Introduction
It is perhaps trite to emphasize that the child is not merely a small adult, but nowhere is this distinction more apparent than in the neonate.

The transition from intrauterine to extrauterine life requires fundamental changes in the circulatory, respiratory, metabolic, and immune functions of the newborn. When a surgical pathology is added to the mix, these essential adaptations can be compromised, leading to organ dysfunction. Single organ dysfunction is frequently the start of a cascade that rapidly results in failure of the entire organism. Thus, the emphasis in neonatal care is on the prevention of problems rather than on the management of disasters once they have occurred. In order to prevent dysfunction, it is important to recognize patients at particular risk but also to have in place general principles of care and to train nursing staff and paramedical personnel in their application. Neonatal care is a team effort. Whenever possible, the team should include the mother and other family members, if culturally appropriate.1

Neonatal physiology is not defined by geography or politics, but our ability to recognize and respond to system dysfunction is a factor of the human and material resources available. In a developing country where scarce resources must be utilized for the maximum benefit of numerous constituencies, imaginative alternatives to standard Western care are required. It is in this environment that maternal ill health and deficient antenatal care add to the considerable difficulties faced by neonates during the perinatal period.

Many of the neonate’s survival mechanisms are installed during the third trimester of pregnancy, and preterm delivery can additionally challenge the successful transition to independent life, the difficulties being directly proportional to the degree of prematurity.

Occasionally, the uterus proves to be a hostile environment for the developing foetus and, in conjunction with obstetric colleagues, pregnancy management will need to take account of the interests of both the foetus and the mother. Certainly, the antenatal recognition of surgical disease calls for skilled management of the pregnancy and delivery and provides the surgeon with an unborn patient for whom diagnosis, prognosis, investigation, and management are difficult.

A few specific anomalies can be ascribed to genetic, teratogenic, or infectious causes, but the pathogenesis of most congenital malformations remains unknown. It is improbable, however, that any insult that results in a congenital abnormality will affect a single system or structure without affecting other structures that are developing at the same time. Thus, multiple abnormalities should be suspected and sought in every neonate presenting for surgery.

Neonatal Classification
Neonates come in a variety of sizes and degrees of maturity. It is important to recognize risk factors in any patient presenting for surgery, but particularly so in the neonate, in whom body weight and prematurity are easily assessed and critical to defining the likely co-morbidities and risk factors and help to determine appropriate management (Figure 2.1).

Many “surgical” babies are born before term; in addition, they are often small for their gestational age (SGA). Causative factors include maternal infections and poor nutrition, placental insufficiency, maternal cigarette smoking, and maternal substance abuse such as drugs and alcohol. Frequently, polyhydramnios complicates a pregnancy in which the foetus has an intestinal abnormality due to the inability to ingest and recyle amniotic fluid, stimulating early labour. Such babies are therefore exposed to the risks of prematurity and its associated problems as well as the morbidity of a surgical pathology. Recognition of a neonate’s status allows prediction of potential clinical problems, thus allowing preventive steps to be taken. Table 2.1 outlines the traditional risks faced by SGA and preterm infants.

The risks associated with prematurity are reflected in the mortality of preterm babies without surgical disease and depend upon the degree of prematurity and body weight (Figure 2.2). This risk of mortality must be weighed against the available resources and the nature of the surgical problem before a decision on management can be taken. Such considerations are particularly germane to the practice of neonatal surgery in a developing country where both human and material resource limitations may be extreme.

Temperature Control
The neonate is designed as a radiator with a large surface area relative to its mass. Heat is lost through convection, conduction, radiation, and the latent heat of evaporation of transdermal fluid loss. In the term neonate, heat loss is reduced by a layer of insulating subcutaneous fat and a thick skin that reduces transdermal fluid loss. Heat production comes from hepatic glycogenolysis and the metabolism of brown fat, a metabolic response termed “nonshivering thermogenesis”.2 All of these defences against heat loss are weakened in the preterm infant, who has a thin skin, increased transdermal water loss, no subcutaneous fat,1 and who has been born before having the opportunity to lay down any brown
fat. In premature babies, insensible water loss can amount to 3 ml/kg per hour, and even in term babies it is around 1 ml/kg per hour. These losses can be minimised by nursing the baby in a humid environment, but this is rarely practicable. Heat loss through convection and conduction can be reduced by nursing the neonate in a warm environment.

In surgically ill neonates, further heat loss occurs in vomitus, tachypnoea, and, of course, during the massive increase in surface area that occurs when the baby’s abdomen is opened by the surgeon, or where there is evisceration at birth, as occurs in babies with gastroschisis or ruptured exomphalos.

Babies who become cold must try to maintain temperature by using their scarce energy stores, but these are rapidly exhausted. The well baby can replenish these energy stores by feeding. The surgically ill baby cannot. Cold then leads to further depletion of energy stores, protein breakdown, acidosis, sclerema, increased oxygen consumption, sepsis, and death. It is clear from Figure 2.3 that keeping the baby warm minimises the metabolic rate and oxygen consumption, but the zone of thermal neutrality is narrow. Hypothermia is formally defined as a core temperature lower than 36°C.

Prevention is much better than cure. Keeping a baby warm requires strategies different to those required to warm up a cold baby. A baby can be kept warm by enveloping him, and his head, in an insulating material such as a blanket or aluminium foil, obviously ensuring that the airway is not obstructed; doing this to a cold baby will simply keep him cold. The mother’s body is an excellent heat source and so-called “kangaroo” care also aids in maternal bonding. It would appear that, at least in the short term, fathers are capable substitutes.

Ideally, surgically ill babies should be kept warm in incubators when these are available. Most babies can be accommodated in incubator temperatures of 32–33°C. Babies in incubators still lose heat by radiating it into space. In a perfect world, double-lined incubators would be standard, but radiation losses can also be reduced by covering the baby with a sheet of paper.

Making a cold baby warm requires an external heat source, and warming should take place slowly; attempting to rapidly warm a baby with an electrical heater inevitably results in dermal burns. During rewarming, it is wise to check the baby’s blood sugar level.

Cardiovascular Adaptation

Before birth, the baby’s circulation is based upon the placenta, which acts as lung, kidney, and nutrient supply. Thus, the umbilical vessels are of paramount importance. Blood arriving at the foetus from the umbilical vein is shunted across the liver through the ductus venosus and away from the lung through the foramen ovale. The foramen ovale is simply a flap “gate” that is held open because the pressure in the right atrium is higher than the left atrial pressure. Because the lungs require little blood flow before birth, blood is also shunted from the right ventricular outflow into the aorta through the ductus arteriosus before returning to the placenta through the umbilical arteries (Figure 2.4).

When the obstetrician clamps the cord, flow to the right atrium is reduced and the right atrial pressure falls. There is a simultaneous increase in left atrial pressure in response to increased pulmonary blood flow that follows the decreased pulmonary vascular resistance caused by lung expansion with the first breath. This allows the “gate” to close the foramen ovale. With the onset of breathing, there is an increase in peripheral oxygen concentration that stimulates the ductus arteriosus to close, a muscular contraction probably mediated through prostaglandin. The closure of both the foramen ovale and the ductus arteriosus are temporary. They can be reopened by anything that increases right atrial pressure relative to the pressure in the left atrium or that decreases peripheral oxygen concentration. Permanent closure is not achieved in the neonatal period.

Reopening these temporarily closed shunts restores the infant to the foetal circulatory pattern, but there is no longer a placenta that can act as lung or kidney, and unless the adult circulatory pattern can be rapidly re-established, the infant will die. Pulmonary hypertension, seen, for example, in neonates with diaphragmatic hernia, will cause an increase in pressure on the right side of the heart, a right atrial pressure that will exceed left atrial pressure, reopening of the foramen ovale, and ultimately reduction in peripheral arterial oxygen concentration and reversal of ductal closure. Without a placenta, this circulatory pattern

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**Table 2.1: Predictable problems in small for gestational age (SGA) and preterm average for gestational age (AGA) babies.**

<table>
<thead>
<tr>
<th></th>
<th>SGA</th>
<th>Preterm AGA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung</td>
<td>Pulmonary haemorrhage</td>
<td>Hyaline membrane disease</td>
</tr>
<tr>
<td>Apnoea</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Hypoglycaemia</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Hypothermia</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Jaundice</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Haemoglobin</td>
<td>Polycythaemia</td>
<td>Normal</td>
</tr>
<tr>
<td>Feeding capacity</td>
<td>Normal</td>
<td>Reduced</td>
</tr>
<tr>
<td>Congenital</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>malformations</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality</td>
<td>+++</td>
<td>Depends upon gestational age</td>
</tr>
</tbody>
</table>

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**Figure 2.2: Neonatal classification and mortality risk (LGA = large for gestational age; AGA = appropriate for gestational age; SGA = small for gestational age).**

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**Figure 2.3: Metabolic rates, temperature, and oxygen consumption.**

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**Figure 2.4: Cardiovascular Adaptation.** Before birth, the baby’s circulation is based upon the placenta, which acts as lung, kidney, and nutrient supply. Thus, the umbilical vessels are of paramount importance. Blood arriving at the foetus from the umbilical vein is shunted across the liver through the ductus venosus and away from the lung through the foramen ovale. The foramen ovale is simply a flap “gate” that is held open because the pressure in the right atrium is higher than the left atrial pressure. Because the lungs require little blood flow before birth, blood is also shunted from the right ventricular outflow into the aorta through the ductus arteriosus before returning to the placenta through the umbilical arteries (Figure 2.4).

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is unsustainable. Pulmonary vascular resistance can be increased, and the foetal circulation reproduced, by hypoxia, acidosis, catecholamine secretion, hyperthermia, or hypoglycaemia, as well as conditions that primarily cause pulmonary hypertension.

The circulating blood volume of a term neonate is in the order of 80 ml/kg body weight. This small volume means that precision is essential in the prescription of intravenous fluids, as an apparently trivial error of 1 ml/kg per hour in a 3-kg baby will result in an error of 10% of the circulating blood volume by the end of the day. Similarly, all losses should be carefully measured and replaced.

**Respiratory Adaptation**

During normal delivery, the fluid that has filled the lungs during foetal life is expelled and the lungs are expanded with air during the first breath. Along with lung expansion, there is a reduction in pulmonary vascular resistance and a redirection of blood flow to allow gas exchange.

Neonates are obligatory nasal breathers and obligatory diaphragmatic breathers. Resistance to air flow is increased by nasogastric intubation, and for this reason—as well as the danger of perforation of the cribiform plate during insertion—orogastric intubation is preferred in this group of patients.

Abdominal distention of any cause will impair diaphragmatic mobility and therefore impede breathing. The number of alveoli in the neonatal lung is less than 10% of the adult quota, but new alveoli are continually added up to 8 years of age. Despite this paucity of alveoli, the resting neonate requires more oxygen per kilogram body weight than an adult, so the neonate is at risk if oxygen requirements are increased or if any pathology diminishes the surface available for gas exchange.

Alveolar stability is maintained by surfactant, a phospholipid wetting agent produced by the type II pneumocyte, which reduces the surface tension in the fluid lining the alveoli. Adequate levels of surfactant are achieved around 35 weeks of gestation. Babies born before this are at risk of developing hyaline membrane disease. It is possible to predict lung maturity antenatally by measuring amniotic fluid phospholipid concentrations.

Air flow is proportional to the fourth power of the radius of the airway, and a small reduction in calibre (for example, by mucosal oedema) can have a major effect on resistance to air flow and therefore on the work of breathing. Decreased ventilation will result in alveoli being perfused but not aerated, creating an intrapulmonary shunt, with a fall in peripheral oxygen saturation and an increase in the partial pressure of carbon dioxide in the arterial blood (paCO₂).

Aspiration of vomitus is common in surgical babies at all phases of their management and is a leading cause of airway oedema, lung contamination, and death. It can be prevented simply by never allowing a surgically ill baby to be nursed supine. A neonate cannot turn over to protect his airway, and vomiting in a supine position inevitably leads to aspiration. Babies are perfectly happy on their sides or prone, and the culture of nursing babies supine has little merit. The canard that it reduces the risk of sudden infant death syndrome (SIDS) is vastly outweighed by the numbers lost each year to aspiration pneumonia.

Similarly, analgesia is important for postoperative respiratory care, as a baby in pain will not breathe deeply, or cry, and will have diminished respiratory excursion, leading to atelectasis, intrapulmonary shunting, and ultimately infection. After thoracic or upper abdominal surgery, adequate analgesia may obviate the need for postoperative ventilation.

Immaturity of the respiratory centre is held to be the cause of apnoea in prematurity. This usually responds to tactile stimulation but may require treatment with theophylline. The risk of apnoea following a general anaesthetic remains for up to a year postnatally in formerly premature babies. All such babies undergoing an anaesthetic for whatever reason should be kept under observation, with apnoea monitoring, for 24 hours after surgery.

**Clinical Evaluation**

Because babies cannot vary their tidal volume, their initial response to inadequate ventilation is to increase the rate of breathing. Due to the flexible cartilaginous nature of the chest wall, any increase in the work of breathing is manifest by intercostal, sternal, and subcostal recession as well as alar flaring. As the neonate tries to increase positive end expiratory pressure (PEEP) to maintain alveolar patency, grunting may occur. The increased work of breathing will eventually tire the baby, who will be unable to sustain these compensatory tactics and will go into respiratory failure.

Babies with clinical signs of respiratory insufficiency should receive supplementary oxygen pending investigation with a chest x-ray and blood gas analysis, if available. Any increased work of breathing associated with abdominal distention can often be ameliorated by the passage of an orogastric tube and maintenance of gastrointestinal decompression. Viscid tracheal secretions can sometimes be suctioned following humidification, best effected by nebulisation with saline.

**Nutrition**

The provision of energy as well as the substrate for growth and development is critical to the neonate, and the provision of adequate nutrition is particularly important for the developing brain. Perinatal deficiencies may have lifelong consequences for the patient, particularly with regard to brain growth and development. Nutrition is also pivotal to wound healing, temperature maintenance, and immune function.

Babies who start life with the handicap of intrauterine growth retardation, and those with surgical disorders that are not promptly recognised, are at particular risk of neonatal malnutrition. Whereas normal babies can be fed through the alimentary tract, the surgically ill neonate is frequently unable to tolerate feeding.

In the developed world, this conundrum is resolved by using total parental nutrition (TPN), but in many developing countries this is unavailable. The standard of care in the developed world has evolved on the back of the availability of TPN and is often inappropriate care.
when TPN is unavailable. As getting energy and substrate into the patient is a priority, there may be no alternative to adjusting surgical strategy to allow early use of the alimentary tract. This may involve placing feeding tubes distal to, or through, an anastomosis or creating a stoma above, or instead of, an anastomosis. An extracorporeal gastrointestinal bypass can be created by aspirating bile-containing fluid from above an obstruction and returning it with a feed either via a stoma or via a trans-anastomotic tube, distal to the obstruction. Even when full feeds are not tolerated, there is merit in providing “trophic” or “trickle” feeds that maintain the integrity of the intestinal mucosa.15

The advent of the human immunodeficiency virus (HIV), particularly the recognition of the seroconversion of breast-fed babies, has added a further confounding variable.16 Breast milk is the best, cheapest, and generally most readily available feed for babies, and it is ideal for the surgical patient. These advantages must be weighed against the risk of transmission of HIV and the economic circumstances of the family.17 It should be remembered that breast milk contains lactose and that many gastrointestinal disorders result in temporary lactose intolerance with resulting diarrhoea.18

The term neonate requires about 120 kCal/kg per day to maintain health. The surgical neonate, after a very brief postoperative increase in metabolic rate that lasts only four to six hours,19 may require fewer calories than normal due to immobility and growth inhibition as well as reduced thermogenesis.20 Providing too many calories (overfeeding) may increase CO₂ production from lipogenesis.

The premature baby has an increased caloric requirement, up to 130 kCal/kg per day.21

Hypoglycaemia
SGA infants, those with diabetic mothers or who have specific conditions such as Beckwith-Wiedemann syndrome are at risk of hypoglycaemia in the first few hours of life. Failure to recognise hypoglycaemia will result in unnecessary neurological morbidity. Clinical signs include apnoea, the tremors or jitteriness, followed by convulsions. The blood sugar should be kept above 2.2 mmol/l by infusion of 10% dextrose if necessary. The blood sugar level should be monitored in all at-risk babies.

Hyperglycaemia
The stress response results in hyperglycaemia in many neonates with emergency surgical conditions, and is common after surgery.22 Premature babies appear to have a higher normal blood sugar, and moderate degrees of hyperglycaemia (blood glucose < 15 mmol/l) can be tolerated. Glycosuria with a resultant osmotic diuresis occurs only with glucose levels around 12 mmol/l due to greater renal retention in the premature infant.

Immune Function
The normal intrauterine environment is sterile, and the neonate has limited exposure to antigens before birth. Both the B-cell and T-cell populations are naïve, and the neonate depends primarily on the “innate” or nonspecific functions of the immune system.

Circulating neutrophils have a half-life of around 8 hours and must be constantly replenished to effectively combat infection. However, the neonate has a low bone marrow storage pool of neutrophils, and although they are functionally competent, they respond poorly to chemotactic stimuli and are rapidly consumed. The neonate also has deficiencies in the complement system.23 Almost all immunoglobulin at birth is maternally derived immunoglobulin G (IgG). This immunoglobulin has a half-life of around 3 weeks. Immunoglobulin M (IgM) production is very limited. Immunoglobulin A (IgA), the mucosal surface immunoglobulin, is acquired from breast-feeding.24 The naïve T-cell population exposes the neonate to the risks of viral and fungal infections, and the “lazy” leukocyte population means that infections are poorly localised and septicemia is frequent.

Thus, the neonate is immunodeficient when compared to adults. This deficiency becomes more apparent when the nonspecific defences against infection are breached, usually by a well-intentioned physician. Thus, intravenous cannula placement, urinary catheter insertion, endotracheal intubation, and orogastric intubation all bypass important defence structures. Thought must be given to the necessity of these interventions because each has an inherent risk. When deemed essential, all interventions must be performed aseptically. Tubes and catheters should be removed as soon as practicable. It is not possible to make up for deficiencies of hygiene by prescribing antibiotics.

Clearly, antibiotics do have a place in the management of infection but the importance of fungal and viral sepsis must be remembered. The more premature the patient, and the longer the intensive care unit (ICU) stay, the greater must be the clinical suspicion of fungal sepsis, even when cultures are not possible.

Neonatal Transport
The centralisation of neonatal surgical services has made neonatal transportation25,26 inevitable. From the outset it must be recognised that transportation is not good for babies; it adds to their stress and represents a break in the continuum of care. Neonatal transportation is an exercise that demands the highest professionalism and planning and should only be undertaken in close cooperation with the unit receiving the infant. Formal training in transportation skills is provided in most US paediatric training programmes, but forms only a small part of the undergraduate or postgraduate training in most African schools.

In the Third World, conflicting health care agendas transpire to limit the human and material resources allocated to interhospital transport. This is something of a false economy, as the inexpert transport of a baby results in either a death on arrival (DOA) or delivers a patient in whom resuscitation and avoidable morbidity adds to the cost of management.

Care in transport is also compromised by the infrequency with which any individual medical officer is required to effect transfer, and the small proportion of his practice that this involves. This lack of familiarity often leads to unseemly haste in attempting to get a baby to a tertiary centre. Speed is never an issue; care is.

In an ideal world, the baby would be stabilised in the ICU at the referring hospital, and transported by dedicated medical and paramedical staff in a mobile ICU to the welcoming staff of the ICU at the referral hospital. It is, however, not an ideal world.

Fortunately, most surgically ill babies require minimal technological support during transport, and there is much that can be done without recourse to mobile ICUs and high technology. Most transferred babies have some form of intestinal obstruction and require little other than gastric decompression, intravenous fluid, and temperature maintenance.

Incubators and other devices that rely on electricity or batteries, which always seem to be flat when needed, are not essential. Heat and fluid loss can be minimised by wrapping exposed viscera in clear plastic sheeting such as can be found in most kitchens, or, when necessary, a plastic shopping bag. A warm baby can be kept warm by enveloping him in an insulating blanket of aluminium foil, another kitchen accessory. It is important to include the head, which represents heat loss, but not the face.

Denim bags containing mung beans or any grain, heated for 1 minute in a microwave oven, can provide sufficient heat to keep a neonate warm for two hours during transport, or, more traditionally, hot water bottles (held remote from the skin to prevent burns) can be used. Portable incubators that have hot water bottles as their source of heat were the standard of care when New York City introduced a transport service for premature babies in 1948. That principle is still sound.

An incubator is just a transparent box designed to keep a baby warm. The heat it provides is no better than the heat provided by mung beans or hot water, and it is certainly more expensive. We re-emphasize that there is a difference between keeping a warm baby warm, which is what is required during transport, and making a cold baby warm, which is something
that must be done as part of stabilisation before transport. The critical importance of temperature maintenance is demonstrated in the higher mortality of babies who are hypothermic on arrival at a tertiary centre.

Gastric decompression is a vital intervention. By keeping the stomach empty, the risk of vomiting and aspiration is reduced. It can be reduced still further by nursing the baby in the lateral position, or prone, during transport. Gastric decompression also relieves pressure on the diaphragm, reducing the work of breathing. It reduces the diameter of the bowel, thus reducing tension in the bowel wall and allowing greater mucosal blood flow. All of these effects are beneficial, but can be assured only if the nasogastric or orogastric tube is supervised and regularly checked for blockage. None of the benefits of gastric decompression are achieved if the nasogastric tube becomes blocked or displaced.

Pretransport stabilisation should ensure that the baby does not start on a journey with a fluid deficit. Nonetheless, fluid replacement during transport remains essential to replace ongoing losses, particularly nasogastric losses, and to provide maintenance fluid for the duration of the journey. Isotonic fluid losses should be replaced with isotonic fluid, not paediatric maintenance fluids. Modified Ringer’s lactate is a reasonable choice. It is overly optimistic to expect a needle to remain in a vein during transport, and fluid will inevitably extravasate if this is attempted. An intravenous cannula should be placed at a site that is accessible for checking during transport.

The amount of fluid required to correct a preexisting deficit can be answered simply. Give enough! “Enough” means that the baby has well-perfused peripheries, adequate urine output (at least 1 ml/kg per hour) signifying adequate renal perfusion, and appropriate cerebral function. If this has not been achieved, “enough” has not been given. Resuscitation is best achieved by aliquots of 10 ml/kg of Ringer’s lactate repeated until the desired clinical parameters have been met. Additionally, patients will require maintenance fluids determined by their age and degree of prematurity.

Oxygen administration by mask or by head box supports the baby through any respiratory embarrassment and improves oxygen delivery, particularly to the bowel. Along with the increased mucusal flow following gastric decompression, oxygen administration may play a role in reducing bacterial translocation through a compromised mucosa.

Stabilisation is the sine qua non of transportation. If a baby cannot be resuscitated in a primary care facility, it is unlikely that this can be achieved in a moving vehicle. Stabilisation includes restoration of circulating blood volume with an appropriate fluid, ensuring that there is a clear and sustainable airway, treating hypoglycaemia, and ensuring that the patient is warm.

Whenever possible, the child should be escorted by the most experienced staff available. Successfully supervising the care of a surgically ill neonate during transport is the pinnacle of nursing achievement, and should be recognised as such by medical staff and nurse administrators. To maintain high standards of professionalism in the cramped conditions of a vehicle with a patient who, in the best of circumstances, is a nursing challenge deserves the plaudits of the entire team. Unfortunately, escorting ill babies is often left to the most junior and inexperienced staff member because this impacts the least on the functioning of the referring institution. The mother alone is never a suitable escort. Rarely do mothers have intensive care nursing experience, and the emotional turmoil of giving birth precludes the required precision and objectivity.

Documentation, including a letter of referral outlining the pre-referral progress of the patient and management provided at the primary care facility, must accompany all transported babies. This should augment the telephone discussions with the referral hospital that preceded the decision to transfer the baby. Likewise, any x-rays taken to support the diagnosis should be included with the documentation, or patients will have to endure a repeated radiological examination on arrival, which is a waste of time and money.

Mothers should accompany their babies whenever they are fit enough to do so. If they are unable to do so, it helps if a clotted specimen of the mother’s blood is made available to allow a safe matching of blood in case a transfusion becomes necessary.

The principles involved in safe neonatal transfer are outlined in Table 2.2, which presents the mnemonic TWO SIDES as a memory aid for those whose exposure to neonatal transfers is limited.

Table 2.2: Checklist for neonatal transfer.

<table>
<thead>
<tr>
<th>Tube</th>
<th>Orogastic, maintain gastric decompression; prevent aspiration; improve diaphragmatic excursion; maximise bowel perfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warmth</td>
<td>Conserve energy stores by reducing energy required for thermogenesis</td>
</tr>
<tr>
<td>Oxygen</td>
<td>Maximise O₂ delivery to the bowel, brain, myocardium</td>
</tr>
<tr>
<td>Sabilisation</td>
<td>Fluid volume restored, rewarmed, before transfer</td>
</tr>
<tr>
<td>Intravenous fluids</td>
<td>Ringer’s lactate through cannula</td>
</tr>
<tr>
<td>Documentation</td>
<td>Referral letter including pre-referral history and progress</td>
</tr>
<tr>
<td>Escort</td>
<td>The most skilled nurse or paramedic available</td>
</tr>
<tr>
<td>Specimens</td>
<td>X-Rays, mother’s clotted blood</td>
</tr>
</tbody>
</table>

Evidence-Based Research

Table 2.3 presents a study on postnatal transfer of preterm infants between hospitals.

Table 2.3: Evidence-based research.

<table>
<thead>
<tr>
<th>Title</th>
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<tbody>
<tr>
<td>Authors</td>
<td>Fowlie PW, Booth P, Skeoch CH</td>
</tr>
<tr>
<td>Institution</td>
<td>Aberdeen Maternity Hospital, Aberdeen, UK; Princess Royal Maternity Hospital, Glasgow, UK</td>
</tr>
<tr>
<td>Reference</td>
<td>BMJ 2004; 329(7471):904–906</td>
</tr>
<tr>
<td>Problem</td>
<td>Newborn infants and pregnant mothers may have to move between hospitals for appropriate care because of prematurity or the threat of preterm delivery. Sometimes this move means that the infant and family have to travel hundreds of miles.</td>
</tr>
<tr>
<td>Intervention</td>
<td>This article focuses on the postnatal transfer of preterm infants between hospitals.</td>
</tr>
<tr>
<td>Comparison/ control (quality of evidence)</td>
<td>When no regional transport service is available, medical and nursing staff from either referring or receiving units undertake the transport on an ad hoc basis. The staff will have variable experience in neonatal transport and the equipment used, and the vehicle may not be dedicated for neonatal use. Running these ad hoc teams often puts resources under strain because there will be fewer staff on site in the unit that carries out the transport.</td>
</tr>
<tr>
<td>Outcome/ effect</td>
<td>Anticipating the need for transfer early, appropriate preparation for transfer, and ongoing high-quality care during transfer are the cornerstones of good neonatal transport. To achieve this, staff need to be trained appropriately, all equipment and vehicles must be fitted out for the purpose, and lines of communication must be well established.</td>
</tr>
<tr>
<td>Historical significance/ comments</td>
<td>Some newborn infants will always need to be moved between hospitals. Neonatal transport services must be well organised and should aim to provide clinical care to a high standard. The service should be staffed by professionals trained in neonatal transport medicine and in using appropriate equipment.</td>
</tr>
</tbody>
</table>
Key Summary Points

1. A congenital surgical anomaly is usually associated with other system involvement.
2. Birth weight and gestational age affect outcomes.
3. Temperature control is pivotal.
4. Cardiovascular adaptation may be reversed during a surgical insult.
5. Neonates are obligatory nasal breathers and obligatory diaphragmatic breathers.
6. In the absence of TPN, innovative use of tubes and stomas may be necessary to provide nutrition.
7. Unrecognised hypoglycaemia will result in unnecessary neurological morbidity.
8. The neonate is immunodeficient when compared to adults.
9. All interventions must be performed aseptically. Antibiotic prescription will not make up for less than aseptic technique.
10. The principles involved in safe neonatal transfer are present in the mnemonic TWO SIDES.

References