to 6 months of life on their mothers' milk alone. Most mothers are able to secrete adequate quantities of milk for the requirements of their babies in the first six months of life and appreciable amounts thereafter, to make a significant contribution to the diets of their children until the age of 2 years.

The various nutrients in human milk are delicately balanced for the optimum growth of the baby. In addition, breast milk contains several substances which act in combination as defence mechanisms against infection. It contains immunoglobulins with antibody activity against several micro-organisms. The predominant immunoglobulin is IgA, which is conjugated in breast tissue and is present in breast milk in concentrations higher than those in mother's serum. The IgA molecule in breast milk has a configuration which makes it resistant to pH changes and enzyme attack in the gut. Human milk also contains a large number of leucocytes – 2,000 to 4,000/cu mm – mainly as macrophages and lymphocytes which protect against invading bacteria and viruses. Other factors like lysozyme, which is bactericidal, and lactoferrin (an iron binding protein) which is bacteriostatic against pathogenic *E. coli,* also contribute to the overall defence against infection. In addition, the low phosphate content and the poor buffering capacity of breast milk together with several factors like the bifidus factor and other lesser known substances help to keep out *E. coli* and enable non-pathogenic organisms like the lactobacillus to colonise the baby's gut. All these various agents act in concert and constitute a biological system for the protection of the baby against infection. Thus, by providing nutrition as well as protection against infection, the mammary gland performs many of the functions which the placenta carries out in intrauterine life.

In recent years there has been an increasing decline in breast feeding in almost all developing countries. Several factors are responsible for this decline, but perhaps the most important are the intensive promotional activities by manufacturers of powdered milks, including the issue of 'gift packs' consisting of a tin of milk powder, a feeding bottle and a colour brochure, in maternity wards. Such gift packs sow seeds of doubt in the mothers' minds regarding their choice of giving breast milk to their babies. The most effective stimulus to the establishment of lactation in mothers is active suckling and emptying of the breasts by

the baby. If feeds are offered to the baby by bottle in the crucial early days, such stimulation does not occur and there is a failure of lactation.

The dangers of bottle feeding the infant in the unsanitary environment of the rural home are well known. In addition, the constituents of most milk powders make them highly unsuitable for infant feeding. Most of them are based on cows' milk, which has low calcium and high phosphate, so that neonatal tetany is more common in artificially fed infants. Many of the powdered milks contain large amounts of sodium, potassium and other solutes, and since kidney function in all newborns is not well developed they find it difficult to handle the excessive amount of solutes. Babies who are fed on artificial milks have a higher plasma osmolality compared with those fed on the breast. In such cases even a mild diarrhoea can precipitate hypernatraemia, with serious consequences. During their manufacture, carbohydrates like lactose, sucrose, fructose, dextri-maltose are added to several brands, and half the quantity by weight of the powdered milk may consist of such carbohydrates. In the same way the composition of fats and proteins are also altered. Unlike the adult who eats a variety of foods, the newborn is committed to form its body tissues from only one food – that is, the milk which he receives – and interference with the constituents of milk is likely to produce adverse effects. There is therefore a calculated risk in bottle feeding any infant, and *the baby in the rural area of a developing country who has no access to breast milk has virtually no chance of survival.*

The healthy newborn is endowed with the appropriate nervous and reflex mechanisms necessary for his food intake. These consist of the rooting, suckling and swallowing reflexes. The rooting reflex consists of the baby turning his head so as to take the nipple into the mouth, when his cheek is touched by the mother's breast. The baby does not suck at the nipple, but 'milks' it by means of jaw and tongue movements which press the nipple against the hard palate.

The cause of a feeding difficulty may be present in either the mother or the baby.

CAUSES IN THE MOTHER

Cracked nipples – This is a very common cause usually due to a hungry baby who chews at the nipple producing minor ulcerations. It causes painful tender nipples.

Treatment consists of stopping breast feeding for a few days. The milk from the affected breast can be expressed either by hand or by a breast pump and offered by spoon to the baby.

Any infection in cracked nipples should be treated energetically because it may spread to the acinar tissue and cause a breast abscess.

Failure of lactation – Nervousness about her ability to establish lactation in a tense mother is a known cause of failure in the higher social class. However, in village mothers any failure to lactate is usually due to ill health, poor nutrition or some other organic cause. A search for the underlying cause and its treatment, with reassurance, attention to diet and putting the baby on the breast regularly may help to initiate lactation. Serpasil (0.25 mg twice daily) has been found useful in some cases. In the unfortunate event of a maternal death, lactation has been successfully induced in a grandmother or a female relative by this method.

In rural societies availability of breast milk is a vital factor in infant survival; hence before a mother is discharged from hospital it is necessary to ensure the establishment of adequate lactation.

Multiple pregnancies – In cases of twins or triplets the mother's milk may be inadequate. The mother needs reassurance most of all; she should be advised to feed the smaller baby first and offer both the breasts, and then follow suit with the bigger baby. In most cases the production of milk will increase, keeping pace with the emptying of the breasts. If this fails, supplementary feeds should be given using expressed breast milk from other mothers. All such supplementary feeds should be offered with a cup and spoon, avoiding the use of bottles.

CAUSES IN THE BABY

Absence of sucking and swallowing reflexes – in cases of immaturity of the central nervous system as in premature babies, or in disease, the baby may not be capable of food intake. Such a baby needs to be fed by means of a naso-gastric tube.

If any of the feeds are brought up, aspiration into the lungs may occur because of the absence of a swallowing reflex. The danger in tube feeding is from too large rather than too small a feed, and especially from too rapid administration. The milk should be allowed to flow down slowly under the influence of gravity rather than forced with a syringe.

Physical inability to suckle – The baby may not be able to suckle in certain congenital defects involving the oral cavity (e.g. hare lip, cleft palate or micrognathia). In such cases the mother should be taught how to express her milk and feed the baby with a teaspoon. Careful supervision of the baby for adequate weight gain is essential.

Some babies with cerebral palsy may not be able to feed on the breast and need to be fed with a spoon or by means of a tube.

In those with respiratory distress or congenital heart disease with failure, breast feeding may not be possible or advisable, and tube feeding will have to be resorted to.

Candida infection of the oral cavity may interfere with suckling. It

responds well to painting the mouth with gentian violet or to nystatin drops. (Concentrated solutions of gentian violet have been known to cause stomatitis in the newborn.)

Inability to suckle in a week-old baby who could feed well at birth is a common mode of presentation in neonatal tetanus. Feeding difficulties are noticed a day or two before the onset of spasms and may suggest the diagnosis of tetanus of the newborn.

Regurgitation and vomiting – These are common symptoms in the newborn baby. In a large majority vomiting is due to gastric irritation caused by swallowed liquor, and emptying the stomach contents relieves it; in some babies vomiting may be due to obstruction in the gastrointestinal tract or a symptom of cerebral birth trauma, and further studies are needed if vomiting does not settle with a stomach wash.

Diarrhoea in a breast fed baby – After the stage of transitional stools, the average breast fed baby has about four semi-solid stools per day. If frequency increases or consistency becomes more liquid, diarrhoea may be suspected. After ruling out infection a metabolic abnormality in the form of lactose intolerance may be considered; if the pH of the stool is below 5 the stool should be examined for sugar. The hemacombistix and clinitest serve as useful screening tests (Appendix III). Where laboratory facilities permit, a lactose tolerance test can be performed. Babies with alactasia will not show a rise in blood sugar when given oral lactose.

THE WORKING MOTHER

With the present rate of socio-economic development in most of tropical Africa, the problem of feeding the baby of the working mother assumes an ever increasing importance. The provision of crêches and day-care centres may become necessary as women form an increasing proportion of the labour force. Until such facilities become widespread the question of feeding the baby of a working mother will have to be tackled at the individual level. It will be advantageous to advise on as many breast feeds as possible when the mother is at home; during working hours the baby will have to be fed on fresh cows' milk. Bottle feeding must be avoided and the mother should be taught how to feed with a cup and spoon. Instructions in simple methods of cleanliness and preparation of feeds should be given before the mother leaves the maternity wards and, where possible, she should be visited at home so that practical instructions can be given in the home environment and an assessment be made of home facilities for the care of the baby.

Chapter 11

Low Birth Weight

The normal newborn tends to weigh much less in developing countries compared with his counterpart in Western Europe. Thus, in Nigeria the average birth weight is reported as 3,090 g and in Gambia it is 2,835 g. In the same country there may be significant differences in birth weights in different regions depending upon the general standards of health and the socio-economic level of the community. Mothers from a higher social class who live in healthy surroundings and enjoy good health tend to produce heavier babies than those who come from a lower social class, have marginal nutrition and suffer chronic ill-health. In a comparison of birth weights between two social classes, it was found that the mean birth weight in social class I was 3,290 g as compared with 2,950 g in social class IV.[21]

In the unhygienic environments of many homes in tropical Africa, newborns, especially those with a low birth weight, are prone to a variety of infections. In an analysis of infant mortality in Madras it was found that 73 per cent of all deaths occurred in infants with birth weights of less than 2.0 kg[27]. It would appear that whereas birth weight in an individual child may convey little in the form of prognosis, in any given community the average birth weight serves as an index of the newborn's viability.

A large proportion of early neonatal deaths take place in the low

TABLE 11.1 Neonatal deaths in two centres in East Africa

D.DAR-ES-SALAAM, TANZANIA				*KAMP.KAMPALA, UGANDA*			
Total born	*Total died*	*Deaths under 2,500 g*	*Mortality %*	*Total born*	*Total died*	*Deaths under 2,500 g*	*Mortality %*
5,476	128	95	74.2	4,093	108	76	70.3
6,841	146	93	63.7	7,001	146	78	53.4
8,456	180	131	72.7	8,696	164	103	62.8
10,778	153	146	95.4	10,460	208	144	69.2

birth weight baby. In table 11.1, mortality figures from two large maternity centres are shown over a four-year period.

The level of 2,500 g in table 11.1 is the normally accepted criterion in Western Europe and North America. It has been the experience in many developing countries that babies weighing 2,500 g or less often possess a full potential for growth and health and are viable. Hence a different criterion for low birth weight is necessary. Table 11.2 compares neonatal deaths with birth weights over a period of one year.

TABLE 11.2 Neonatal deaths compared with birth weights

Birth weight	*Total born*	*Deaths*	*% Mortality*
2,270	503	5	1
2,050	299	15	6
1,800	184	13	7.1
1,600	100	25	25
1,360	68	33	49
1,127	21	15	71.4
900	15	14	93.3

The mortality per cent increases from birth weight of 2,000 g downwards. Taking the mortality data alone it would appear that low birth weight *per se* contributes to the neonatal mortality from 2,000 g and below, and that intensive care is necessary for this group.

CAUSES OF LOW BIRTH WEIGHT

Antenatal care

Adequate antenatal care is by far the most important factor in the prevention of low birth weight. Besides the total number of antenatal visits made the quality of care is also important.

In one study of low birth weights it was found that from a group of mothers who gave birth to very small babies (1600 g and under), 26.5 per cent did not attend for antenatal care as compared with only 8 per cent in mothers who gave birth to babies weighing more than 1,600 g. It was also found that of all early deaths in the low birth weight babies, about a quarter take place amongst those whose mothers did not receive any antenatal care.[28]

Socio-economic factors

Reference has already been made to the effects of nutrition and social class on birth weight. Women of the poorer classes not only suffer from chronic ill-health and under-nutrition but also have to continue to do

manual labour in the fields until very late in pregnancy. The birth weights in these two classes are compared in table 11.3 (see also figure 11.1).

TABLE 11.3 Birth weight according to social class*

Class	*Average birth weight (g)*	*% Born under 2,500 g*	*% Born under 2,050 g*
High social class	3,200	4	3
Low social class	2,950	13.8	4.9

* Figures from Ocean Road Maternity Hospital, Dar-es-Salaam (1966)

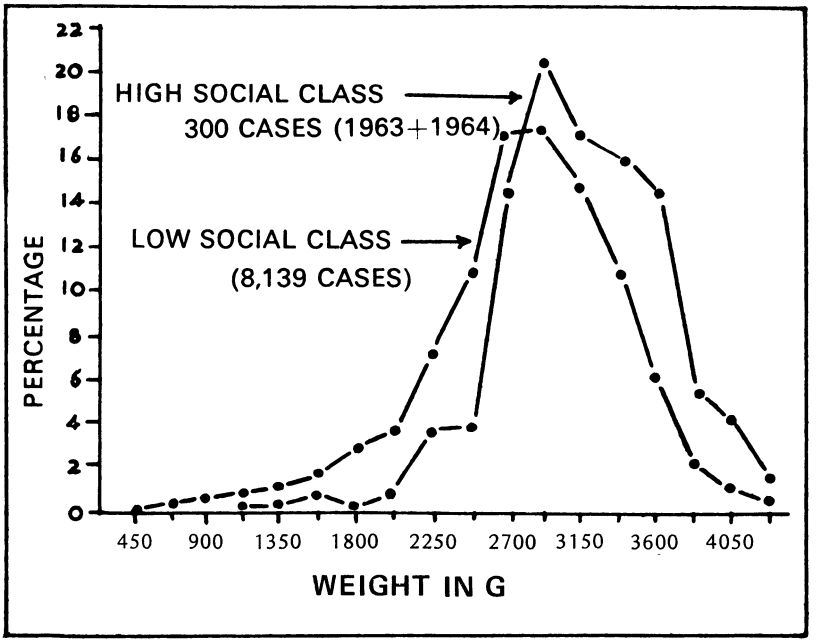


FIG. 11.1 Birth weight according to social class

Parity and sex

The first born comprises a large proportion of the small babies (24.7 per cent in one series) and after the fourth pregnancy the risk of perinatal mortality is high.

Though there is a higher incidence of low birth weight amongst female babies, the overall mortality is higher among male infants.

Multiple pregnancy

In multiple pregnancies, the average birth weight is lower than normal but the infants are more mature than the birth weight would indicate.

Maternal illnesses

Common maternal illnesses which interfere with foetal growth are pre-eclamptic toxaemia, eclampsia, hypertension, and anaemia. The overall effect of these factors in the production of low birth weight is set out in table 11.4.[21]

TABLE 11.4 Effect of maternal illness on birth weight

Weight group	*Mothers attending for antenatal care*	*Maternal illness in those attending for antenatal care*	*Abnormal labour*	*Mortality*
(g)	*%*	*%*	*%*	*%*
900	33	100	Nil	93.3
1,127	33	100	24.9	71.4
1,360	69	38	11.8	49
1,600	64	34	17	25
1,800	66	40	1.6	7.1
2,050	67	37	18	6
2,270	67	26	13.3	1

The effect of malarial infection of the placenta on the birth weight of the infant has been mentioned previously.

FACTORS IN THE PREVENTION OF LOW BIRTH WEIGHT

In most developing countries, facilities for the care of low birth weight babies are virtually non-existent, hence emphasis should be put on preventive programmes. Such a programme, built into the overall maternal and child health programme of the country, will have two broad objectives.

(1) *Improvement of the general health of the mother* – Since the health of the mother cannot be separated from the general standard of health in the community of which she is a part, it is important to develop basic health facilities in the community with relevant emphasis on maternal and child

health. A community which is better nourished, has an adequate socio-economic standard and from which chronic ill-health has been eliminated has a lower incidence of low birth weight.[29]

(2) *Improvement of the antenatal health of the mother* – A health education programme is essential to stress the need for antenatal care and health supervision of the mother during pregnancy. Together with such a programme there should be an expansion of available services for the pregnant women with prenatal complications.

A well-balanced diet, early detection and treatment of anaemia, prevention of heavy malarial infection and chronic ill-health in the mother will help to decrease the incidence of low birth weight in the community.

CARE OF THE LOW BIRTH WEIGHT BABY

The principles of care are based on the ordinary management of any newborn with certain modifications because of the special needs of the low birth weight baby.

(1) Establishment and maintenance of respiration.
(2) Maintenance of body heat.
(3) Avoiding physical exhaustion.
(4) Management of feeding.
(5) Protection against infection.

To what extent these needs can be met will depend upon available staff and equipment.

The nursery

All low birth weight babies should be admitted to a separate nursery together with the mother who stays in an adjoining dormitory. The nursery should be situated in the maternity wing of the hospital as far away as possible from the general sick wards. Special full-time nursing staff should be assigned to the premature nursery. The likelihood of bacterial infection is reduced if all relatives and visitors are excluded from the nursery and the coming and goings are reduced to a minimum.

Admission policy

(1) All babies of low birth weight born in the hospital should be admitted to the nursery without delay.
(2) All such babies born outside the hospital and less than 24

hours old should be kept in a separate room and given Inj. Penicillin and Streptomycin for three days. They should be sponged with Savlon solution (1 in 200) on entering the hospital and again on admission to the nursery.

(3) All low birth weight babies born outside the hospital and more than 24 hours old should not be allowed into the nursery, but cared for in the side-room of a children's ward.

WARMTH IN THE NURSERY

The resting or basal heat production (B.M.R.) of the baby is low at birth and rises progressively during the first ten days of life. Heat production per unit surface area of the body remains less than that of the adult for nearly three months after birth, during which period it is important to conserve body warmth in the baby. Resting heat production per unit surface area is particularly low in babies of low birth weight. The insulation of the body against heat loss is only half of that in the adult male in the case of the full-term baby, and only a third in the case of a baby weighing 1kg.

An effective way of conserving body temperature is by means of clothing. All small newborns should be well covered with a woollen vest, a towelling napkin, a long nightdress and swaddled with a flannel sheet with two layers of cotton blanket. In such babies the body temperature will fluctuate very little, and then only gradually, with variations in the room temperature. Table 11.5 provides a guide to desirable room temperatures for a draught-free nursery.

TABLE 11.5 A guide to desirable room temperatures

Birth weight (kg)	*Room temperature*		
	29.5°C (85°F)	*26.5°C (80°F)*	*24°C (75°F)*
1.0	For 2 weeks →	After 2 weeks →	After 1 month
1.5	For 2 days →	After 2 days →	After 2 weeks
2.0	–	For 1 week →	After 1 week
3.0	–	For 1 day →	After 1 day

Mortality rises if small babies are nursed in surroundings more than a a degree or so below the temperature required to maintain body warmth. In most tropical countries environmental temperature is not a problem, and an ordinary electric wall heater with or without a steam kettle in a corner of the room is enough in the majority of cases. A room at 24°C provides neutral conditions for a full-term cot-nursed baby after the age of 2 days. A temperature of 30°C is necessary to provide comparable warmth for a 1 kg baby in the first few days of life.

PROTECTION FROM INFECTION

All medical personnel with obvious upper respiratory tract infections should be excluded from the nursery.

Washing of hands before and after touching a baby, gown and mask technique, and scrupulous care during any handling of the baby are essential. These principles should also be impressed on the mothers who have to handle the babies during feeds.

FEEDING

When the baby has recovered from the effects of labour, which varies in time between 2 to 6 hours, the first feed may be offered in the form of breast milk or, if not available, as 5 per cent glucose. The blood sugar tends to be lower in the low birth weight baby and early feeding will protect against hypoglycaemia. Except where the baby is grossly oedematous, prolonged starvation is not indicated.

Even very small babies can tolerate breast milk well and therefore dilution of feeds is not necessary. The possibility of introducing infection during dilution is greater than that of gastric upsets due to undiluted breast milk.

It is more important to devote time and attention to the *method* of feeding than to any complicated formula. Where suckling and swallowing reflexes are present, the baby should be breast fed. Since the small baby tires easily, 15-30g expressed breast milk should be given as a supplement by pipette after breast feeding. If the suckling reflex is not present, the baby should be fed by means of a naso-gastric tube with expressed breast milk (15g) at three hourly intervals. Depending upon the baby's growth and progress the quantity and method of feeding can be adjusted. As a rough guide, babies above 1.5 k in weight are able to suckle and swallow; also those babies who have a well developed Moro reflex and supraciliary tap reflex are usually mature enough to be able to suckle and swallow.

DISCHARGE FROM HOSPITAL

Most low birth weight infants can be discharged when they are able to feed satisfactorily, show stable temperature control under normal room temperature, and have no clinical evidence of disease. Usually these criteria are fulfilled when the body weight is 1,800 g or higher. Hypothermia, especially during the winter months, and infection are the main hazards for such babies after they have been discharged. Regular supervision, in the form of home visiting, and parental education in the care of the small baby at home are important for the wellbeing of these infants.

SUBSEQUENT PROGRESS

The low birth weight baby is prone to several complications for which a close watch should be maintained.

Respiratory distress syndrome – Irregular breathing and spells of apnoea are common in the low birth weight baby. There may also be early signs of lung complications or hypoglycaemia and therefore all such babies should be kept under close observation. Babies who have a respiratory rate of more than 50 per minute after the first hour of life need to be observed for this complication.

Hypoglycaemia – Early detection and treatment of hypoglycaemia is essential because of its effect on the glucose-dependent central nervous system. The symptoms may be non-specific; thus colour changes and cyanosis, apnoea or irregular respiration, refusal to feed, a high pitched or weak cry and 'jitteriness' are more common than convulsions. Several of these signs occurring together should attract attention; the diagnosis is established by a blood sugar level of 20 mg/100 ml or less, and the response to intravenous glucose.

Once the diagnosis is established, therapy with intravenous glucose should be rapidly instituted. Oral glucose has little value since the hypoglycaemia is resistant and responds only to prolonged intravenous glucose, such as 10-15 per cent strength at the rate of 30 to 40 ml per 500 g body weight in 24 hours.

Necrotising enterocolitis – This is particularly common in small and pre-term infants, especially if the infant has also suffered from birth asphyxia, respiratory distress syndrome, apnoeic attacks, or jaundice. The symptoms indicate intestinal obstruction. In severe cases perforation of the ileum or the colon can occur leading to peritonitis, shock, generalised sepsis and death. Immediate action should involve gastric aspiration and intravenous fluids. If umbilical catheters are present they should be removed. Enterocolitis occurs at a time when the bowel is first being colonised by bacteria, and blood cultures are positive for a variety of faecal organisms. Appropriate antibiotics should be administered. These infants do not stand up to surgery well, but recovery is possible in many with conservative management, and an operation is necessary only if the bowel perforates.

Haemorrhage – The haemorrhagic tendency present in all newborns is more pronounced in the low birth weight baby. Sudden deaths due to intraventricular haemorrhage are often noted. Injection of vitamin K 1 mg should be routinely given to all low birth weight babies.

Jaundice – Physiological jaundice is more intense and more prolonged than in the case of normal newborns.

Anaemia – As growth proceeds the available iron stores may become rapidly utilised and anaemia may result. All low birth weight babies should be given oral iron routinely.

Tolerance to drug therapy – The low birth weight baby has poor tolerance to drugs and so in prescribing any therapy the dosage should, as a general rule, be half of that for normal newborns. Adverse reactions may occur with drugs like vitamin K and sulphonamides, both of which may increase the tendency for hyperbilirubinaemia; chloramphenicol in the usual dosage produces a syndrome of collapse and shock referred to as 'the grey syndrome' because of the ashen grey colour of the baby. Even oxygen therapy may be toxic and produces retrolental fibroplasia if administered in a concentration of more than 40 per cent for long periods.

LONG TERM PROGNOSIS

All the complications mentioned above provide an explanation for the higher perinatal mortality of these babies. There is a steep rise in neonatal deaths for birth weights of 2,000 g and below; the mortality risk is about 25 per cent in the 1,800 g weight group and rises to about 50 per cent in the 1,500 g weight group. However, survival is only the initial objective; as more and more of these babies are being saved and followed, it has become apparent that many of them suffer from physical and mental handicaps in later life, usually in the form of cerebral palsy and developmental retardation. Therefore, after survival has been ensured the problem of normal growth and development still remains.

The effect of social background and home environment is important in the intellectual development of a baby. Follow-up studies in low birth weight babies have shown that babies brought up in homes of higher social classes do well developmentally and have a higher I.Q. score compared with those brought up in families of lower social class. Thus, not only is the incidence of low birth weight babies higher in the lower social class but the overall intellectual growth and stimulation at home appears to be lacking.[30] There would therefore be all the more reason to follow these 'at risk' babies of low birth weight in clinics separate from the ordinary young child clinic.

Many low birth weight babies catch up with normal infants in growth by the second year of life. The major problems in early childhood are congenital defects and physical abnormalities in addition to the cerebral palsy and mental defects mentioned above. These complications are, as a general rule, more often seen in babies with birth weights lower than 1,600 g.

Chapter 12

Jaundice

The normal destruction of red blood cells accounts for 80 to 90 per cent of the bilirubin production in the adult. In the newborn infant it accounts for 75 per cent or less; the remainder comes from the catabolism of tissue haeme and ineffective erythropoiesis, that is, the destruction of red blood cells precursors in the bone marrow or soon after their release into the blood.

The conversion of haemoglobin to bile pigment takes place in the reticulo-endothelial system. 1 g of haemoglobin gives rise to 35 mg of bilirubin. The normal newborn produces 8.5 mg/kg body weight of bilirubin per day, which is more .than twice the rate in the adult. On leaving the reticulo-endothelial system, bilirubin is transported in the plasma in a combined form with albumin. The parenchymal cells of the liver have a selective capacity for taking up unconjugated bilirubin from the bloodstream. Inside the liver cell, bilirubin gets conjugated with glucuronic acid to form the conjugated form (bilirubin glucuronide) which is then excreted in bile. The enzyme concerned with the conjugation of bilirubin is glucuronyl transferase. Drugs given to the newborn or other metabolic factors like acidosis and asphyxia may delay the development of this enzyme or present an extra load for excretion and thus interfere with bilirubin metabolism. This explains the association of jaundice with the administration of drugs like chloramphenicol, vitamin K analogues, salicylates, etc. in the newborn. On the other hand, enzyme activity is stimulated by phenobarbitone, which is now occasionally used in the prophylaxis of neonatal jaundice.

The liver of the newborn baby has a limited capacity for conjugation of bilirubin. The average baby weighing 2,950 g has to clear a bilirubin load of about 17 mg in twenty-four hours. Any portion that is not excreted will accumulate in the blood stream in the unconjugated form. This is the basis of physiological jaundice which is seen in a large proportion of babies. It does not usually appear until the second or third day after birth and in the full-term infant it will usually not be present after the sixth day. In the low birth weight baby where the

development of enzymes may be slow, physiological jaundice may last longer.

Because of this tendency for hyperbilirubinaemia, the cord blood in the newborn shows a mean bilirubin value of 1.8-2 mg per 100 ml. Peak values in the serum will vary according to the excretory capacity of the liver and range between 2 mg and 12 mg per 100 ml in the first twenty-four hours. On average, 25 to 50 per cent of all full-term normal newborns and a considerably higher percentage of pre-term babies show clinical jaundice in the first week of life. In dark-skinned babies, clinical jaundice may not be readily apparent and physiological jaundice may be missed. Any level of bilirubin greater than 12 mg per 100 ml in the full-term infant calls for investigation.

Jaundice may also occur in cases of haemolytic disorders which increase the pigment load to be cleared by the liver, or in cases of incapacity of liver cells, either developmental or due to noxious agents; in which case the serum will also show raised levels of conjugated bilirubin.

In a survey of 8,000 consecutive births[31] it was found that 40 cases of jaundice needed clinical and laboratory investigations. The aetiology of the jaundice was as shown in table 12.1.

TABLE 12.1 Causes of jaundice in 40 cases investigated[31]

	Cases	*Percentage*
Neonatal hyperbilirubinaemia	20	50
Jaundice due to infection	5	12.5
Jaundice due to biliary atresia	25	12.5
Miscellaneous (including prematurity)	10	25

Twenty of these needed exchange transfusion. In 12 (60 per cent) it was carried out for A-B-O incompatibility and in 5 (25 per cent) it was due to Rh incompatibility. In three (15 per cent) no incompatibility was demonstrable (see figures 12.1 and 12.2).

CRITERIA FOR INVESTIGATION

In all investigations of hyperbilirubinaemia, it is important to realise that the pigment is affected by exposure to ultra-violet light. Serum exposed to ordinary daylight may lose as much as 33 per cent of the pigment in one hour. All blood samples should be carried to the laboratory in a covered container immediately after collection.

In the following circumstances, jaundice in the newborn is pathological, and detailed laboratory studies may be necessary.

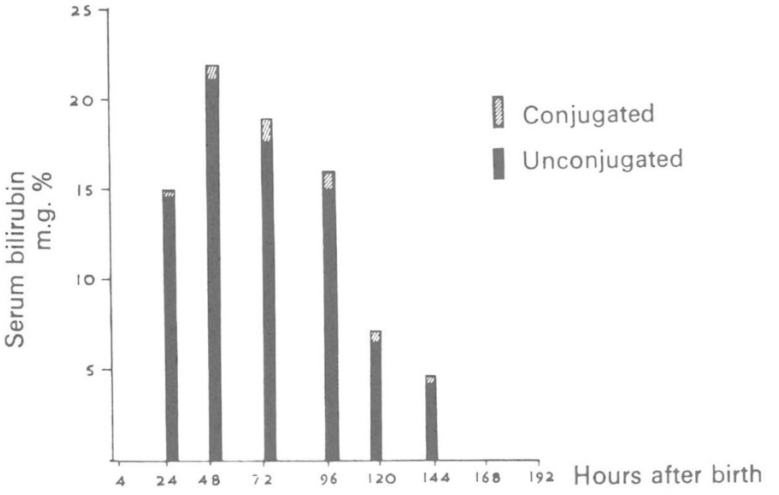


FIG. 12.1 A-B-O incompatibility not needing exchange transfusion

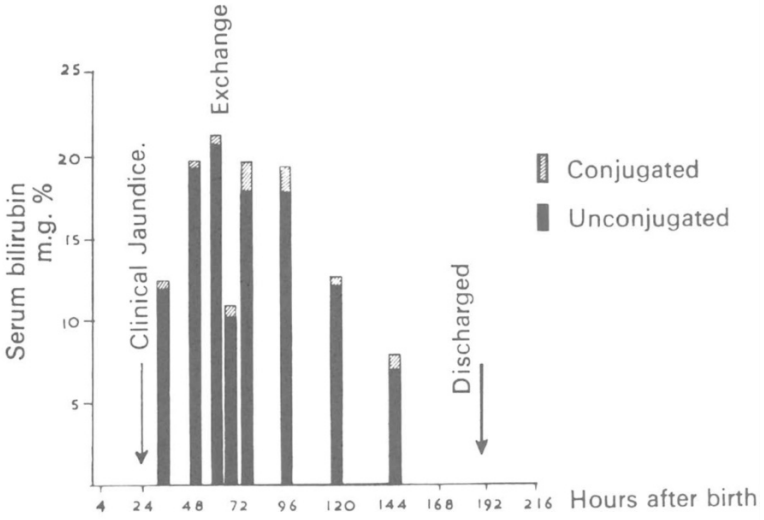


FIG. 12.2 A-B-O incompatibility needing exchange transfusion

(1) *Early appearance of jaundice or rapid accumulation of bilirubin* – Any jaundice that appears in the first 24 hours of life may indicate a haemolytic process. Daily increments of more than 4 mg per 100 ml also indicates haemolysis. In

addition to repeated serum bilirubin estimations on the baby, the following investigations should be done:

(a) Blood groups of the mother and the baby.
(b) Direct and indirect antiglobulin tests on baby's blood.
(c) Reticulocyte count, nucleated red blood cells and spherocytes in the baby.
(d) G-6-P-D estimation in male babies.
(e) Estimation of the haemoglobin and the haematocrit.

(2) *Jaundice which has lasted beyond the first week of life* – This indicates a delayed development of enzymes, e.g. in low birth weight babies and metabolic disorders like hypothyroidism, galactosaemia, anoxia at birth or drugs administered at birth.

(3) *Serum bilirubin levels above 12 mg per 100 ml* – As in (1) above. If all tests are negative, respiratory distress, anoxia or drugs administered at birth should first be ruled out. A-B-O incompatibility produces a milder form of haemolysis as compared to rhesus sensitisation; jaundice may be late in appearing and may be first noticed on the second or third day.[32]

(4) *Conjugated (direct) bilirubin in excess of 2 mg per 100 ml* – Neonatal hepatitis, toxoplasmosis, cytomegalic inclusion disease, sepsis and congential syphilis should be considered. Lack of bile pigments in stools associated with progressively rising direct bilirubin levels in blood indicate biliary atresia.

KERNICTERUS

Unconjugated bilirubin has a toxic effect on the central nervous system. The pigment gets deposited in the neurones where it causes cell damage, and a characteristic clinical picture results. Many affected infants die, and the survivors show mental retardation with other evidence of neurological deficit. The risk of kernicterus rises with bilirubin levels in excess of 20 mg per 100 ml. In the pre-term infant and in those who have suffered from asphyxia, acidosis or hypothermia, kernicterus can occur at a much lower level of bilirubin. Similarly, prolonged starvation of babies and hypoglycaemia increase the risks.

In western countries such high levels of serum bilirubin are seen in cases of rhesus isoimmunisation; they have also reported in Greek, Nigerian and Chinese babies who are deficient in G-6-P-D. Rhesus incompatibility is rare in many of the developing countries of Africa and Asia, but A-B-O incompatibility and G-6-P-D deficiency as causes of hyperbilirubinaemia are occasionally seen. In one study,[33] out of

19,664 consecutive deliveries, 51 babies needed laboratory investigations for hyperbilirubinaemia. In 29 of these, the mothers were of group O and of these, 10 babies required exchange transfusions. Jaundice is late in a majority of the cases; the direct antiglobulin test is negative in a large proportion of babies. Similarly, antenatal diagnosis by examining the mother's blood during pregnancy is not easy and therefore in most instances brain damage in the baby can be avoided only by close observation of all babies of group O mothers for a period of two to three days after delivery. If clinical jaundice becomes evident it should be followed up with serial estimations of serum bilirubin.

MANAGEMENT OF HYPERBILIRUBINAEMIA

When jaundice is due to high levels of unconjugated bilirubin, the treatment consists of one of the following:

(1) Mechanical removal, e.g. exchange transfusion.
(2) Acceleration of the normal metabolic pathway, e.g. use of phenobarbitone.
(3) Use of alternatives for the breakdown of bilirubin, e.g. phototherapy.

Exchange transfusion

In 1948, Diamond described a method of treating babies who exhibit hyperbilirubinaemia following rhesus sensitisation. The principle of treatment is to remove sensitised cells from the baby's circulation and to replace them with normal cells (figure 12.3). In addition, bilirubin gets washed out of the circulation. As serum bilirubin levels fall, more is withdrawn from the tissues and thus toxic effect on the tissue cells is minimised.

There are two main objectives of carrying out an exchange transfusion: (i) to correct anaemia rapidly and (ii) to treat potential or actual hyperbilirubinaemia.

The type of donor blood has to be carefully selected. If the exchange is performed for rhesus sensitisation, group O Rh-negative blood is used. With A-B-O incompatibility, group O Rh-specific blood may be used. Since naturally occurring haemolysins are often present in Group O blood, all such donor blood should first be screened for haemolysins. Fresh blood is preferred to stored blood since the latter may give rise to electrolyte disturbances.

The volume of donor blood to be used at any one exchange is in the range of 150 ml to 200 ml per kg body weight and should not exceed 300 ml/kg body weight. For a baby weighing 2,750 g an exchange trans-

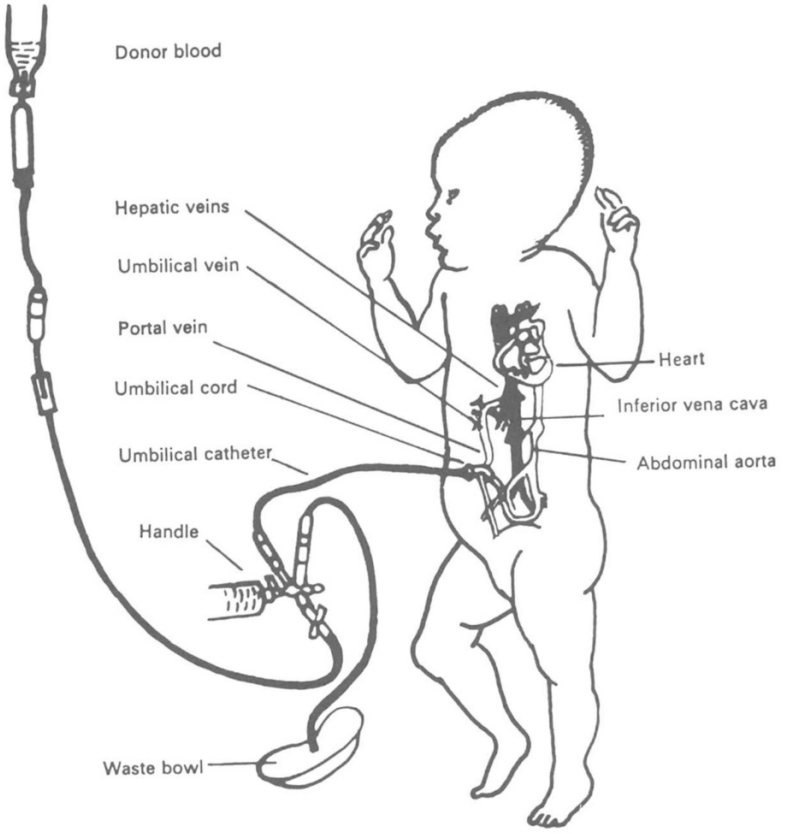


FIG. 12.3 Exchange transfusion

fusion with 500 ml of blood will accomplish a 40 to 50 per cent exchange.

The infant should be well sedated. Stomach contents should be aspirated and a feeding bottle with 5 per cent dextrose should be available as a pacifier if necessary.

Exchange transfusion is a major procedure, the success of which depends upon attention to every little detail. A team of three carries out the procedure and a fourth person keeps a close watch on the baby's condition, respiratory and pulse rate and maintains a record of the volume put 'in' and taken 'out'.

All precautions should be taken to prevent air embolism which can be immediately fatal. The catheter should be introduced only as far as necessary for free flow of blood; if the tip of the catheter rests above the

diaphragm, air may be sucked in with the negative pressure during inspiration. The catheter should be watched for air bubbles and all apparatus should be leak-free.

The other dangers of exchange transfusion are congestive cardiac failure, electrolyte disturbances, sepsis, transfusion reactions from mismatched blood and a spreading thrombophlebitis in the portal tract as a late complication.

Treatment with phenobarbitone

Phenobarbitone is a powerful inducer of hepatic enzymes and has been shown to improve bilirubin conjugation and excretion in experimental animals and in infants. It can also accelerate the metabolism of hormones (especially steroids) and other drugs in the baby. One side-effect of phenobarbitone is its sedative action so that the baby may not suck vigorously or for prolonged periods. Treatment with phenobarbitone has very little to offer in established jaundice because there is a delay of 60 to 84 hours in the onset of its action.

Phototherapy

Phototherapy is now increasingly used in the treatment of jaundice (see figure 12.4). Its use is based on the observation that the bile pigments can absorb light and undergo decomposition, the major part of

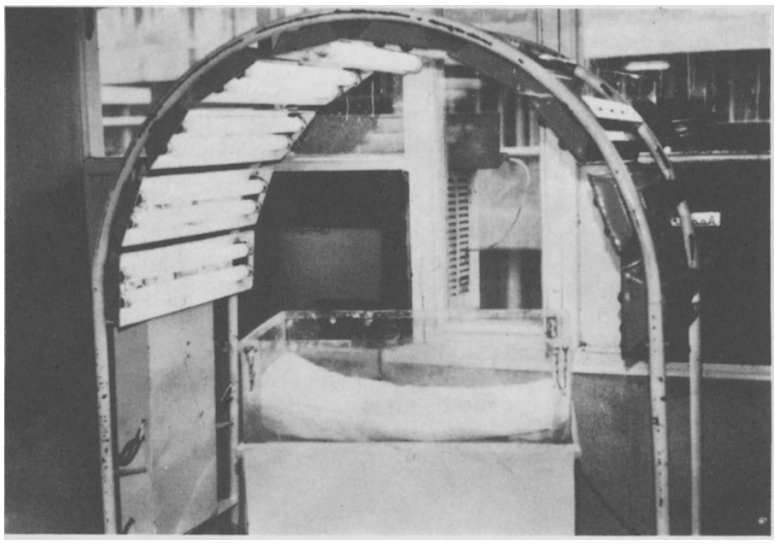


FIG. 12.4 Fluorescent lights for phototherapy

which occurs in the skin. Serum bilirubin levels are not influenced until after 24 hours, hence phototherapy is not very effective in controlling a rapid rise in serum bilirubin as in rhesus isoimmunisation, but it can be of use in A-B-O incompatibility where the rise is more gradual, and as an adjunct to exchange transfusion in rhesus incompatibility. Low birth weight babies who do not have haemolytic disease but are jaundiced with serum bilirubin levels of 10 mg/100 ml, phototherapy for 12 to 24 hours prevents the levels from rising beyond 15 mg/100 ml in 80 per cent of the cases. It is therefore of value as a prophylactic in such infants. Similarly, in infants with G-6-P-D deficiency, early institution of phototherapy will keep serum bilirubin levels low and thus exchange transfusion is avoided.

The types of lights which are most effective in this respect are ones with a rich energy output in the spectral range absorbed by bilirubin, that is, 450 to 460 nm, which falls in the blue end of the light spectrum. Many fluorescent lamps are rich in this characteristic spectrum and have been used for phototherapy. An important observation has been made that an infant in an incubator near a window who is exposed to 6 hours of summer daylight receives approximately 4,700 microwatts/cm^2 at 450 nm in 24 hours, compared with an infant receiving 24 hours of phototherapy (daylight bulbs), who will get 3,800 microwatts/cm^2 in the same wavelength. Thus, in the tropics daylight can be effectively utilised for phototherapy. Ordinary daylight and some of the fluorescent lights also provide significant amounts of ultra violet light which can be hazardous to the infant. A perspex shield helps to absorb the ultraviolet radiation and protects the baby. Other precautions to be taken are as follows-

(1) All diagnostic studies should be accomplished before commencing phototherapy. Suppression of jaundice may obscure or delay the diagnosis of neonatal disease.

(2) In jaundice due to a mild haemolytic process, the response to phototherapy is good, but many such babies go on to develop a late anaemia due to haemolysis. Regular haemoglobin estimation should be carried out in babies in whom exchange transfusion has been successfully avoided.

(3) Overheating and dehydration may be induced in children undergoing phototherapy, so the body temperature should be monitored regularly and additional fluids should be provided during phototherapy.

(4) The eyes should be protected against damage while the baby is being exposed to the light.

JAUNDICE IN THE SECOND WEEK OF LIFE

When jaundice appears for the first time in the second week of life, it needs careful clinical evaluation, since in a large majority of cases its basis is liver pathology. Usually the bilirubin is of the direct type; if both indirect and direct bilirubins are present, the latter is above 2 mg per 100 ml. The commonest cause is liver damage secondary to viral, bacterial or protozoal infections, congenital anomalies of the bile ducts, such as biliary atresia or a choledochal cyst, or a metabolic abnormality like galactosaemia or hypothyroidism.

In neonatal hepatitis, the onset is insidious with low grade pyrexia and progressive jaundice. The liver is enlarged; later the spleen may also be palpable and there may be a moderate degree of anaemia. Serum transminase levels are raised and a biopsy of the liver shows an inflammatory process.

When due to the cytomegalic virus, a centrifuged deposit of freshly voided urine may contain large multinucleate cells with inclusion bodies.

Transplacental infection with *Toxoplasma gondii* may be acquired from a carrier mother. The baby may be stillborn; on the other hand there may occur clinical features resembling haemolytic disease of the newborn, such as anaemia, jaundice and hepatosplenomegaly. Intracranial calcification, demonstrable on x-rays, and chorio-retinitis may be present as late features. Treatment with a combination of sulphadimidine and pyrimethamine is effective.

Septicaemia, portal pyaemia and congenital syphilis should always be considered in the differential diagnosis especially in babies who were delivered at home. Appropriate bacterial culture studies and blood Wasserman's test on the mother will help to establish the diagnosis.

Galactosaemia, though very rare, may be considered in the baby who is irritable, vomits frequently, refuses feeds and becomes icteric late in the first or in the second week of life. Cataracts may appear in the third week. If, in addition, a reducing substance is found on testing the urine with Benedict's solution or clinitest, the diagnosis may be confirmed, though the exact nature of the sugar can only be established by paper chromatography of the urine. Feeding on lactose-free milk will prevent damage to organs like the brain and the liver. Maintaining the baby on a lactose-free diet is extremely difficult, especially in rural areas; one alternative may be to feed the baby on an egg-based solid formula as early as possible, in the meantime maintaining him on lactose-free milk. Sobee, a soya-based formula, is available more freely, but is not preferred because of the presence of several galactosides.

Jaundice is also a prominent sign in other inherited disorders like fructose intolerance, tyrosinaemia and cystic fibrosis.

Chapter 13

Anaemia

The mean cord blood haemoglobin value for the baby in Western Europe is reported to be 16.8 g/100 ml and it has been suggested that any value below 13.6 g/100 ml should be considered as anaemia. For a proper interpretation, the nucleated red cell count and the reticulocyte count should also be taken into account. If, together with a low cord haemoglobin, either of these two values shows a rise it could mean that the bone marrow is attempting to compensate and the low haemoglobin value may then be taken to denote anaemia.

The haemoglobin value of capillary blood in the first few days of life is slightly higher, ranging from 5 per cent to 10 per cent more than the value of cord blood.

In the case of 107 consecutive cord haemoglobins examined at the Ocean Road Hospital, Dar-es-Salaam, 87 were found to be within the range of 12-16 g per 100 ml (see table 13.1).

TABLE 13.1 Cases of low haemoglobin values

Number	*Haemoglobin value g/100 ml*
20	12 – 13
28	13 – 14
13	14 – 15
26	15 – 16

There was only one baby with cord haemoglobin higher than 16 g/100 ml and 12 babies had cord blood haemoglobin values lower than 12 g/100 ml.

The haemoglobin value in the newborn depends on several factors such as time of tying the cord after delivery, duration of gestation, antenatal care and maternal nutrition.

TYING THE CORD

It has been estimated that at birth the blood volume of the infant may

be increased by as much as 61 per cent by allowing complete emptying of the vessels before the cord is clamped. About one quarter of this flow of blood from the placenta to the baby takes place within 15 seconds of birth and one half by the end of the first minute.[33]

GESTATION TIME

Mean haemoglobin concentration of cord blood increases during the last two weeks of gestation and continues to increase in infants born after the 40th week of pregnancy as a result of progressive oxygen lack.[34]

ANTENATAL CARE AND MATERNAL NUTRITION

Little correlation has been noted between maternal anaemia and low haemoglobin values of the cord blood in the baby except in cases where maternal haemoglobin is less than 6 g/100 ml. In one series of severe iron deficiency anaemia in late infancy,[35] it was found that a large majority of mothers (66 per cent) did not receive any antenatal care and were probably iron deficient. However, other factors like birth weight, velocity of growth, iron content of the infant's diet and incidence of malaria and other infections play an important role in the causation of anaemia in late infancy.

The foetus receives his iron from the maternal circulation. The maternal iron is transported by transferrin which is a beta globulin. Iron does not cross the placenta, but is taken up by the placenta and stored as ferritin or haemosiderin. From these depots, the iron is then transported to foetal tissues by the transferrin of the foetus.[36]

CAUSES OF ANAEMIA – (i) IMMEDIATE NEONATAL PERIOD

(1) *Haemolytic disease*
Incompatibility of blood groups in the Rh system is not common in Africans and Asians but A-B-O incompatibility is occasionally seen. Mild cases of haemolytic disease will produce anaemia in the first week of life without any visible icterus.

(2) *Haemorrhage from the placenta or cord*
   (a) Accidental incision of the placenta during caesarean section.
   (b) Placenta praevia and abruptio placentae.
   (c) Tear of the umbilical cord during precipitate delivery.
   (d) Anomalous umbilical vessels.
   (e) Oozing from the umbilical stump if not securely tied.

(3) *Concealed haemorrhage prior to birth*
(a) Foeto-maternal bleed. In about half the pregnancies, some foetal cells may be seen in the maternal circulation. In 8 per cent of pregnanices 0.5 ml to 40 ml of blood is transferred from the foetus to the mother, and in about 1 per cent foetal blood loss exceeds 40 ml.
(b) From one twin to another.

(4) *Internal haemorrhages*
(a) Cephal-haematoma (jaundice may also appear as the products of blood haemolysis are absorbed).
(b) Bleeding into organs (e.g. liver and spleen).
(c) Intracranial haemorrhage.
(d) Haemorrhagic disease of the newborn.

CAUSES OF ANAEMIA – (ii) LATE NEONATAL PERIOD

(1) Generalised sepsis (e.g. staphylococcal or *E. coli* septicaemia).
(2) Pyleonephritis (may be secondary to abnormalities of the urinary tract).
(3) Chronic haemorrhage into the gut (e.g. hiatus hernia).
(4) Delayed anaemia from haemolysis:
(a) Blood group incompatibility.
(b) Drug induced (e.g. vitamin K or naphthalene).
(c) From nitrites in drinking water.

DIFFERENTIAL DIAGNOSIS

The baby with severe anaemia may present the following symptoms:

(a) Pallor as the only clinical sign.
(b) Jaundice and pallor.
(c) Lethargy and/or distress with blood oozing from the cord stump.

In the very pale baby or where there is circulatory distress immediate steps should be taken to replace the blood loss by canulating the umbilical cord. If a plastic catheter or umbilical canula (Portex) is used for this purpose, a sample of blood may also be obtained for laboratory studies. If there is no urgency, the scalp vein or any peripheral vein is preferred for blood transfusion.

For the differential diagnosis, haemolysis as a major cause should be excluded first by means of the following investigations:

(1) Blood group of the mother and the baby.

(2) Coomb's test on baby's blood (negative in the case of A-B-O incompatibility; positive in Rh disease).
(3) A smear from the baby's blood for reticulocyte count, nucleated red cells and spherocytes or similar other rare morphologic abnormalities of the red cells.
(4) In male babies G-6-P-D deficiency may be looked for.

If no evidence of haemolysis is found, the next step is to consider post-haemorrhagic anaemia. A review of the obstetric history should be made:

(1) Maternal bleeding in excessive amounts. Foetal haemorrhage from a tear in the placenta or umbilical vessels may be mistaken for maternal bleeding.
(2) Placenta praevia or delivery by caesarean section.
(3) Twins.

If all these are negative, the mother's blood should be examined for the presence of foetal cells by means of the acid elution technique.[37]

When the above two major causes of anaemia in the newborn have been ruled out, infection as a rare cause of anaemia remains to be considered and appropriate bacteriological studies or urine culture should be carried out. Similarly, hiatus hernia may be the cause of unexplained anaemia due to bleeding into the gastro-intestinal tract. Stool examination for occult blood and x-ray contrast studies will help to establish the diagnosis.

TREATMENT

The treatment depends on the degree of anaemia and distress. In the very ill child, immediate steps should be taken for a blood transfusion. The donor blood is cross-matched with the mother's serum and given at the rate of 4 to 5 drops per minute, the total quantity not to exceed 10 ml/kg in 24 hours.

All babies who are anaemic at birth need iron therapy for four to six weeks whether they have been transfused or not. They should be seen at regular intervals for haemoglobin estimations until the age of about six months, by which time mixed feeding has been introduced in most cases.

Chapter 14

Sepsis

The newborn's ability to cope with infection is variable. Antibodies derived from the mother provide some protection against a variety of illnesses, such as diphtheria, smallpox, measles, chickenpox. On the other hand, the baby has no protection against gram-negative organisms and therefore in any major disease like septicaemia, meningitis, osteomyelitis, etc., infection by these organisms should always be suspected and ruled out first by means of appropriate laboratory studies.

The defence mechanism against infections is poorly developed and so any infection, once established, spreads rapidly and can give rise to septicaemia. Because of this, all kinds of sepsis, however trivial, should be energetically treated.

SOURCE OF INFECTION

Prenatal infection

Such infection may be transplacental or ascending from the genital tract of the mother. Transplacental infections like rubella, cytomegalovirus and syphilis have already been discussed on page 23. In addition, certain rare infections like *Listeria monocytogenes* and *Vibrio foetus* can also occur through this route. All such infections are blood-borne and in the immunologically immature tissues of the foetus will tend to affect several organ systems though there is always a predilection for nervous tissue.

Ascending infection from the genital tract of the mother has been known to spread to the conceptus. There is usually a localised focus of sepsis at the cervical os from where an ascending infection results in the invasion of the amniotic sac. Spread of infection to the foetus is of course more likely when there has been a prolonged rupture of the membranes, but instances are known where ascending infection has occurred in the absence of ruptured membranes. The foetus may swallow the bacteria in the amniotic fluid or aspirate them, resulting in pneumonia. Myocarditis, hepastosplenomegaly and pustular rash of the

skin have been described in infants who have suffered prenatal infections.

Home deliveries

The commonest infection is cord sepsis which may appear as 'smelly cord', omphalitis, septicaemia and in some instances as tetanus neonatorum. The other common forms of infection in babies delivered at home are skin sepsis and repiratory infections.

Babies who are delivered at home and later brought to hospital should be considered as 'infected' and should receive:

(1) Immediate cord care – in the form of five per cent proflavine in spirit, application of triple dye or antibiotic ointment (e.g. Neomycin-Bacitracin) and ligating the cord with proper cord ligatures.
(2) Tetanus antitoxin in a prophylcatic dose of 750 units.
(3) Parenteral antibiotics in the form of a combination of Inj. crystalline penicillin (250,000 units 12 hourly) and Inj. Streptomycin (50 mg daily for three days), if more than 24 hours old.
(4) Sponging with Savlon solution (1 in 200 strength).

Such a baby should be isolated in a separate room away from the ordinary nursery population for a period of three days.

Hospital deliveries

Overcrowding in labour wards and maternity hospitals increases the chances of infection in the baby. It has been the experience of many centres that the peaks of neonatal sepsis coincide with periods of increased admissions or lack of proper staffing in the nursery. Moreover, because of pressure on beds, many mothers are discharged within a day or two of delivery. Under such circumstances the baby may be incubating an illness which may not manifest itself until he reaches home, and thus may escape notice. In such a manner, the maternity hospital may indirectly become a source of sepsis in the community.

In hospital deliveries, the sources of infection to the baby are:

(1) *From the mother*
(a) Blood-borne (e.g. mothers who are running pyrexia shortly before delivery).
(b) During passage through the birth canal (e.g. from infected vaginal secretions).
(c) Ascending infection through the genital tract, e.g. where membranes have been ruptured for more than 24 hours. In

such a case, and also where the liquor is 'smelly', a vaginal swab for culture should be taken in the delivery room, so that if the baby shows any signs of infection later, the appropriate antibiotic can be given.

(2) *In the labour room*

(a) If aseptic precautions are faulty, skin and cord sepsis may occur; in overcrowded labour rooms with insufficient staff this possibility may arise.

(b) In babies who need resuscitation at birth, mouth-to-mouth breathing is a source of infection. As a routine all babies who need such resuscitative measures as endotracheal intubation, mouth-to-mouth or mouth-to-tube breathing, should be given Injection Streptomycin (50 mg for three days).

(3) *In the nursery*

Infection may be introduced by visitors, mothers who come to feed their babies, or the attending doctors and nurses. Scrupulous washing of hands in between handling babies is essential. Infected dust from blankets and linen has been known to cause outbreaks of sepsis in the nursery; all linen from the nursery should be laundered and handled separately from that of the rest of the hospital. The common bath-tub is dangerous; it is best to avoid the routine of a daily bath and instead to use Savlon or Phisohex solution for cleansing the skin.[39]

Catheters and equipment used for resuscitation, administering oxygen and for humidification can easily get contaminated, especially with pseudomonas, and give rise to sepsis in the labour room or the intensive care unit. All such equipment should be regularly sterilised. Wolff bottles and tubing connected with a source of oxygen should be autoclaved or boiled daily.

In many nurseries a common source of infection is the feeding bottle and infected milk formulae. The resulting diarrhoea is due to pathogenic *E. coli* and is usually severe with a high mortality rate. Moreover, it invariably spreads through the nursery causing epidemics which are difficult to control. Hence it is always wise to insist on breast feeding and to put a ban on all artificial feeding. More recently, several nurseries have experienced an increasing incidence of infection by group B beta haemolytic streptococci. The infected baby may show infection of the lung causing respiratory distress which may be mistaken for the idiopathic respiratory distress syndrome.

CAUSATIVE ORGANISMS

Gram negative organisms are responsible for a variety of infections in the newborn, and should always be suspected in the first instance while laboratory studies are in progress. *E coli* not only produce urinary infections but can also give rise to septicaemia, meningitis and gastro-enteritis in the newborn.

In Western countries patterns of nursery infections have been changing over the years. In the past, the most common organism isolated was *E. coli;* this has been largely replaced by *Staphylococcus aureus* in recent years. It may cause any of the following:

(1) Outbreaks of severe sepsis in the nursery population.
(2) Minor infections like boils, conjunctivitis, umbilical sepsis.
(3) Symptomless colonisation of infants.

There are indications that *Ps. pyocyaneus* may replace *Staphylococcus* as the common infecting organism in the nursery. This group of organisms has the tendency to grow in oxygen administering apparatus, in tubings connected with oxygen cylinders and on the inner sides of containers of antiseptic solution.

DIAGNOSIS

Because of the newborn's inability to localise sepsis, early diagnosis and intelligent use of antibiotics is essential. All superficial infections like boils, umbilical sepsis, etc., should be given prompt attention and appropriate specimens should be taken for bacterial culture so that if infection does become generalised, treatment with the correct antibiotics can be available.

Since the meninges are commonly involved, a lumbar puncture is always indicated in sick infants. Organisms are often present before the appearance of a large number of white cells in the spinal fluid, so a centrifuged specimen must be examined under the microscope after gram staining.

The symptoms of generalised sepsis in the newborn are nonspecific, e.g. vomiting, lethargy, refusal to feed and sudden fluctuations of temperature. Loss of weight in a baby who has previously been doing well should arouse suspicion. Anaemia, jaundice, and an undue tendency for bleeding indicate massive sepsis.

The baby has an anxious staring look, or an ashen grey colour. There may be undue lethargy or irritability. Enlargement of the liver and spleen or of the kidneys are important clinical signs.

LOCAL SEPSIS

(1) *Eye* – Any purulent discharge within the first 48 hours of

birth should be assumed to be gonococcal. A swab should be taken for culture and freshly prepared penicillin eye drops (2,000 units/ml) should be instilled hourly for six hours, followed by chloramphenicol eye ointment.

A sticky eye with a purulent discharge and inflammation of the surrounding tissues occurring 48 hours after birth is due to infection acquired in the nursery and usually due to staphylococcus or in some instances to *E. coli.* If the sensitivity of the common staphylococcal infections in the nursery is known, the appropriate antibiotic should be used; otherwise, pending laboratory investigation, treatment should be started with chloramphenicol eye drops locally and parenteral crystalline penicillin (250,000 units 12 hourly) together with streptomycin (50 mg daily) for three days.

(2) *Skin or umbilicus infection* – After taking a swab local application of a combination of Neomycin and Bacitracin should be made twice daily. If there is inflammation around the umbilical stump or if there are signs of a generalised illness, systemic antibiotics are indicated as above.

SYSTEMIC INFECTIONS

(1) In chest infections or in cases of suspected septicaemia the proper antibiotics to start with are a combination of penicillin and streptomycin. Depending upon sensitivity results this combination can be modified or changed afterwards (see also Appendix I). Laboratory studies are important because of a close co-relation between eventual outcome and a positive culture. Material for bacterial culture can be obtained from any one or more of the following:

blood
cerebro-spinal fluid
urine
umbilical swab
swabs from the nose and throat.

Certain categories of infants are more prone to infection and a high degree of suspicion and watchfulness is necessary when caring for them. These are:

(a) Babies of low birth weight.
(b) Those with congenital anomalies.
(c) Those born after prolonged rupture of the membranes or after protracted and difficult labour.

(d) Babies who are suffering from any serious illness not necessarily of infective origin (e.g. respiratory distress).
(e) Those who have tubes and canulae inserted anywhere.
(f) Those who have been connected to any apparatus containing moisture.

(2) If there is an additional localised lesion suggesting that the sepsis is staphylococcal in origin, cloxacillin (100 mg/kg/day in four divided doses) may be substituted for penicillin.
(3) In proven meningeal infections the treatment should begin with a combination of streptomycin (50 mg/kg/day), chloramphenicol (50 mg/kg/day) and sulphadiazine (90 mg/kg/day) until results of culture on the cerebro-spinal fluid are available.
(4) About 1 per cent of newborns have bacteriuria as shown by urine culture. Many are asymptomatic and the urine will clear without any treatment. Those with clinical symptoms usually show a vague illness with loss of weight, vomiting, lethargy and pallor, and only rarely a severe systemic illness. Thus, the clinical features of neonatal urinary infection may be classified into four groups:

(a) A severe systemic illness, with clinical evidence of pyelonephritis and septicaemia.
(b) Infection secondary to a major congenital abnormality of the urinary tract.
(c) Mild non-specific symptoms with no signs pointing to the renal tract.
(d) Asymptomatic infants in whom infection of the urine is a chance finding.

Because of such a wide clinical spectrum it is important to suspect urinary infection in any newborn who is not doing well and urine culture should be carried out as part of routine investigations. It is not always easy to obtain uncontaminated urine from infants. Suprapubic puncture is a safe technique and is commonly used to obtain a sample of urine for examination.

PROPHYLACTIC ANTIBIOTIC TREATMENT

Treatment with antibiotics as a prophylaxis should be considered in the following situations:

(1) Membranes ruptured for twenty-four hours or more before delivery.
(2) Maternal infection or infected intra-uterine environment at the time of delivery (e.g. cystitis, pyelonephritis maternal fever, cervicitis and foul smelling liquor).

(3) Infants needing mouth-to-mouth respiration and/or endotracheal intubation at birth.
(4) Infants with respiratory distress especially if deteriorating or if moist sounds develop in the lungs.
(5) Exchange transfusion. A swab should be taken for culture from the umbilical stump before and after transfusion.
(6) Local sepsis e.g. profusely discharging eye especially if there is periorbital oedema, discharging umbilicus with presence of local induration or skin sepsis.

Chapter 15

Hypoglycaemia

During intrauterine life the foetal tissues receive a constant supply of glucose from maternal blood through the placenta; after birth and until feeding is established there is an interval during which the newborn has to depend upon his own reserves of carbohydrate for his glucose requirements. These stores have been laid down during the latter part of gestation in the form of glycogen in the liver, in skeletal muscle and in the cardiac muscle. Of these, the most important is liver glycogen, since it is available for conversion to glucose and subsequent metabolism for the production of energy.

In the first 24 hours after birth rapid depletion of liver glycogen takes place. It is more marked in babies who have suffered from hypoxia at birth. This is because glycolysis occurs along the anaerobic pathway during anoxia, and uses up more glycogen for the production of the same amount of ATP as compared with aerobic utilisation of glucose.

At the time of birth the blood glucose of the newborn is proportionate to that of the mother, and values of blood sugar in the cord blood are 70 to 80 per cent those of maternal venous blood. After birth the blood sugar level falls rapidly, reaching the lowest in about three hours and then rises again a little, after which it is stabilised at that level. Thus, the infant uses up carbohydrate reserves within hours of birth after which his main energy source switches from carbohydrates to lipids. Hypoglycaemia is therefore more likely to be due to deficient gluconeogenesis in the liver or due to lack of delivery of substrate in the form of lipids and amino-acids to the liver, or a combination of both. If in a full-term baby any two consecutive blood sugar values are less than 30 mg/100 ml in the first 72 hours of life, or less than 40 mg/100 ml after the third day, the baby should be considered to be suffering from hypoglycaemia. In the low birth weight baby, the average blood sugar values tend to be lower still and therefore 20 mg/100 ml is taken as the lower limit of normal.

Besides hypoxia and low birth weight (especially if small for dates)

hypoglycaemia may occur in other clinical states like severe sepsis, respiratory distress syndrome, maternal toxaemia, cerebral birth injury, kernicterus, etc., and must be treated in addition to the management of the underlying disease. On the other hand, if hypoglycaemia in a baby does not respond to treatment, a search should be made for underlying metabolic or endocrinologic causes.

METHODS OF ESTIMATING BLOOD SUGAR

(1) The 'Dextrostix' is a reliable and easily available method. In any average sized nursery, all members of the staff should be conversant with this method of blood sugar estimation.
(2) In all other methods requiring collection and transport of blood to a laboratory, the container should have sodium fluoride added to the anticoagulant. Red blood cells of the newborn utilise glucose rapidly and therefore a sample which has been stored for some time may show erroneous results due to this rapid glycolysis. Fluoride helps to block glycolysis.

SYMPTOMS

In uncomplicated hypoglycaemia of the newborn, symptoms are not common in the first 24 hours; they usually develop between the first and the third days after birth. Early institution of feeding is a good prophylactic; breast milk is best in this respect as it contains 67 calories /100 ml compared with 40 calories/100 ml in 10 per cent dextrose.

Symptoms, when they occur, are non-specific and consist of convulsions, cyanotic or apnoeic attacks, irritability, lethargy, hypothermia and poor feeding. More commonly, the infant is found to react excessively to sound and touch and is described as 'jittery'. Abnormal behaviour in a newborn may be due to hypoglycaemia and blood sugar estimations should be carried out in all babies whose reactions are unusual or if they show any of the above symptoms.

TREATMENT

The criteria for diagnosis are two consecutive blood sugar values of less than 30 mg/100 ml in a baby of normal birth weight or 20 mg/100 ml in a low birth weight baby. The so-called 'small for dates' baby should always be put under close observation.

When the diagnosis is confirmed, therapy should be started immediately with 50 per cent glucose, 1 to 2 ml/kg given intravenously followed by a drip of 15 per cent glucose in water or in N/5 saline at the rate of 75 to 100 cc/kg in 24 hours.

Oral feeding should begin as soon as possible.

If symptoms persist or the blood glucose level remains below 40 mg/100 ml after 12 hours of intravenous glucose, hydrocortisone in a dose of 5 mg/kg/day should be added.

As recovery occurs, the concentration of glucose in the intravenous fluid should be gradually reduced. If it is stopped abruptly, reactive hypoglycaemia may occur.

Chapter 16

Minor Problems

Many of the common problems seen in the nursery could be termed 'minor' because they are either self-limiting or respond to simple therapy. Some of them may be due to the stresses and pressures of the birth process, and others may be acquired in the nursery.

PROBLEMS DUE TO BIRTH TRAUMA

Caput succeedaneum – This is the name given to a collection of subcutaneous fluid at the presenting part. It is usually absorbed in 26 to 36 hours.

Cephal-haematoma – This is caused by bleeding under the periosteum of one of the bones of the skull. It therefore takes up the shape and outline of the bone over which it has formed. It may persist for several weeks before being finally absorbed and may become a source of parental anxiety. Any attempt at aspiration of the contents may result in the introduction of infection and should be avoided. Very large haematomas may give rise to anaemia and rarely jaundice due to absorption of the products of haemolysis of the blood clot.

Depressed fracture of the skullbone – This is usually due to pressure of the sacral promontory of the mother on the foetal skull. In the vast majority of cases it needs no surgical treatment, and the depression gradually becomes less as brain growth occurs.

Excessive moulding – When moulding has been excessive the baby should be disturbed as little as possible. Sedation with chloral hydrate (60 mg twice daily) may be required. Because of the latent haemorrhagic tendency in all newborns such babies should be given Inj. vitamin K 2.5 mg at birth.

Excessive moulding may be associated with underlying tentorial tears. When such tears occur, bleeding takes place from one of the venous sinuses or from the vein of Galen. In most cases, such a haemorrhage is subdural. When it tracks beneath the tentorium the vital centres of the medulla are compressed, and death results.

Clinically, such a baby is lethargic with irregular respiration, grey or cyanosed and unable to suck. On the other hand there may be signs of hyperirritability, twitchings or a high-pitched cerebral cry.

When a subdural haemorrhage is suspected, a diagnostic tap will confirm it. Repeated taps may be necessary to evacuate the subdural collection of fluid.

Fractures – The commonest fractures are those involving the clavicle and the humerus. A unilateral Moro reflex points to the affected side.

Treatment consists of strapping the arm on the affected side to the chest wall.

Nerve palsies – Facial nerve palsies is usually due to pressure on the facial nerve during a forceps delivery. It is apparent when the baby cries. Spontaneous recovery is the rule. In rare cases when recovery does not occur, congenital absence of the facial nerve nucleus may be suspected.

Erb's palsy – The affected arm lies limp by the side of the baby and does not move when a Moro reflex is elicited. The arm can be put in a splint in a position of abduction with the elbow flexed and the forearm supinated. While the splint is being arranged the sleeve of the affected arm can be pinned to the bed clothes alongside the baby's head to keep the arm in position.

It is now suggested by some that splinting may be harmful and can give rise to contractures. Many cases of Erb's palsy heal spontaneously and the best form of treatment is to put the affected limb through a full range of passive movements several times a day while awaiting recovery.

Sternomasoid tumour – This may be noticed on the second or third day after birth as a painless firm swelling in the body of the sternomastoid muscle in its lower half. The mother should be advised on daily massage of the muscle and passive movements of the head in its full range to avoid the late sequela of torticollis.

MINOR PROBLEMS OF THE BABY'S SKIN

Seborrhoea of the scalp – This is also called cradle cap. It occurs in the form of thick crusts covering the scalp. If the scalp is dry, the crusts should be touched daily with cotton wool soaked in olive oil. A weekly shampoo with cetavlon *pro capitis* is helpful in clearing the scalp.

Napkin rash – This is rather rare in African babies. It occurs as a painful dermatitis affecting the napkin area and is due to chemical irritation of the skin by ammonia from decomposing urine.

The affected part should be kept exposed to air and dry. Local application of zinc and castor oil cream may be helpful.

If the dermatitis is severe, the baby should be placed in a bath of

weak potassium permanganate solution for a few minutes twice daily, followed by application of local corticosteroid. Resistant cases may be due to superadded fungus infection and respond well to Dequadin ointment. (Prolonged or repeated use of Dequadin may cause skin necrosis.)

Wet umbilicus – This should be cleaned several times in the day with spirit of flavine in spirit. If there are obvious signs of infection, a local antibiotic ointment may be applied.

Thrush –Candida albicans infection of the oral mucosa may cause painful deglutition and interfere with feeding. It is seen as thick white crusts on the tongue and mucus membrane of the mouth.

Most cases respond well to daily application of gentian violet solution. Resistant cases may need Nystatin drops.

Scaling or peeling of the skin – This can be quite normal when it occurs after two or three days. If present at birth, it usually indicates intrauterine malnutrition or excessive prolongation of pregnancy.

Neonatal breast engorgement – This is common in both boys and girls. It is due to the effect of maternal hormones in the baby's bloodstream and will subside spontaneously in most cases. Squeezing the engorged breasts to express secretions may introduce infections and cause mastitis.

ABNORMALITIES OF THE GENITALIA

Undescended testes – In about 97 per cent of all full-term babies the testes are either completely descended or can be easily drawn down as far as the upper part of the scrotum. Most undescended testes will descend in the first few weeks. If they have not descended by the age of one year, later descent is unlikely and surgery is necessary.

Vaginal discharge – All female infants have a mucoid vaginal discharge. In some the sudden withdrawal of maternal hormone causes vaginal bleeding. No treatment is required, and the bleeding stops spontaneously.

ORTHOPAEDIC PROBLEMS

Talipes equinovarus – The characteristic deformity has four elements:

(1) Equinus (the foot is plantar flexed).
(2) Varus (there is inversion of the heel with adduction and inversion of the forefoot).
(3) A tendency for the forepart of the foot to dislocate medially.
(4) Internal torsion of the tibia.

Prognosis depends on how early the treatment was begun, and therefore manipulation should begin at birth or soon thereafter (see figure 16.1).

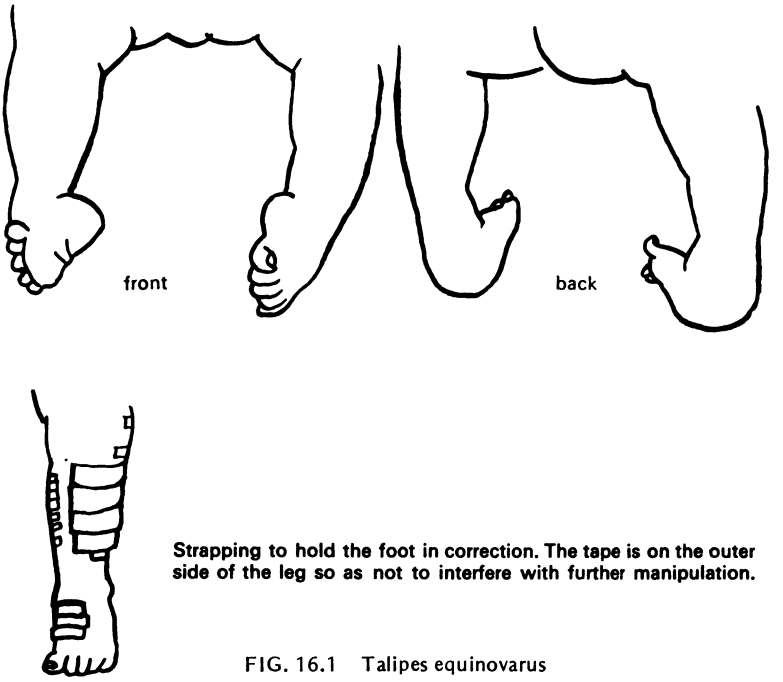


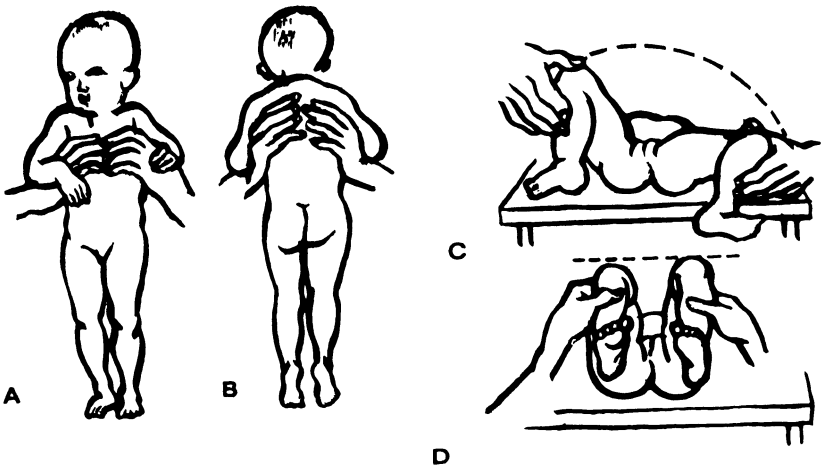
FIG. 16.1 Talipes equinovarus

The baby's foot is manipulated out of forefoot adduction, heel and forefoot inversion and equinus. Adhesive tape is applied to hold the foot in correction. The tape should be only on the outer side of the ankle and leg so as not to interfere with further manipulations. Several manipulations a day at regular intervals are necessary for three to six weeks.

Genu recurvatum – The affected knee should be flexed to the degree that is possible and fixed in this position by adhesive tape. Every day the degree of flexion can be gradually increased until proper flexion is possible.

Congenital dislocation of the hip – A quick screening test consists of looking for limitation of passive abduction of the hip in a position of 90 degree flexion. In the normal newborn, with the knees fully flexed and hip joints flexed to 90 degrees, passive abduction is possible until the knee touches the table. If there is dislocation of the hip there will be resistance to abduction. In some cases, while the test is being per-

formed one can hear a click as the femoral head slides in across the posterior of the acetabulum and enters the socket. The diagnosis can be confirmed by the presence of specific x-ray signs (see figure 16.2).



A. Apparent shortening of the (R) leg
B. Asymmetry of the thigh folds and popliteal and gluteal creases
C. Limited abduction of (R) hip
D. Apparent shortening of (R) Femur

FIG. 16.2 Dislocation of the hip

On diagnosis, the femoral head is manipulated into the acetabular socket by flexing the hip to 90 degrees and then adbucting it in flexion, all the while applying pressure with the fingertips on the greater trochanter. It is then maintained in this position by using special splints.

Alternatively, the baby should be nursed on the abdomen with the hip joints abducted in 90 degree flexion; this position can be maintained by pinning the napkin to the bedclothes. The hip has to be kept in this position for a period of two to three months depending upon how early the diagnosis was made.

The incidence of congenital dislocation of the hip varies from one racial group to another, and is higher in the newborn than in the older infant which means that many cases recover spontaneously. It is estimated that of every four cases of dislocation of the hip picked up in the neonatal period, three will recover without treatment. However, all newborns so diagnosed should be treated since it is not possible to find out which cases will correct spontaneously. Sibs and close relations of known cases should be checked with care since they have 25 times the risk of the condition as compared with the general population.

Chapter 17

Congenital Physical Defects

The diagnosis and treatment of congenital defects continues to be a major problem in neonatal paediatrics. Although the infant and perinatal mortality rates have shown appreciable reductions since the beginning of the century, the death rate of babies with congenital defects has undergone no significant change. It is estimated in the U.K. that congenital malformations account for one in five of all infant deaths. Corresponding figures for the developing countries will differ since traditional midwifery, infection in early life, poor sanitation and overcrowding in homes take a heavy toll of infant life in these parts of the world.

In the British perinatal mortality survey[6] it was found that of 2,188 perinatal deaths, 415 occurred in babies with congenital defects. In more than half of these the malformation involved the central nervous system. Besides early death in the immediate neonatal period, a congenital defect may lead to repeated illness and death in early childhood as in the case of congenital heart disease, or cause a chronic handicap as in the case of meningocele with involvement of nerves. It has been estimated that of all babies with a congenital malformation who are alive at four weeks about 80 per cent will survive.

The incidence and pattern of congenital malformations vary from one population group to another depending upon a combination of genetic and environmental factors. One of the commonest congenital defects seen in African babies is polydactyly; malformations involving the neural tube are frequent, and anomalies of the gastrointestinal tract together with other miscellaneous conditions like hare lip, cleft palate, talipes, etc. bring up the list of the common malformations seen in a neonatal unit.[38]

AETIOLOGICAL FACTORS

In animal experiments a variety of factors including nutritional deficiencies have been found to cause congenital malformations; yet very

little has been demonstrated which can have direct bearing on human reproduction. Two important environmental agents – drugs administered in early pregnancy and maternal infections – have already been mentioned on page 22. As regards drugs, it should be remembered that it is a common practice in many communities to administer indigenous potions and herbs to the mother in various stages of pregnancy. The presence of alkaloids and other chemicals in such medications could conceivably cause harmful effects on the developing embryo in the same way as some pharmaceutical agents do.

The association of maternal rubella with congenital defects involving various organ systems has been well established. During an epidemic of rubella in the United States, the effects of the infection on the embryo have been studied in more detail and the extended rubella syndrome has been described.[39] Clinical observations and epidemiologic studies have also helped to define the effects of other infections like the cytomegalovirus, varicella and toxoplasma. Vaccination against rubella is now common in many western countries, and as the importance of the other infections becomes established vaccines for protection against them will no doubt be developed. Thus it is obvious that one important step in the prevention of congenital defects from the above factors is regular health supervision and prenatal care of the pregnant woman.

GENETIC FACTORS

These may be (1) chromosomal aberrations, (2) single gene effects which may be dominant, recessive or sex-linked, and (3) polygenic.

1. *Chromosomal aberrations*

Most such aberrations end in abortions and miscarriages, hence the incidence of chromosomal abnormality as a cause of malformation is low at 1.5/100 births. The most common abnormality is the Down's syndrome (mongolism) which has an incidence of 1 in 600 live births. It is caused by the presence of an extra chromosome so that the affected individual carries 47 chromosomes (instead of 46), the additional chromosome being attached to the chromosome pair number 21 – hence the term 'trisomy 21 syndrome'. Similar trisomies have been described in relation to chromosome pairs 15 and 18 (D and E trisomy syndromes).

Chromosomal aberrations can also involve the sex chromosomes, such as the Turner syndrome (1 in 4,000 female live births) in which the individual has only one X chromosome and a total of 45 chromosomes. On the other hand the Klinefelter syndrome (1 in 1,000 male live births) is characterised by two or more X chromosomes in addition to one Y.

Rarely, chromosomal abnormalities are due to a portion of one chromosome pair becoming attached to the wrong pair during cell-division, giving rise to translocation. If such portions get lost in mitosis, there is partial deletion.

In cases of Turner's and Klinefelter's syndromes the risk of recurrence in later sibs is low. Down's syndrome is often seen when pregnancy has occurred after the age of 40. The risk of recurrence following the birth of one affected child is about 1 in 50, as compared with 1 in 600 for the general population. If a young mother happens to give birth to a child with Down's syndrome, it is highly likely that she has a translocation, in which case the risks of having a second affected child are greatly increased.

In all chromosomal aberrations, the development of several organs is faulty so that the infant has multiple defects. In the same aberrations, identical organs are involved with similar abnormalities causing syndromes.

2. *Single gene effect*

(a) Dominant inheritance is caused by genes which can produce their effects in the heterozygote, that is, the affected person inherits the condition from one parent. The parent will have the same abnormality extending backwards through several generations. It is, however, possible for abnormal genes to arise by fresh mutation so that in some cases an affected child may be born to normal parents. On the other hand, an affected person married to a normal spouse will have affected and normal children in equal proportions, and the risk of abnormality for each birth may be described as 1 in 2. Examples of dominant inheritance are: achondroplasia, osteogenesis imperfecta, multiple neurofibromatosis, multiple telangiectasia, etc.

(b) Recessive inheritance is due to genes which can produce their effects in the homozygote only, that is, the affected person must receive the abnormal gene from both parents. Each parent is thus a carrier, though normal in appearance. Such a carrier state is detected usually after the birth of the abnormal child. For each subsequent birth, the risks of recurrence are 1 in 4. Since close family relatives tend to have similar genetic patterns, consanguinity is an important contributory factor in the aetiology of abnormalities which are due to inheritance of recessive genes. First cousin marriages are common in many developing countries. Similarly, in several village societies endogamy is commonly practised so that the chances of recessive inheritance are greatly increased. In a world-wide WHO study of congenital malformations, a significant association between cousin marriages and the incidence of neural tube defects was found.[40]

Sickle-cell anaemia and thalassaemia are examples of recessive in-

heritance. Many of the inborn errors of metabolism like phenylketonuria, galactosaemia, etc., are also inherited on a recessive basis.
(c) Sex-linked inheritance – The sex chromosomes carry several genes other than those determining the sex of the individual. Since the X chromosome is the larger of the two sex chromosomes (X and Y), the genes carried on it are dominant from the point of view of inheritance in the male. On the other hand, most of the genes on the X chromosome are recessive, so that a woman with an abnormal gene on one of her two X chromosomes will not manifest the abnormality, but her sons will. The risk to the sons of developing the abnormality is 1 in 2; there is a similar risk of 1 in 2 for the daughters becoming carriers. Haemophilia, G-6-P-D deficiency, Duchenne-type muscular dystrophy and nephrogenic diabetes insipidus are some examples of sex-linked inheritance.

3. *Polygenic*

For many common defects where the malformation is single, such as congenital dislocation of the hip, cleft lip and palate, and pyloric stenosis, the incidence in close relatives of the index patient tends to be higher than in the general population, yet no clear-cut mode of inheritance can be made out as in the case of single gene effects described under (2) above. This indicates that such malformations are influenced by several genes. Brothers, sisters and offspring (first degree relatives) are affected 30 to 50 times more often than the general population. The incidence in nephews, nieces and cousins (second and third degree relatives) is lower but still significant at 5 to 7 times that in the general population. Parents who have a malformed child stand a slightly increased risk of another offspring being similarly affected – the frequency of malformation in such offspring being ten times that in the general population. When a couple have two children with the same malformation, the risks to subsequent offspring are much higher and limitation of family on genetic grounds is advisable.

POSTURAL DEFORMITIES

These mostly involve the musculo-skeletal system and are thought to arise from mechanical factors operating during intrauterine life. Talipes, postural scoliosis, torticollis, facial deformities and deformities of the skull are examples of such malformations. Epidemiologic studies have helped to identify several associated factors. For example, such deformities are common in breech presentation, first pregnancy, oligohydramnios, maternal hypertension and growth retardation of the foetus.

GENETIC POLYMORPHISM

It has been postulated that environmental influences may give rise to perpetuation of an abnormal gene (figure 17.1). There is a close correlation between malaria endemicity and prevalence of the sickle-cell and thalassaemia genes in many parts of the world.

In the descriptions that follow, the common congenital malformations are arbitrarily classified into several groups. The classification is based on the effect of the congenital defect on the newborn and the urgency with which diagnosis and/or treatment may be required.

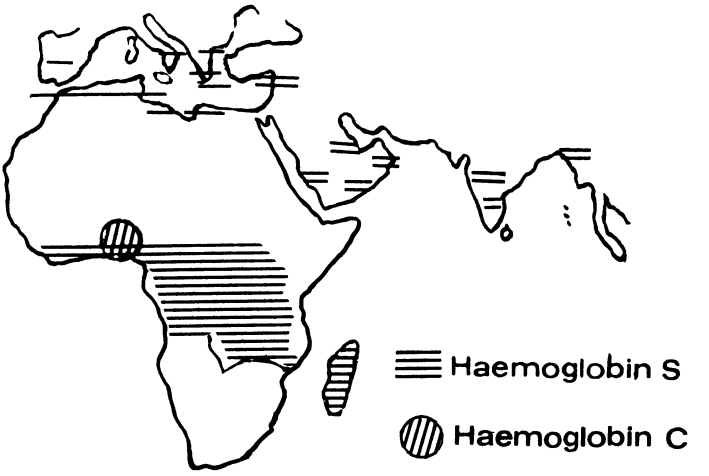


FIG. 17.1 Areas of prevalence of haemoglobin S and haemoglobin C

MALFORMATIONS NOT COMPATIBLE WITH SURVIVAL

Anencephaly – The vault of the skull is not developed and the brain tissue may be completely lacking or protruding with a cover of meninges.

Cyclops and other forms of 'monsters' do not survive.

Hydrocephalus – If the head is large at birth the outlook is worse than when the head begins to enlarge in the early months of life. Many cases are due to congenital abnormalities causing obstruction to the flow of the cerebro-spinal fluid; some are due to haemorrhages at the time of birth or meningitis in the perinatal period, both of which cause a block in the cerebro-spinal pathways. Many of these babies die in early childhood. In one follow-up of untreated cases it was found that about half arrested spontaneously though only half of the survivors showed normal intelligence.[41]

Ectopic bladder – The anterior abdominal wall is not formed and the bladder and other related structure are exposed on the abdominal wall.

MALFORMATIONS NEEDING EARLY SURGERY

Obstructions in the gastrointestinal tract – When any of the following symptoms are present, anomalies of the gastrointestinal tract should be suspected and prompt action taken to confirm the diagnosis:

(1) Frothing at the mouth or excessive pharyngeal mucus.
(2) Attacks of cyanosis or dyspnoea.
(3) Persistent vomiting especially if bile-stained.
(4) Failure to pass meconium.

The common conditions causing obstruction in the gastrointestinal tract in the neonatal period are:

Hirschsprung's disease – The usual cause is an aganglionic segment of the bowel extending from the anus to the lower sigmoid colon causing a functional obstruction. Extreme distension of the abdomen which can, at times, even embarrass respiration is the usual feature. Rectal examination results in a bowel movement and deflation. After a few days the constipation and abdominal distension builds up again. Water enemas are dangerous and may cause water intoxication. Diagnosis can be confirmed by a barium enema which shows dilated colon or by means of a rectal biopsy. On suspicion the baby should be transferred to a centre where surgical help is available.

Meconium plug – The baby shows all the signs and symptoms of intestinal obstruction which are relieved after the passage of a thick meconium plug either spontaneously or after a rectal examination. Meconium ileus is the term applied to obstruction caused by inspissation of abnormal mucus in babies who have mucoviscidosis (fibrocystic disease of the pancreas).

Atresias of the bowel – These may be multiple and surgical treatment may turn out to be difficult because of the extensive length of the bowel that would need resection. Distension of the abdomen occurs late, and in the case of high obstruction, there may even be some evacuation of bowel contents. If the faeces passed are pale and mucoid, obstruction should be considered. Vomiting, early and persistent, is an important sign and if bile-stained should lead to prompt x-rays even though few other clinical signs are present. Babies with high obstruction appear deceptively well and suddenly collapse on the fourth or fifth day of life.

Ano-rectal anomalies – These are usually visible and do not cause diagnostic difficulties. In most cases an orifice is present – in the perineum, in the male urethra, or on the vulva; hence flatus may be passed

and there can be meconium-staining of the napkin. This may, at times, lead to a delay in diagnosis. An x-ray of the baby in the inverted position with a metal strip on the perineum will indicate the distance between the bowel and the perineum (figures 17.2, 17.3, 17.4).

In two kinds of anomalies – a covered anus where a thin membrane covers the anal area, and the ectopic anus – the obstruction can be relieved by minor surgery. This should be followed by regular finger dilatation to avoid stenosis. In the third kind, rectal agenesis, where the bowel stays above the pelvic floor, major surgery is needed. This anomaly may also be associated with fistulous connections to the genito-urinary tract.

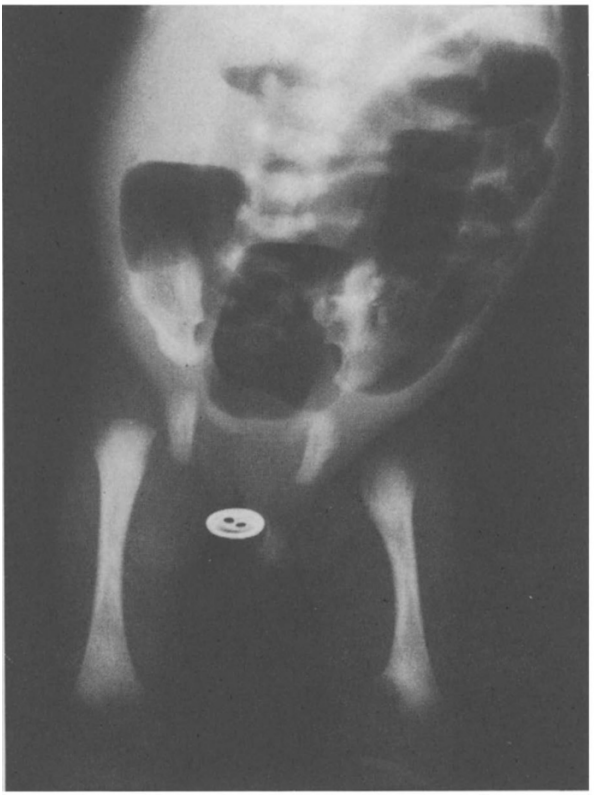


FIG. 17.2 Imperforate anus

Note distance between air shadow in the bowel and the marker on the perineum

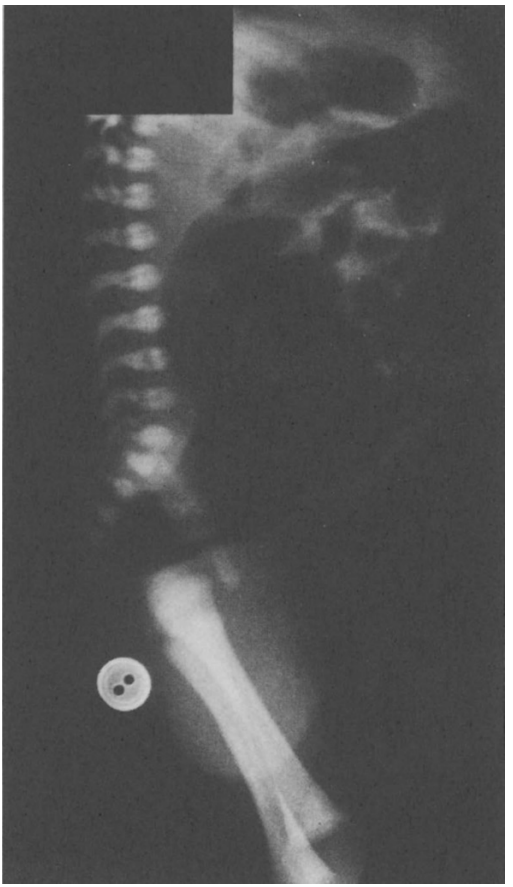


FIG. 17.3 Imperforate anus

Note distance between air shadow in the bowel and the marker on the perineum

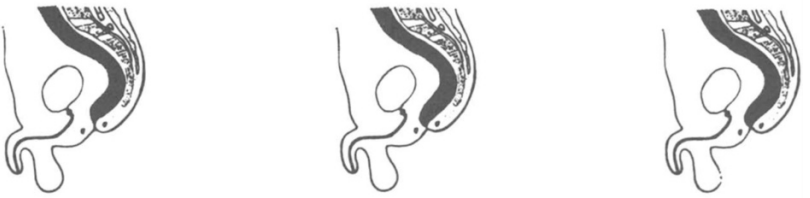
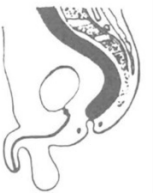


FIG. 17.4 Ano-rectal anomalies

FIG. 17.4 continued

Anal agenesis

Rectal agenesis



Rectal atresia

Exomphalos – This is a readily visible anomaly in which the cord is distended due to herniation of the abdominal contents. These can be manipulated into the abdomen by slowly twisting the cord and thus gradually squeezing the contents back through the umbilical orifice. This manoeuvre is more successful when carried out immediately after birth; delay may lead to distension of the bowel with air and reducing the contents may then become difficult. After reduction, strapping is applied to prevent further herniation. Where the exomphalos is large and reduction is not possible, surgical help may be necessary.

Meningocele and myelomeningocele – May need early closure to avoid leakage and introduction of infection. These babies may need long-term supervision because of associated complications. There may be paralysis and sensory loss in the lower extremities if nerve roots are involved; incontinence of urine and faeces may also occur for the same reason, and associated anomalies like Arnold Chiari malformation may cause hydrocephalus.

MALFORMATIONS REQUIRING PROLONGED SUPERVISION

Congenital heart disease – Transient heart murmurs may be heard in about one-third of all newborns during the first 48 hours of life. Such murmurs are systolic, of the ejection type, not loud and not accompanied by a thrill. Beyond observation of the baby for development of symptoms and repeated cardiovascular examinations nothing further is required.

In newborns who die of congenital cardiac malformation, more than half do so within the first week of life. Hence, whenever a cardiac malformation is suspected the baby should be observed in hospital for a minimum of one week. The common feature of cardiac disease in the newborn is the triad of tachycardia, tachypnoea and hepatomegaly. In addition, there may be persistent cyanosis or a loud murmur.

Hypertrophic pyloric stenosis – The classical symptom of projectile

vomiting usually begins in the second or third week of life but rarely can occur early. The combination of poor weight gain, constipation and vomiting should alert the physician. Examination during a feed will show the characteristic 'rolling ball' movements due to gastric peristalsis and a hard contractile tumour will be palpated in the region of the pylorus (see figure 17.5).

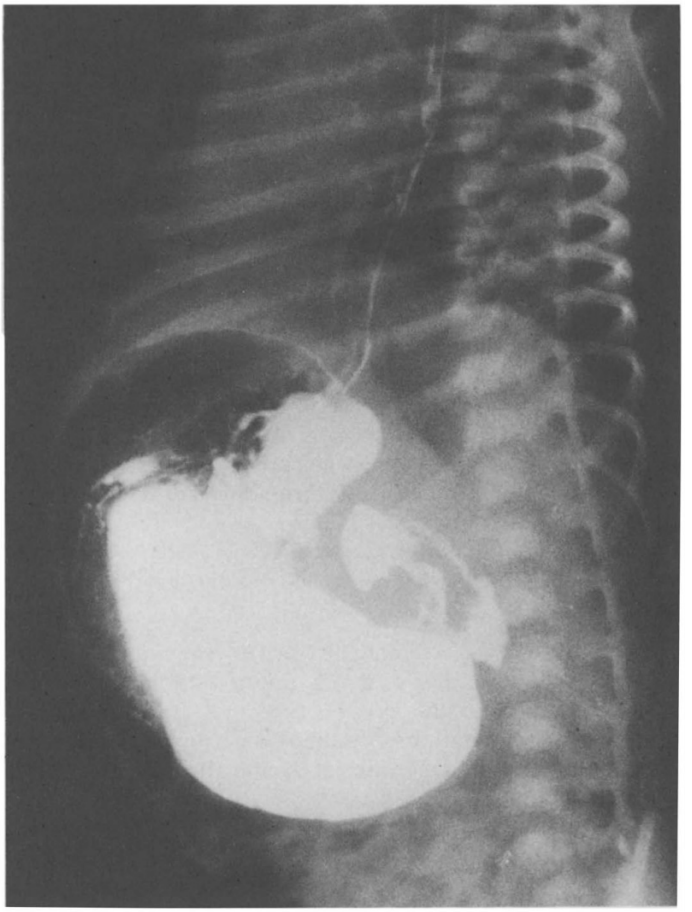


FIG. 17.5 Congenital pyloric stenosis.

Note the 'string sign' – a very narrow flow of barium from the stomach filling the duodenal cap.

The baby may be observed on medical treatment for a few days. This consists of sedation, thickening of feeds, and methyl-atropine nitrate drops, one to two, put on the baby's tongue five minutes before each feed. If there is no relief of symptoms, a Ramstedt operation to split the hypertrophic pylorus will be required.

Hiatus hernia — May also cause persistent vomiting which could become projectile. The onset is usually earlier than in the case of hypertrophic pyloric stenosis. The vomitus shows excessive mucus and occasionally streaks of blood. Nursing the baby in an upright position helps to relieve symptoms.

MISCELLANEOUS CONDITIONS

Harelip and cleft palate — The method of feeding has been mentioned (see p. 62). It is usual to operate on the lip after the age of three months if the baby is growing well. The operation on the palate is delayed until the age of 18 months.

A special problem may arise in children who have this abnormality in association with a small mandible (Pierre-Robin syndrome). The tongue tends to fall back and cause respiratory obstruction. Such babies should be nursed on their abdomen and fed by a naso-gastric tube. In severe cases it may become necessary to pull the tongue out by means of a stitch which is then fixed to the anterior abdominal wall.

Congenital cataract or clouding of the cornea — This is a common abnormality. It may be genetic or secondary to rubella infection in pregnancy. Glaucoma may or may not be associated with corneal opacities. When a lens opacity is reported, the possibility of galactosaemia or retinoblastoma should be eliminated.

The treatment of cataract is operative; in some cases of galactosaemia regression has been reported on dietic therapy.

Occlusion of the naso-lachrymal duct — This will cause continuous watering of the eye and recurrent conjunctivitis. Spontaneous canalisation may occur; if symptoms persist for more than four weeks syringing the naso-lachrymal duct may help to clear the obstruction.

Umbilical hernia — This is very common. In a large majority of the cases no treatment is necessary. Complications of umbilical hernia are rare in the newborn.

In some babies, after the cord has fallen off, granulomatous tissue may be left behind. Daily cleansing with spirit followed by touching with a silver nitrate stick cures it. Occasionally a 'granuloma' is encountered which is resistant to this treatment. If the surface is wet to touch, a patent urachus or remnants of intestinal epithelial tissue may be suspected and appropriate investigations undertaken to rule this out.

MALFORMATIONS ASSOCIATED WITH MENTAL RETARDATION

The most common are mongolism (Down's syndrome) and microcephaly.

Whenever any obvious congenital malformation is encountered, it is necessary and useful to speak to the parents in order to explain the basis of the condition and to give simple genetic counselling if requested. Prolonged supervision of such babies is essential; because of beliefs about evil spirits and witchcraft it is common to see such babies being neglected.

Chapter 18

Morbidity and Mortality in the First Month of Life

The first month of life is a period of stress both for the newborn and the mother. If the baby has been delivered at home, the lack of hygiene, overcrowding and the comings and goings of relatives and friends expose the baby to infections. In addition to this, several ceremonies have to be performed, like naming the baby or 'bringing the baby out', all of which necessitate handling by several people and thus increasing the chances of exposure to infections. In urban areas, where institutional deliveries are becoming popular, overcrowding in the maternity units and nurseries provides an added hazard.

Several traditional concepts persist as regards the care of the newborn; the most important are the ones related to feeding and cord care. Some mothers consider the colostrum to be unhealthy and either do not offer it to the baby or get anxious about it and seek advice from the elders. Often they are told that the milk is bad and consequently only one breast is offered or artificial feeding is resorted to. Unilateral breast feeding is a common cause of failure to gain weight in early infancy.

The first feed offered to the baby may be some form of gruel or water boiled with herbs or roots, which may cause gastrointestinal upsets; on the other hand, force feeding may cause aspiration pneumonia.

Improper methods of cord care are responsible for septic cords or even tetanus. Infection may be acquired from the bath water or from herbs and other applications or dressings on the cord stump.

Often the mother's actions at home may be traced to half-truths learned in hospital relating to the use of feeding bottles in special care units or use of dusting powder for cord dressing. Impersonal attitudes in the maternity unit are thus potentially harmful, and wherever possible mothers should receive careful instruction in the care of their babies. This need is greater in urban hospitals because of the rapid turnover. As a matter of fact, a strong maternal and child health service to support the obstetric team is very necessary; without it, ignorant

motherhood will multiply and give rise to much sickness in babies.

In a survey of Gambian children[42] it was found that the wet season is one of stress and that wide fluctuations in weight gain and haemoglobin levels occur at this time. Certainly, admissions to hospital due to bronchopneumonia, malaria and anaemia are more numerous during the wet months.

In an analysis of hospital admissions of 133 children under the age of one month in Dar-es-Salaam, it was found that sepsis was the commonest cause of admissions – bronchopneumonia, septicaemia, tetanus and meningitis being the major diseases. Feeding problems and undernutrition constituted the second large group, followed by anaemia and jaundice. A miscellaneous group consisting of congenital anomalies and various forms of birth trauma brought up the rest (see table 18.1).

TABLE 18.1 Analysis of admissions of babies under one month of age over a period of six months

Cause of admission		*No. of cases*
(i)	Sepsis – 59 (55.3%)	
	bronchopneumonia	17
	tetanus	15
	septicaemia	12
	meningitis	7
	cord sepsis	6
	conjunctivitis	2
(ii)	Nutritional – 8 (6%)	
	poor growth	6
	feeding problem	2
(iii)	Anaemia – 3.7%	5
(iv)	Icterus neonatorum – 6.7%	9
(v)	Miscellaneous – Birth trauma (8), spina bifida (4), imperforate anus (2), intestinal obstruction (3), cleft palate (1), hypospadias (1), mastitis (1), vaginal bleeding (1), low birth weight (5).	

The commonest causes of death encountered in the above babies were:

tetanus	6
meningitis	3
septicaemia	6
low birth weight	5

When the data of child welfare clinics are analysed a suprisingly low incidence of disease is found. It is probable that mothers who have

learned to make use of the available services have also learned the basic principles of baby and child care. The newborn has inherent protective mechanisms against infection and probably against malaria; these mechanisms become overwhelmed due to maternal ignorance or neglect, and disease occurs. Therefore, the preparation of the mother to look after her newly born baby should be a prominent feature of all health education in ante and postnatal clinics. This should be in the form of instruction in feeding, cleanliness in handling small babies, dressing, protection from flies and mosquitoes, and the acceptance of immunisations in early childhood.

Chapter 19

Traditional Midwifery

Various estimates have put the number of institutional deliveries in developing countries at 20 to 25 per cent of the total number of births. A vast number of children are delivered in the traditional way. The number will, of course, vary from one country to another depending upon the state of development of the health services and the socio-economic level of the people; in the same country there may be variations from one region to another and between urban and rural areas. In many countries there are already indications of increasing demands for Western type of medicine and with this the pattern of midwifery will inevitably change.

To the uninitiated, traditional midwifery may at first glance appear to be the confused attempts of village women at procuring a live birth. This is not really so; many social anthropologists have confirmed that the various procedures and rituals are steeped in tradition as old as the community itself. They are time honoured and as strictly adhered to as the 'scrub up' or 'gown and mask' technique of the modern obstetrician. Any criticism or ridicule is certain to create offence and unnecessary friction between the health personnel and the community they are expected to serve.

The rituals of indigenous midwifery are part of the 'rites of passage' and represent the passage from one stage of life into another and incorporation or acceptance into this new phase. These 'rites of passage' form an important aspect of rural life and constitute a large proportion of all the different ceremonial rites like the ceremony of 'ikipiki' at the time of child birth in the Nyakusa and similar ceremonies in many other tribes, the 'mwali' custom of the Zaramo and similar initiation ceremonies at the time of puberty in many other communities, the wedding rites and the rites performed at the time of death.

In the traditional Bantu society, the female has been socially inferior to the male; from the point of view of procreation, however, she has been ascribed superior powers. In a traditional society where infant and child mortality is high, such a superior role for the pro-

creator can easily be imagined; however, there is much more to it than this. Relationships with another generation, with the ancestral spirits or other clans are maintained through children, especially male children. In the Chagga, for example, it was considered very important that the first born should be a male because at his father's death he would take up duties to perpetuate the memory and authority of the ancestors through prayers and sacrifice. A man who dies without offspring would not be accepted into the company of the ancestors as he would not be receiving any prayers or offerings. Thus, as the one who conceives and brings up the offspring, the female is important to the family, the clan, and the whole tribe. Like the fertility of the land and the cattle, the women of the clan represent a manifestation of the same life-creating power.[43]

It is not surprising therefore, that the fertility of the woman decided her status in society. The continuity of the clan depends on it and the ancestral spirits are thought to guard and nurture this procreative power. Most rituals involving the female of the clan emphasise this role and aim at reinforcing her inborn source for creating the life force. Barrenness has been regarded as a curse; a strict code of ethics must be observed by the adolescents and the young if it is to be avoided.

The pregnant woman must look after her unborn baby by observing proper food taboos and a correct social behaviour. The evil eye or witchcraft is much feared; for this reason some tribes like the Zaramo disregard pregnancy until the seventh month when the ceremonial presentation of the 'sambo' cloth takes place. Such customs may mean a delay in attendance at the antenatal clinic until pregnancy is far advanced.

The delivery is conducted at the home of the wife's mother or that of the husband's mother depending upon whether the clan is matrilineal or patrilineal. The woman of the house and other elderly women with experience attend the delivery.

No special precautions for cutting the cord or care of the cord stump are observed from the point of view of health, though elaborate care of the cord is to be maintained lest by falling across the mother's legs it 'causes' barrenness. Neonatal tetanus and convulsions in children are recognised as dangerous and are feared, and various forms of ritualistic precautions are taken. The association between dust and tetanus is not described in any available accounts of traditional midwifery though certain communities do touch the cord stump with hot ashes from a fire. The placenta is regarded as 'another baby' by many communities and in some, the baby may not be touched by the assistants, even for resuscitation, until after the placenta has been delivered.

Amongst the tribal women in India,[44] most of the deliveries are conducted by traditional birth attendants. Childbirth is commonly

regarded as a time of impurity so that only women of low caste become professional birth attendants. A large majority of them learn their trade from their mothers or mothers-in-law and a few by being apprenticed to practising birth attendants. No antenatal care is usually provided by such attendants. The only nutritional advice given to the mothers is to take very little food in the first three to four days after birth.

Amongst the rural Malay[45] the traditional cultural paractises are the result of several influences – Hinduism since the third century, Islam since the fifth, and the Western system of scientific medicine since the sixteenth. As a result the indigenous medical system is largely based on supernatural beliefs, the ceremonial is partly Hindu, the religious beliefs are mainly Moslem and the Western system of medical care has remained confined to urban areas. It is believed that childbirth attracts various evil spirits and precautions must be taken to protect the mother and child from their influences. It is customary to place obstacles in the form of thorny bushes near the door of the house or to mark it with charms and spells. The afterbirth is considered as a sibling and great care is taken to dispose of it correctly. After delivery, the mother is confined to bed for a period of forty days; during this time she is visited daily by the birth attendant who gives body massage and advises on minor problems as they arise. This period of enforced rest from household chores, during which the baby is the mother's only thought and preoccupation, must help to establish a strong bond of love and is useful for successful lactation.

In New Guinea, the pregnant woman has a great fear of spirits of the forest and she must observe correct practices and behaviour in order not to offend them. The work of changing the behaviour of the mother through health education and attendance at the antenatal clinic is thus made incredibly difficult. The birth must take place in a part of the house specially reserved for childbirth; the mother delivers the baby and the placenta herself, cuts the cord and buries the placenta inside the house, and no assistance is given by the attendants except in dire emergencies. This practice is responsible for the high incidence of neonatal tetanus in isolated rural areas. If labour is difficult, it is thought that the spirits of the forest are withholding the birth of the baby and various rituals must be performed to placate the spirits.

Any change in the indigenous practices of midwifery will be acceptable only when it comes within the context of the traditional role of the female in the clan. Then too, indiscriminate rejection and criticism of age-old traditions and customs can only produce resistance. Modifications in the various adminstrative aspects of institutional deliveries may be necessary to make such deliveries more acceptable to rural people. For example, an elder female of the family may be allowed to attend the delivery in the hospital or health centre or may be

allowed to take the placenta and other products of conception home for appropriate disposal. Health education compaigns may be needed against certain concepts which are positively harmful. For example, when the delivery is prolonged or if any difficulties arise, they are thought to be due to misconduct during pregnancy, or adultery, or offence given to the elders. The woman in labour may be asked to confess, or the offended elder has to forgive or a sacrifice is to be offered to the ancestral spirits. Valuable time may be lost in this, and finally when medical aid is sought from the local hospital it may be too late. Medicines and herbs given to 'help' the delivery may produce disastrous results. The same also applies to the tradition of considering twins, breech deliveries and babies with congenital physical abnormalities as a curse on the family and the clan and calling for 'purification' ceremonies. Even in urban maternity hospitals it is common to see mothers neglecting or disowning such babies.

In the developing world, a large majority of children are being delivered in rural areas by traditional birth attendants. The first step towards improving perinatal care will logically be the integration of the indigenous midwives into the national health services. Many countries have organised programmes of training for the traditional birth attendants, notably the Sudan where institutions for the training of these workers have been in existence for more than fifty years. Almost all countries have expressed success with such efforts; for example, it has been shown that with adequate training of indigenous birth attendants, the mortality from neonatal tetanus can be reduced by as much as two-thirds in a period of six months. It has also been demonstrated that with adequate training such birth attendants are able to primarily detect high risk pregnancies for referral to the health centres. When they feel themselves to be part of the health team, suspicion disappears and they are pleased to be supervised and supported by health personnel from the local sub-centre.

In all developing countries forces of social change are gaining strength. Increasing literacy and basic education, national women's organisations, youth leagues, community development programmes and many such activities provide the basis for the spread of modern ideas on health and disease. It is likely that under the impact of social change, rural midwifery will also change and become based on scientific concepts.

Appendix I

DRUG THERAPY IN THE NEWBORN

(1) ANTIBIOTICS

Inj. penicillin crystalline – 125,000 units twice daily.

Penicillin eye drops – 20,000 units/ml (to be freshly prepared before use).

Inj. Streptomycin – 50 mg daily.

A Cloxacillin – 100 mg/kg/day
Ampicillin – 50 mg/kg/day
} commonly used combination for systemic infections.

Chloramphenicol – 50 mg/kg/day. In prematures use only half the dose.

Neomycin – 50-100 mg/day (in four equal doses). Preferred for gastro-intestinal infections.

Sulphadiazine – 100 mg/kg/day (in four equal doses).

Gentamycin – 2.5 mg/kg (six hourly).

(2) RESPIRATORY STIMULANTS

Nikethamide – 25 per cent solution (taken from an ampoule) 0.25 ml to be placed on the tongue as drops, four drops at a time.

Inj. N-allylnormorphine (Nallorphine) – 0.2 mg/kg (I.m. or i.v.) to be used only if the depression of respiration is due to excessive amounts of morphine or related compounds given to the mother in labour.

Oxygen – By funnel – rate 4 litres/min.
By face mask – rate 2 litres/min.
By nasal tube – rate 1 litre/min.
In incubators, concentration not to exceed 35-40 per cent.

(3) TREATMENT OF CARDIOVASCULAR DISEASE

Digoxin – digitalising dose 0.04-0.08 mg/kg/day
Half the total calculated dose is given stat and the remaining in two portions at 6 hour intervals.
} Available as Lanoxin syrup 0.05 mg in 1 cc
Maintenance dose – $^1/_3$ the digitalising dose given once daily.

Inj. Mersalyl – Not to exceed 0.25 ml in 24 hours.
(Recommended dose: 0.25 ml once a week.)

(4) ANTI-CONVULSANTS

Inj. Paraldehyde – 0.2 ml/kg can be repeated after six hours if necessary.

Phenobarbitone – 3-6 mg/kg/day divided in two doses.

ACTH – 1.M. 3-5 units/kg/day in four divided doses; for massive myoclonic seizures.

Pyridoxine – 2-5 mg/day for pyridoxine-dependent convulsions.

(5) TETANY OF THE NEWBORN

Inj. calcium gluconate i.v. -- 20 mg/kg as a 10 per cent solution to be given in two divided doses at four hour intervals.

Orally 0.5-1.0 g per dose, two to three times daily in milk.

Vitamin D – 1,000 units/day.

(6) HYPOGLYCAEMIA OF THE NEWBORN

On diagnosis, 50 per cent glucose 1-2 ml/kg given i.v. stat followed by i.v. drip 15 per cent glucose in water or N/5 saline at the rate of 75-100 ml/kg/day.

(7) ANTIPYRETICS

Paracetamol (available as a paediatric suspension) – 40 mg per dose can be repeated after six hours.

Soluble aspirin – 30-60 mg dissolved in water. Can be repeated after six hours.

(8) HAEMORRHAGIC DISEASE

Inj. vitamin K – Therapeutic 2.5-5.0 mg i.v. to be repeated after six hours. Preventive: 1.0-2.5 mg (one dose only). Excess of vitamin K can produce jaundice.

(9) VITAMINS

A – 2,500-5,000 i.u. Excess produces anorexia, pain and thickening of bones (Caffey's syndrome).

B1 (thiamin) – 10 mg i.m. therapeutic dose.

B6 (pyridoxin) – 2.5 mg/day.

C (ascorbic acid) – 25-50 mg/day (therapeutic: 100 mg twice daily for 3 days).

D – 500-1,000 units/day.

K – Preventive: 1-2.5 mg as a single dose (therapeutic: 5 mg i.v. to be repeated after 6 hours if necessary).

(10) CANDIDA ALBICANS INFECTION

Gentian violet – 1-2 per cent in water for local application.

Mycostatin – 100,000-200,000 units thrice daily by mouth.

(11) SEDATIVE

Chloral hydrate – 30-60 mg as a single dose twice daily.

Phenobarbitone – 3-6 mg/kg/day in two divided doses.

(12) NEONATAL TETANUS

Inj. A.T.S. – 40,000 units i.m. stat and 5,000 units for subcutaneous infiltration around the umbilical stump.

Cord care with Savlon solution (1 in 200).

Inj. crystalline penicillin – 125,000 units b.d. for two days followed by procaine penicillin 200,000 units daily for one week.

After controlling spasms with Inj. paraldehyde or phenobarbitone, pass nasogastric tube for feeding and medication.

Phenobarbitone 6-12 mg/kg body wt. in 24 hours divided in four equal doses at 6 hourly intervals (average 15-30 mg at 6 a.m., 12 mid-day, 6 p.m., 12 midnight) to alternate with chlorpromazine (4-16 mg/kg in 24 hours) or diazepam (2-8 mg/kg in 24 hours). (Average – chlorpromazine 8-10 mg and diazepam 2-4 mg at 9 a.m., 3 p.m., 9 p.m., 3 a.m.)

Dose of phenobarbitone to be adjusted according to sedation and of chlorpromazine or diazepam according to control of spasms.

Appendix II

FLUID THERAPY IN THE NEWBORN

Daily requirements

The fluid and caloric requirements of the normal newborn are estimated as 75 cc and 100 calories per kg/body weight, in twenty-four hours. Babies with a low metabolic turnover existing at or *below basal caloric requirements*, and *without excessive losses*, require less water; on an average such babies require 60 cc/kg in twenty-four hours.

Assessment of dehydration

Loss of weight is a reliable measure of assessing the degree of fluid loss. Any rapidly occurring weight loss in the first few weeks of life should cause concern. If any of the clinical causes for dehydration exists, e.g. vomiting, diarrhoea, aspiration of stomach, etc., about 50 per cent of a rapid weight loss may be assumed to be due to loss of water and electrolytes and an equivalent amount must be added to the daily requirement. Thus, if a baby with a known weight of 3 kg loses 300 gm in weight, at least half of this amount (i.e. 150 cc) of fluid should be added to the daily requirement of 225 cc making the total of 375 cc to be administered in twenty-four hours.

The majority of diseases which cause acute dehydration in the newborn are associated with isotonic or hypotonic dehydration. In most instances 4.3 per cent dextrose in 0.18 N saline will be found useful. Infants with diarrhoea however are an exception to this generalisation – they may have hypertonic dehydration.

Precautions

In dealing with fluid therapy in adults there is a wide margin of safety; in the newborn renal function is not efficient and the margin of safety is narrow.
The following precautions will be useful in all newborns to whom intravenous fluids are being administered:

(1) Fluid containers in common use have a capacity of 500 or 1,000 cc. In order to avoid accidental overhydration all extra fluid in the container other than the calculated amount should first be poured out before beginning the infusion. Recently, small disposable burette flasks are commercially available, and these may be used as an alternative.

(2) Clinical assessment of the hydration status of the baby should be done every four hours.

(3) If a drip has to be continued for more than twenty-four hours, serum electrolyte studies should be performed.

(4) A close watch should be maintained for signs of overhydration like puffy face or feet, tachycardia and tachypnoea.

(5) Sepsis and thrombophlebitis at the site of the intravenous infusion are common complications especially if a cut-down has been done. In the rare instances where a drip has to be continued for more than 72 hours the site should be changed.

Route of administration

In some cases, administration of fluid and electrolytes by naso-gastric tube may be possible, e.g. prematures who cannot suck, babies with cerebral palsy who cannot maintain adequate intake, babies with chest or systemic infections who are too weak to suckle.

Subcutaneous fluid administration is unreliable and may even prove risky; it should be avoided.

Intravenous therapy is commonly required for dehydration due to gastro-enteritis (which will not arise in the first place if bottle feeding is prohibited in the nursery) and in surgical conditions with acute intestinal obstruction. In the latter, most surgeons now recognise that early relief of intestinal obstruction combined with emptying of the distended loops of the bowel above the obstruction is important. The fluid in the lumen of the distended loops is in equilibrium with the extracellular fluid; when saline is administered before operation, the abdominal distension may increase because much of the administered fluid accumulates in the obstructed bowel. Relief of obstruction should not be delayed until full hydration has been achieved, but should be attempted early.

Appendix III

MISCELLANEOUS SIDE-ROOM TESTS

Diarrhoea due to sugar intolerance

Much of the sugar which is not absorbed or fermented by the intestinal flora is excreted in the stool and the diarrhoea is caused either by bacterial fermentation of the sugar or by the osmotic effect produced by the sugar in the lumen of the intestine. It has been found that children proven to be intolerant to sugar excrete 1 g or more per 100 ml of faeces when fed the offending sugar.

'Clinitest' may be used as a rapid screening test for diagnosing sugar intolerance. As the stools are fluid, they should be collected on mackintosh or plastic sheet and not on an absorbent napkin.

Two volumes of water are added to one volume of the stool and mixed well. Fifteen drops of the suspension are added to a clean Ames test-tube and a clinitest tablet is added. The chemical reaction is similar to that of urine and the quantity of sugar can be found by reference to the colour chart. A result of ¼ per cent or less is negative; between ¼ per cent and ½ per cent suspicious, and above ½ per cent indicates presence of abnormal quantities of sugar in the faeces.

Maternal blood as a cause of malaena

Swallowed maternal blood at the time of delivery may cause malaena stool in the first day of life. Ingestion of over 30 cc of maternal blood is required for the production of such a stool. Whether the blood in the stool is derived from the mother or the baby can be differentiated by the alkali denaturation test: the adult haemoglobin turns brown (due to formation of alkaline haematin) when exposed to alkali, while foetal haemoglobin being alkali-resistant, does not undergo a colour change.

To do the test a small amount of the stool is mixed with water. The mixture is centrifuged, and the supernatant haemoglobin solution is decanted. One part of 0.25 N solution of sodium hydroxide is mixed with five parts of the haemoglobin solution. Adult haemoglobin will change to yellow-brown colour in two minutes; foetal haemoglobin will remain pink.

Detection of foetal red blood cells in maternal circulation

The test is based on the principle that adult haemoglobin is eluted from erythrocytes whereas foetal haemoglobin resists elution and can be demonstrated by staining. The method is described on page 125.

(1) A thin blood smear is drawn out on a slide with capillary blood obtained by finger prick. The smear is allowed to dry in air for ten minutes.

(2) The slide is fixed by covering with 80 per cent ethanol for five minutes. It is then rinsed thoroughly in tap water and allowed to dry in air.

(3) The fixed slide is immersed in citrate-phosphate buffer at 37^0 C in an incubator for five minutes, agitating at one and three minutes. (Citrate-phosphate buffer is prepared by mixing 73.4 parts of citric acid solution 0.1 M with 26.6 parts of disodium hydrogen phosphate solution 0.2 M.)

(4) The slide is rinsed thoroughly in tap water and then dried completely.

(5) The slide is stained for 3 minutes in acid haematoxylin, rinsed in water, and then stained for five minutes in eosin.

(6) The slide is rinsed, dried and is ready for examination under the microscope. The foetal cells are darkly stained; their proportion can be calculated by counting the dark-staining (foetal) cells and non-staining (maternal) cells in 50 high-power fields.

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