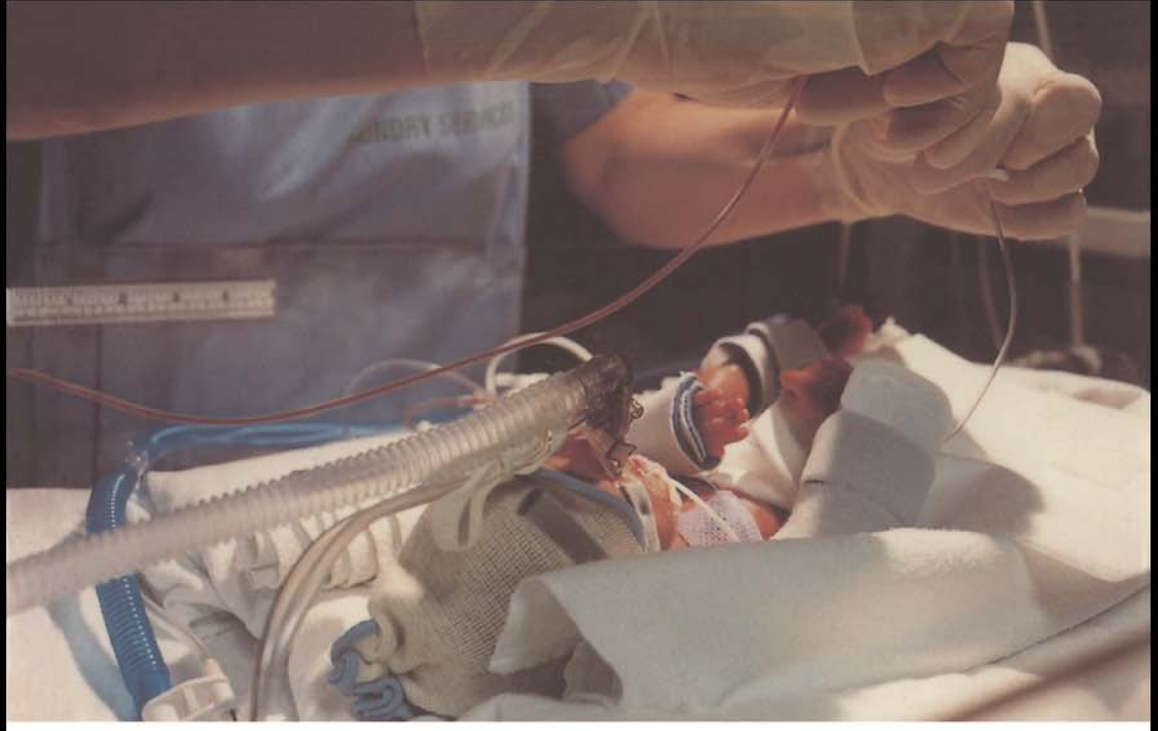

Neonatal Intensive Care Nursing

Edited by Glenys Boxwell

Neonatal Intensive Care Nursing is a comprehensive, evidence-based text for experienced neonatal nurses, paediatric nurses and midwives caring for sick newborn babies. Written largely by and for nurses, it concentrates on the common problems occurring within the neonatal intensive care unit. This user-friendly text will enable nurses to recognise, rationalise and remedy these problems using both a multi-systems and an evidence-based approach. It will be essential reading for experienced nurses and midwives caring for sick newborn babies within the intensive care area of the neonatal unit and also for nurses undertaking qualifications in the specialism of neonatal nursing.

Individual chapters include:

ADVANCED NEONATAL NURSING PRACTICE • DEVELOPMENTALLY FOCUSED NURSING CARE • RESUSCITATION OF THE NEWBORN • MANAGEMENT OF THERMAL STABILITY • MANAGEMENT OF RESPIRATORY DISORDERS • CARDIOVASCULAR MANAGEMENT • BRAIN INJURY IN THE PREMATURE INFANT • HAEMATOLOGICAL PROBLEMS • PAIN AND COMFORT IN NEONATAL INTENSIVE CARE • FLUID AND ELECTROLYTE BALANCE • NUTRITIONAL MANAGEMENT OF THE INFANT IN THE NICU • NEONATAL INFECTION • DIAGNOSTIC AND THERAPEUTIC PROCEDURES • NEONATAL ANAESTHESIA • SURGICAL ASPECTS OF NEONATAL INTENSIVE CARE NURSING • NEONATAL TRANSPORT • FAMILY SUPPORT • ETHICS AND NEONATAL NURSING • MEDICATION IN THE NEWBORN

Features of *Neonatal Intensive Care Nursing:*

- clearly written largely by practising neonatal nurses and teachers
- well sign-posted and user-friendly with a glossary of terms
- includes case studies and exercises to promote critical thinking and decision-making
- contains a wealth of evidence to support nursing procedures and practice

The editor: Glenys Boxwell is a Senior Lecturer in Neonatal Nursing and Course Coordinator at Homerton College School of Health Studies, Cambridge.

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Preface

With the increase in technological interventions and a deeper understanding of processes that can affect the developing fetus, it is hardly surprising that intensive care delivery around the time of birth is increasing. Not only do newborn infants, potentially, get sicker quicker than any other category of patient, they are also becoming increasingly premature. Within this context there has arisen an expectation by both the public and professionals that the survival of extremely sick or premature infants is the norm.

Neonatal intensive care nurses play a key role in ensuring that the best possible care is delivered to this vulnerable population. Working in conjunction with other professional groups, they are primary care givers and are pivotal to the successful outcome of treatment. The neonatal nurse's role has become increasingly multifaceted and demanding and, as a consequence, they are faced with a tremendous need for accurate information surrounding normal anatomical and physiological development as well as a detailed understanding of pathophysiological changes that occur in specific disease processes.

The term 'evidence-based practice' has become common parlance within the provision of health care and there is an expectation that an evidence-based approach will be used in all aspects of care delivery. The focus of this textbook is upon intensive care as it relates to common occurrences in everyday neonatal nursing situations incorporating the information and evidence currently available. Whilst, without doubt, rare cases are fascinating, they are encountered so infrequently in everyday practice that each time they occur specialist advice should (must) be taken, which usually results in the infant being transferred to a specialist centre. Unfortunately, these unexpected and infrequently encountered situations are often the impetus for practitioners to undertake further investigation and research to discover more about the unusual condition and its management. As laudable as this may be, the findings are easily forgotten following the infant's transfer, leaving little chance for the new-found knowledge to be put into practice.

Common clinical events and conditions encountered on a daily basis do not tend to generate such scrutiny, and as a consequence this can invoke a tendency to forget the reason as to *why* a particular situation has arisen. In addition, there can be a tendency of not questioning the rationale as to *why* situations are managed in a particular way. The consequence of this is that events occurring in everyday practice are often not looked at analytically as to *why* they have occurred, the focus being on how they are going to be managed. Unfortunately, this can result in a 'reflex arc' of care delivery, that being 'A (this situation) has occurred so B and C (management) will follow'. Sadly, this can then further crystallise into a 'this is how we do it here' state resulting in an anecdotal approach to care delivery, not one driven by evidence.

It is for this reason that this book takes a rather different approach from most neonatal textbooks, in that the contributors have not attempted to cover every aspect of care and management of infants within the neonatal unit setting. Its focus is upon intensive care and the common occurrences that are present within everyday neonatal nursing situations. Many nurses reading this book will encounter much of what it contains every working day, and all readers will encounter the clinical situations described at some point The aim of the text is to highlight why situations and conditions may have occurred incorporating embryological and developmental contributory factors, as well as a physiological perspective, so that nursing actions and interventions can be based on the information gained. The nursing management of any particular situation is not presented in a detailed prescriptive fashion, neither are protocols for practice included as these will vary from unit to unit and between individual nurses assessing specific individual infant needs. Rather, it is intended that each chapter should present information that will enable the nurse practitioner to make rational decisions in order to initiate and implement nursing interventions more effectively. Each chapter contains a wealth of referenced material that can be applied in everyday clinical practice. All of the contributors are actively employed within a neonatal care setting, and whilst none has taken a 'back to basics' approach to their subjects, all have taken a step back to take a detailed look at commonly occurring situations in order to quantify and qualify practice.

Chapters contain one or more brief case scenarios or activities in which a critical thinking process can be exercised with regard to a particular situation. 'Answers' are not given *per se,* but the solutions to the problems are contained within the chapter so that readers can come to their own conclusions in each case, encouraging an evidence-based approach to care delivery, rather than one that is task- or anecdotally oriented.

As infants do not exist in isolation, the family unit within the intensive care situation is a major consideration and consequently all of the chapters should be read in conjunction with the chapter specific to family care, which focuses on supporting families through their experiences. Likewise, developmental care and ethical issues need specific consideration in the context of each of the system-specific chapters.

It is hoped that *Neonatal Intensive Care Nursing* will actively encourage the process of linking the evidence-based theory to the everyday practice of nursing sick infants and provide a stimulus for challenging current care and management strategies.

G.B.

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Abbreviations

ASD	atrial septal defect
BPD	bronchopulmonary dysplasia
CDH	congenital diaphragmatic hernia
CHARGE	Coloboma, Heart, Atresia choanal, Retardation (growth), Genital and Ear anomalies
CHD	congenital heart disease
CLD	chronic lung disease
CMV	cytomegalovirus
CNEP	continuous negative expanding pressure
CONS	coagulase negative staphylococcus
CPAP	continuous positive airways pressure
CRP	C-reactive protein
CSF	cerebrospinal fluid
DCT	direct Coomb's test
DDH	developmental dysplasia of the hip (formerly CDH—congenital dysplasia of the hip)
DIC	disseminated intravascular coagulation
DSVNI	Distress Scale for Ventilated Newborn Infants
EBM	expressed breast milk
ECF	extracellular fluid
ECG	electrocardiogram
ECMO	extracorporeal membrane oxygenation
EEG	electroencephalogram
FBM	fetal breathing movements
FiO_2	fractional inspired oxygen concentration
GBS	group B beta-haemolytic streptococcus
GCSF	granulocyte colony stimulating factor
GERD	gastro-(o)esophageal reflux disease
GFR	glomerular filtration rate
HDN	haemorrhagic disease of the newborn; also haemolytic disease of the newborn

HFJV	high frequency jet ventilation
HFOV	high frequency oscillation ventilation
HFPPV	high frequency positive-pressure ventilation
HMD	hyaline membrane disease
IMV	intermittent mandatory ventilation
IPPV	intermittent positive-pressure ventilation
IUGR	intrauterine growth retardation
IVH	intraventricular haemorrhage
LBW	low birth weight
LIDS	Liverpool Infant Distress Score
LP	lumbar puncture
MAS	meconium aspiration syndrome
NBAS	Neonatal Behavioural Assessment Scale
NEC	necrotising enterocolitis
NIDCAP	Neonatal Individualised Developmental Care and Assessment Programme
NO	nitric oxide
OA	oesophageal atresia
OFC	occipito-frontal circumference
$PaCO_2$	partial pressure of carbon dioxide in arterial blood
PaO_2	partial pressure of oxygen in arterial blood
PCA	postconceptional age
PCV	packed cell volume
PDA	patent ductus arteriosus
PEEP	positive end-expiratory pressure
PFO	patent foramen ovale
PHVD	post-haemorrhagic ventricular dilatation
PIE	pulmonary interstitial emphysema
PIP	peak inflation pressure
PPHN	persistent pulmonary hypertension of the newborn
PROM	premature rupture of membranes
PTV	patient-triggered ventilation
PVH	periventricular haemorrhage
PVL	periventricular leukomalacia
PVR	pulmonary vascular resistance
RDS	respiratory distress syndrome
ROP	retinopathy of prematurity
SaO_2	saturation of haemoglobin (oxygen)
SGA	small for gestational age
SIDS	sudden infant death syndrome
SIMV	synchronised intermittent mandatory ventilation
TAT	transanastomotic tube
TDM	therapeutic drug monitoring
TEWL	transepidermal water loss
TGA	transposition of the great arteries
TOF	tracheo-oesophageal fistula
TORCH	Toxoplasmosis, Rubella, Cytomegalovirus and Herpes

TPN	total parenteral nutrition
UAC	umbilical arterial catheter
UVC	umbilical venous catheter
VATER	Vertebral, Anal, Tracheal, (O)Esophageal and Renal anomalies
VACTERL	as above, plus Cardiac and Limb anomalies
VKDB	vitamin K deficient bleeding

Glossary

Selected terms are highlighted in bold type in the text at their first occurrence or in key contexts

abduction to move (a limb) away from the midline of the body
adduction to draw (a limb) into the midline of the body
adenosine triphosphate (ATP) organic molecule in body cells responsible for storage and release of energy
amniocentesis removal of amniotic fluid via the maternal abdominal wall for fetal diagnostic purposes
anabolism building phase of metabolism
anastomosis a union between two structures
antecubital in front of the elbow
anterior towards the front of the body
anteroposterior an X-ray view taken using a vertical beam with the patient placed in a supine position
antibody protein released by plasma cells in response to an antigen
antigen substance recognised as foreign by the immune system
apoptosis programmed cell death
aspiration fluid entering the lungs
atelectasis alveolar collapse
atresia a blind ended tube
autoregulation the automatic adjustment of blood flow to a particular body area in response to current need
auscultation the process of listening with a stethoscope
B cells cells responsible for humoral (antibody mediated) immunity
baroreceptors receptors stimulated by pressure change
bradyarrhythmias slow heart, usually due to extracardiac pathology
bradycardia slow heart rate, less than 80 bpm (term) and 100 bpm (preterm)
brown adipose tissue specialised, strategically placed tissue (fat) which is capable of generating heat calcaneus heel bone

cardiac output amount of blood pumped from ventricles in one minute
carina the keel-shaped cartilage at the bifurcation of the trachea into the two main bronchi
catecholamines compounds that have the effect of sympathetic nerve stimulation
caudal relating to the tail end of the body
cell mediated immunity immunity conferred by activated T cells
cephalad towards the head
cephalhaematoma collection of blood beneath the periosteum of a skull bone
chemoreceptors receptors sensitive to chemical change
choroid plexus CSF producing a capillary 'knot' within a brain ventricle
chromatic related to structures within the cell nucleus which carry hereditary (genetic) factors
cytochrome iron containing proteins found on inner mitochondrial layer which function as electron carriers during oxidative phosphorylation
diastole relaxation phase of the cardiac cycle
distal further from the attached limb or the origin of a structure, e.g. the elbow is distal to the shoulder
dorsiflexed backward flexion of the hand or foot
dorsum the upper or posterior surface of a part of the body
ecchymoses discoloured patch resulting from escape of blood into the tissues just under the skin
embolus(i) obstruction of a blood vessel by particulate matter, e.g. blood clot or air
erythropoetin hormone released predominantly by the kidney which stimulates red blood cell production (erythropoesis)
evidence-based (care) the integration of best available clinical evidence with an individual's expertise
extravasation leakage of fluid from a vessel into the surrounding tissue
facilitated tucking supported positioning of a baby to contain a limb
fistula unnatural connection between two structures or body cavities
flexed to curl inwards
fundoplication surgical procedure in which the proximal stomach is wrapped around the distal oesophagus to prevent reflux
gestational age period of time from the first date of last normal menstrual period to the date of birth. Expressed in number of completed weeks or days
gluconeogenesis formation of glucose from a non-carbohydrate source, e.g. muscle
glycogenesis formation of glycogen from glucose
glycogenolysis breakdown of glycogen to glucose
glycolysis breakdown of glucose to pyruvate
haemolysis rupture of red blood cells
Heimlich valve a one-way blow-off valve
histamine chemical substance which promotes vasodilatation and capillary permeability
holistic encompassing all aspects of care
homeostasis a state of equilibrium within the body
humeral immunity immunity conferred by antibody production

hydrocephalus an abnormal increase in the amount of cerebral spinal fluid within the ventricles of the brain
immunoglobulins antibodies that bind to specific antigens
inferior away from the head or towards the lower body or structures
interferon chemical that provides some protection against a virus
intrathecal within the subarachnoid space
isotonic fluids that have the same osmotic pressure
kernicterus yellow staining of brain stem, cerebellum and hypocanthus with toxic degeneration of nerve cells due to hyperbilirubinaemia
lateral away from the midline of the body
lateral decubitus lying on one side
lipophilic substance attracted to fatty tissues
loculated collected in defined areas or pockets
low birthweight (**LBW**) infant of less than or equal to 2499 g
extremely low birthweight infant of less than or equal to 999 g
very low birthweight (**VLBW**) infant of less than or equal to 1499 g see also **small for gestational age**
lymphocytes white blood cells arising from bone marrow denoted **T** or **B cells**
macrophages principal phagocytes found at specific sites or within bloodstream
malrotation anomaly of fetal intestinal rotation and fixation resulting in intestinal obstruction
medial towards the midline of the body
mitochondria organelles found in all cells responsible for production of adenosine triphosophate
myelination the formation of a fatty insulating sheath surrounding most nerve fibres
nociception the perception by the nerve centres of painful stimulation. The term used in relation to pain perception in neonates
nosocomial an infection that develops within the hospital environment
oligohydramnios reduction in liquor volume
opisthotonus severe contraction of the back muscles causing the body to arch backwards
petechiae small haemorrhages in the skin
phagocytes white blood cells (leucocytes) that destroy pathogens by engulfment
pharmacodynamics how drugs affect the body
pharmacokinetics absorption, distribution, metabolism, excretion or what the body does to a drug
plantar relating to the sole of the foot
pleural effusion the presence of fluid in the pleural space
pneumothorax air in the pleural cavity
polyhydramnios an excess of amniotic fluid
postconceptional age current age calculated from date of conception
posterior towards the back of the body
preterm see under **term**
primiparous pregnant for the first time
prokinetic agent that increases gastric motility

proximal closer to the body or origin of a structure, e.g. the knee is proximal to the ankle
recidivity collapsing
small for gestational age (SGA) infant with birthweight less than the 10th percentile
stenosis an abnormal narrowing
stroke volume amount of blood pumped from the ventricles with each contraction
superior towards the head or upper part of the body or a structure
suprapubic above the symphysis pubis
syncope fainting, loss of consciousness
systole contraction phase of the cardiac cycle
T cells cells responsible for cell-mediated immunity
tachycardia heart rate greater than 160 bpm (term) and 180 bpm (preterm) at rest
tension pneumothorax the valve effect of a breach in the pleura allowing air into the pleural space on inspiration but not out on expiration
term from 37 to 42 completed weeks of gestation (259–293 days)
preterm less than 37 completed weeks of gestation (<259 days). Accounts for 8 per cent of births
thermogenesis production of heat
thoracic vertebrae the twelve bones of the backbone to which the ribs are attached
thrombus a clot that develops and persists within a blood vessel
turbidity clouded with a suspension of particles
vallecula a depression in an organ (beneath the epiglottis)
xiphisternum the lower part of the breastbone

Chapter 1

Advanced Neonatal Nursing Practice

chapter 1

Contents

Introduction

As health care provision evolves and changes, so too must the providers of care (Reed and Procter 1993). Within the specialty of neonatal intensive care, technological improvements have resulted in decreased neonatal mortality but increased neonatal morbidity (Williams 1995). As neonatal nursing becomes more technical, neonatal nurses have had to adapt their care provision accordingly. The first part of this chapter examines some of the key factors influencing the development of the role of the neonatal nurse. This includes the introduction of the Advanced Neonatal Nurse Practitioner (ANNP) into the skill mix as one potential means of strengthening the professional development of the neonatal nurse whilst remaining in clinical practice. Some of the legal considerations surrounding the introduction of ANNPs into the skill mix will be explored. As the health service moves more and more towards the notion of evidence-based practice, it is imperative that neonatal nurses are educationally prepared to develop their skills in providing evidence-based care. Being active and critical consumers of research is one important way of ensuring this (NHS Executive, 1996). The final part of this chapter analyses the broader concept of evidence-based practice and explores the contributions that neonatal nurses and parents can make to the provision of evidence-based neonatal care.

Factors influencing the development of the neonatal nurse role

Nursing has evolved through several philosophical eras. Elliot (1995) eloquently describes the move from the asceticism of Florence Nightingale's era, whereby the nurse was entrenched in the values of self-sacrifice and self-denial, through to the task-orientated era of the periods surrounding the First and Second World Wars. Task-orientated care, as observed by Ford and Walsh (1994), resulted in ritualistic practice, a far cry from today's notion of evidence-based practice. During the 1960s, hospitals, recognising the need for post-basic education, set up 'in-house' courses to equip nurses with the necessary skills for varying specialties. These courses tended to be task-orientated, were not transferable and had no academic recognition (Scott 1998). The development of the nursing process, with its philosophy of humanistic existentialism, in the 1970s aimed to replace the task-orientated, traditional model of care and foster the ethos of individual rights and informed choice (Elliot 1995). In neonatal nursing practice this was manifested by the introduction of family-centred care nursing models (Casey 1988). The Joint Board of Clinical Nursing Studies set up post-registration specialty courses, including ones for neonatal intensive care, in an attempt to standardise post-registration nurse training and discourage the previous trend of 'in-house' training (Scott 1998). In 1979 the United Kingdom Central Council (UKCC) replaced the General Nursing Council (GNC) and established the four National Boards. The UKCC's main function was to establish and improve standards of training in post-registration

as well as pre-registration education (the GNC's remit had been primarily concerned with pre-registration education). Nursing was also by this time attempting to become a graduate profession in its own right and shed its medical model of practice (O'Brien and Watson 1993). During the 1970s and 1980s more attention was focused on the provision of post-registration courses such as the ENB 400, 402, 405, 409 and 904 courses, these educational programmes being considered as the basic requirements for nurses wishing to develop clinical and theoretical expertise in neonatal care. One of the driving forces for this initiative was the identification of a lack of nurses qualified in specialty (NHS Management Board 1989).

Most universities are now offering the ENB 405 course at degree level in keeping with consumer demand, as more and more nurses enter the specialty as a diplomate. Today's neonatal nurses not only requires a professional qualification (such as the ENB 405/904) but also evidence of academic ability if they are to progress in their professional development. The UKCC's framework for post-registration education and practice supports the notion of approved programmes to create specialist nurse practitioners and advanced nurse practitioners, although there is still debate as to what academic level and preparation is required to achieve this status (Scott 1998). The UKCC decided not to set academic standards for advanced practice but stated that they 'fully support the notion of advancing practice' (UKCC 1994). They recognised that there is confusion surrounding titles, roles and responsibilities and state that advanced practice is not about tasks but a broader concept of nursing, midwifery and health visiting practice and is particularly concerned with advancing the practice of others. The latter statement would suggest that advanced practitioners are also to play a major role in teaching in the clinical area (UKCC 1994). In order to provide an advanced level of nursing practice, it is essential for nurses to have the appropriate theoretical knowledge to underpin their practice, but what is an appropriate level of theoretical knowledge? Literature suggests that the academic level for registration of specialist practice should be at degree level and advanced practice should be at Master's level (Ball 1997; Gibbon and Luker 1995). This brings about the debate of whether or not nurses as professionals feel it is necessary to have a Master's degree or Doctorate in order to advance nursing practice. It must be remembered that this is not only an educational debate, but also an ethical one, because nurses have a duty to provide a standard of care commensurate with their knowledge base (UKCC 1992). Oldnall (1995) warns that if nursing wishes to be recognised as an academic, practice-based profession it should establish what is unique to nursing and not depend entirely on other disciplines such as medicine and physiology to generate knowledge. This is especially relevant in specialties like neonatal intensive care, where there is potential for a 'medical model' of nursing (Bradshaw 1998). Castledine (1995) sums up the debate succinctly by reinforcing the point that nursing is not second-class medicine but first-class health care. In her discussion on how nursing roles evolve and change, Frost (1998) argues that advancing nursing practice should be primarily concerned with leadership, developing decision-making skills and adapting nursing practice to improve health outcomes for the patients they

serve. She warns of the trap that professionals fall into of assuming that advanced practice is about the acquisition of narrow skills and therefore linked with 'high-tech' areas of practice. The introduction of ANNPs into the skill mix has caused controversy, as some authors (e.g. Yeo 1998) view the role as medical and task-orientated. However, Casselden (1995), whilst acknowledging that her role is medically-orientated, describes how she is able to draw on her nursing skills and experience to provide a holistic approach to care. Walsh (1999), in a study comparing nurses and nurse practitioners, found that nurses rated the technical aspects of their work higher than the nurse practitioners. The nurse practitioners placed more emphasis on the importance of psychosocial aspects of care, thus contradicting the view that nurse practitioners tend towards the medical model and are becoming 'mini-doctors'. The preparation for the role of ANNP appears to be an important factor in determining to what extent nurses retain their nursing identity. Beal *et al.* (1996), in their study on how neonatal nurse practitioners (NNPs) perceived their identity, found that the NNPs educated at Master's level who were preceptored by nurses were more likely to retain their nursing identity than those prepared at Certificate level. The authors suggest that the role does not need to have a medical orientation and that the medical and technical aspects should be incorporated into the nurse's role. It is essential that nurses do not lose their identity and undermine the values of the nursing profession in their eagerness to expand areas of practice.

The developmental history of the Advanced Neonatal Nurse Practitioner role

The nurse practitioner role originated in the USA in the 1960s in response to increased awareness of inequalities in access to health care. The aim of the first demonstration project was to determine the safety and efficacy of nursing practice specifically designed to improve the health care of children and their families (Snyder and Mirr 1995). These nurses were known as 'practitioners of nursing'. They had undergone 24 months of training to improve their knowledge and skills relating to child care. Over the next 30 years the role expanded to include care of other client groups. Neonatal Nurse Practitioners became popular in America during the 1970s and 1980s. Their popularity was prompted by the increased use of technology in neonatal care in conjunction with a recognised shortage of appropriately qualified medical personnel. It must be mentioned that the motivation was also financially driven to reduce medical costs (Modica *et al.* 1991). Initial studies undertaken at this time (for example Martin *et al.* 1985) suggested that total care delivered by neonatal practitioners was of similar quality to that provided by physicians. In the UK, it was not until the early 1990s that the role of the ANNP really became established. Wessex Regional Health Authority was the first to propose a regional training programme for neonatal nurses. This programme was validated by the English National Board and Southampton University and became known as the ENB A19 Course. As Doherty (1996) points out, it is

unfortunate that the ENB award was entitled 'Advanced Practice', as at this time the UKCC had stated that advanced practice was considered to be at Master's level. The 'Wessex model', as it became known, became the benchmark for ENB A19 programmes. Since the Wessex initiative, several more institutions in the UK have developed formal programmes for either advanced neonatal practice, specialist practice or enhanced neonatal practice and it is here that the confusion exists. Salussolia (1997) provides an interesting dialogue about the confusion with differing titles used to describe levels of nursing practice and Frost (1998) suggests that the issue is not about what titles professionals use but what frameworks are established to differentiate between roles and competencies. Perhaps as Rolfe (1998) suggests, an alternative would be to refer to 'advancing practice' rather than 'the advanced practitioner role', as the latter is suggestive of a post or grade as opposed to a philosophy of practice which all neonatal nurses, with adequate preparation, could work towards. This begs the question of what constitutes 'adequate preparation', as the neonatal nurse is confronted with a choice of formal courses such as those approved by the ENB, which carry academic accreditation or the alternative (and it has to be stated, cheaper) option of 'inhouse' training. Whatever the decision made, it is essential to take cognisance of the legal implications of advanced practice.

Legal aspects of advanced neonatal nursing practice

Inevitably, advancing neonatal nursing practice involves undertaking work previously done by doctors, such as task-orientated work (see Chapter 13), clinical decision-making and communicating with parents. Such an increase in professional responsibility also leads to a commensurate increase in legal responsibility (Tingle 1996). Whilst *The Scope of Professional Practice* (UKCC 1992) clearly states that nurses should not undertake any skill they have not been adequately prepared to carry out, Dimond (1995) warns that the inexperienced do not always know their limitations, and suggests that the *Scope of Professional Practice* document is not prescriptive enough, as the determination of competence is left very much to the individual practitioner. To add further to the dilemma, Tingle (1997) describes the conflict between the General Medical Council (GMC) statement on the professional duties of doctors, the British Medical Association (BMA) guidelines on medical procedures and the UKCC (1992) document. The GMC and BMA are in agreement that the doctor must be the delegator and manager of care and it is the doctor who decides whether the nurse practitioner can undertake medical procedures. This is in direct contrast with the UKCC document which seems to assume a degree of nursing autonomy and power which is not, as stated previously, reflected in medical guidelines. To date, this dilemma has not been resolved, but there is every indication that a rewrite of guidelines for practice for both nursing and medical staff is imminent. What both professions agree on, however, is that the patient (or in the case

of the neonate, the parents) should be fully informed as to who is undertaking any procedure or communicating with them. Until this 'grey area' is resolved, neonatal nurses wishing to advance their practice should do so with caution and ensure that they have been fully prepared to undertake any expansion of their role. There is always the potential danger of being coaxed and cajoled into undertaking medical procedures, especially if one's ego is massaged! On a more optimistic note, there are several ways in which neonatal nurses can advance their practice, as the following section illustrates.

Potential areas in neonatal care for advancing neonatal nursing practice

In intensive care and high dependency care areas, some ANNPs are currently working on rotas with Senior House Officers. Critics of this model have suggested that it is impossible to provide holistic care as the ANNP may be called to insert intravenous lines or attend the delivery suite to resuscitate a baby at any time, therefore holistic care is not possible. There is also debate about how much nurse practitioners are able to advance their role in an intensive care area without losing sight of their nursing background. Albarran *et al.* (1998) warn of the danger of merely taking on discarded medical skills, suggesting that this is not advancing nursing practice. Casselden (1995), as mentioned previously, describes an eclectic role for the ANNP working on the SHO rota as well as being unrostered to take part in midwifery, nursing and medical staff training. Oliver and Allan (1998) describe a neonatal unit providing low dependency care which is led by ANNPs. The unit is in daily contact with the Regional Centre but the ANNPs have primary responsibility, caring for babies requiring resuscitation and stabilisation at birth and monitoring the progress of 'well' pre-term babies on the unit. Care after discharge from hospital is also a potential area for role development, especially as more and more babies are being discharged into the community earlier, some still requiring oxygen. In the USA, there is evidence to suggest a shift to community-based neonatal care. Strodtbeck *et al.* (1998), in their analysis of the role of the American equivalent of the neonatal nurse practitioner in the twenty-first century, describe the current downsizing of neonatal intensive care units and explore new opportunities available for experienced nurses in home care. They emphasise the importance of preparation for such practice such as the ability to provide research-based, quality care. Kenner *et al.* (1998) likewise illustrate how the neonatal nurse practitioner could practise in varying settings, such as high-risk follow-up clinics, and could work with child development follow-up teams. Whilst acknowledging the differences between the two countries with regards to health care provision and nurse education, this is certainly an area which the UK nurse practitioners could move towards as an alternative to the hospital setting. For example, ANNPs could be at the forefront in setting up high-risk follow-up clinics and establishing regular resuscitation-training sessions for parents and other interested parties. This innovation would complement the role of the community liaison neonatal nurse. As the role of

the neonatal nurse develops, so too must their knowledge base in order to ensure that practice is rooted in sound learning, hence the importance of developing and providing evidence-based care.

Evidence-based neonatal care

Walsh (1998) suggests that evidence-based nursing originates from the notion that effective clinical decision-making requires the accumulation of appropriate evidence. The government's determination to ensure that treatment is based on the best evidence of what does and does not work in clinical practice has generated a flurry of activity, such as the setting up of national bodies to ensure rigorous evaluation of health care practices (Nolan *et al.* 1998). The notion of 'evidence-based practice' has been interpreted as a rigorous, scientific evaluation of health care practices and unfortunately has tended to ignore knowledge accrued over the years by experiential learning and reflexive practice that many experienced nurses have to offer (Rolfe 1998). The next section will examine the notion of evidence-based neonatal care by exploring two fundamental questions:

- On what knowledge do neonatal nurses base their practice?
- How best may neonatal nurses be equipped to provide evidence-based care?

On what knowledge do neonatal nurses base their practice?

Whilst recognising that medical expertise and scientific knowledge is a crucial aspect of neonatal intensive care, it is important to remember that it is not the only form of evidence on which clinical practice should be based. Neonatal nurses and parents themselves have a vast amount of experience to bring to any given clinical situation. Clinical evidence should not consist entirely of findings from 'rigorous, objective, scientific enquiry', as suggested by the Department of Health (1996), but also of parents' lived experiences and neonatal nurses' reflections in and on practice (Eraut 1994). Neonatal nurses and parents may contribute to the notion of evidence-based practice without relying solely on the medical model definition of what constitutes evidence. For example, one of the ways in which evidence-based health care can be fostered includes utilising parental expertise in evaluating health care provision. Parents' lived experiences of having a baby in a neonatal intensive care unit can provide invaluable evidence of the quality of care provision. Price (1993) studied parents' perspectives of quality nursing care in the neonatal unit. Price refers to the paucity of literature concerning parents' involvement in care provision. Utilising a qualitative methodology, she demonstrates how 'non-technical' aspects of care, such as comforting babies after painful procedures, are of equal importance to parents as 'technical aspects', which are taken for granted in the neonatal intensive unit. Avis (1997) suggests that parents' views about care should be an integral part of any clinical audit. Fitzpatrick and

White (1997) argue that the top-down paternalism, common in health care evaluation, trivialises consumers' views on health care provision by focusing on hotel aspects of care and communication issues, whilst failing to ascertain views on the appropriateness and effectiveness of care provided. As neonatal nurses are the babies' and the families' advocates, they play a key role in gathering data on how they perceive the quality of care provision. For example, how many neonatal nurses ask parents directly their views on the treatment their baby is receiving? There can sometimes be a tendency to assume that the parents (unless of course they are health care professionals themselves) know little or nothing about the technical aspects of neonatal intensive care, so the neonatal nurse may make judgements about what parents are thinking without actually asking their opinions. Raines (1996) describes how neonatal nurses, having now learned technical skills, must focus on the importance of parental wishes and not assume that they will be grateful for all the nursing staff do with regard to intensive care treatment. Auditing parents' views on management of care is the first step in ensuring that care of the family is evidence-based. Clinical governance, with its emphasis on having effective and efficient systems of communication with health care professionals and patients, will be a major contribution to achieving collaboration between professionals and consumers of health care (McSherry and Haddock 1999).

In his analysis of types of knowledge, Rolfe (1998) describes not only scientific knowledge but also experiential knowledge and personal knowledge. Experiential knowledge is knowledge devised from paradigm case studies built up over years of experience and personal knowledge is derived from the things the practitioner knows about individual patients, such as their likes and dislikes. Whilst experiential and personal knowledge are not perceived by the Department of Health as 'scientific', Benner argues that these forms of knowledge are the hallmarks of what she describes as 'expert practice' (Benner 1984). If we accept the notion of Benner's expert practitioner then we must take cognisance of the importance of utilising these forms of knowledge in delivering evidence-based care.

How best may neonatal nurses be equipped to provide evidence-based care?

One way of equipping neonatal nurses with the skills and knowledge to provide evidence-based health care is by encouraging and supporting those who wish to further develop their roles by undertaking further formal, professional post-registration qualifications, such as the ENB R23 course, 'Enhancing Neonatal Nursing Practice', the N96 'Neuro-behavioural Assessment of the Newborn' and the ENB A19 course, 'Advanced Neonatal Nursing Practice'. The plethora of titles given to post-basic courses appears somewhat daunting to the neophyte student and controversy still rages about which courses are the most suitable (Ashworth *et al.* 1998; Doherty 1996). There is evidence to suggest that neonatal nurses wish to extend their parameters of practice in line with changing health care needs (Barrett 1995), but as discussed previously, advancing neonatal nurse practice is not just about extending tasks previously confined to the medical

domain, but advancing nursing practice according to the context of care. Neenan (1997) warns of a potential loss of direction for nurses if their work becomes too task-orientated, resulting in a lack of continuity of bedside care, and adds that there is prolific evidence citing the importance of highly skilled nurses to act as role models to advance nursing practice. In the final analysis, it is more important how the individual neonatal nurse utilises a qualification and not which one he or she obtains. McDougall (1994) and Oliver and Allan (1998) illustrate how ANNPs may be integrated into the skill mix in neonatal units. Perhaps a future role for ANNPs could be to take the lead in promoting evidence-based practice by becoming actively involved with the utilisation of nursing research findings in policy writing and instigating nursing research in the clinical area. Rolfe (1998) suggests that one of the characteristics of advanced practice is the ability to reflect in and on practice. Clinical supervision provides an excellent formal forum for developing such skills. If utilised appropriately, it can provide the opportunity for both personal and professional growth (Butterworth 1992). On a more informal level, neonatal staff could organise forums within the working day to allow for such reflection to occur and perhaps lobby their managers to encourage study leave to prepare for such reflective days. The dissemination of such experiential knowledge is as valuable in promoting evidence-based practice as scientific research, especially if parents are also involved.

Langley (1997), in her review of the impact research has had on nursing practice, states that, unlike their American counterparts, neonatal nurses in the UK have been slow to instigate research findings and generate nursing research. She argues that this is partly due to the fact that nurses have, until recently, lacked the academic ability to undertake research. As a consequence of this, most neonatal nursing practice is underpinned by medical research and many nursing policies and procedures are written primarily by medical staff with a token amount of input from neonatal nurses. This state of affairs may be partly understood if it is placed within the historical context of neonatal nursing. In health care provision, and especially in high-technology areas such as neonatal intensive care, medical expertise still dominates care provision. Walby *et al.* (1994) describe how medical knowledge is perceived as focusing on the diagnosis and treatment of patients whilst nursing knowledge is confined to the caring role. The professional status and scientific knowledge of the doctor is viewed as being superior and of a higher priority than that of the nurse within the organisational structure. As a result of this, nurses have lacked the support and motivation to generate new knowledge, thereby continuing the cycle of a deficit of nursing research. English and Bond (1998) describe the problems of nurses wishing to undertake qualitative research studies and applying for funding to research funding bodies favouring quantitative, scientific methodologies. Not only is there a lack of research undertaken by neonatal nurses, but there is evidence to suggest that nurses in general are unable to analyse research findings critically (Pearcey 1995). Pearcey, however, identifies that research activity is more evident in younger nurses and nurses with higher academic qualifications, so with the current emphasis on achieving academic status, perhaps more nurses will acquire these skills. With more nurses entering post-registration degree programmes that require the undertaking of either a literature review, case

study analysis or small-scale research study, it is a shame that many excellent assignments are consigned to gather dust on shelves and their findings are never disseminated to nursing colleagues. On a more positive note, however, Langley (1997) highlights some of the successes in neonatal nursing research and illustrates how such research can influence neonatal nursing practice, citing examples of research conducted by familiar names such as Boxall, Carter and Lang (Langley 1997).

Conclusion

It is only by reflecting on the development of the role of the neonatal nurse that we can see just how far it has evolved over recent years. There are plenty of exciting prospects in the future, such as the government's recent proposals for Nurse Consultants and the moves in education towards collaborative learning with medical colleagues. If neonatal nursing practice is to continue to develop and take on more responsibility and accountability, it is essential that nursing practice is based on sound evidence and clinical practice underpinned by appropriate, up-to-date theories and research generated by the nursing as well as medical profession. In order to achieve this, neonatal nurses should continue to develop analytical skills and further develop skills as critical consumers of research. Whilst recognising that there is a paucity of research undertaken by neonatal nurses, which needs to be redressed, it is important to acknowledge other available forms of evidence that can also inform practice. Parents can make a valuable contribution to the provision of evidence-based practice, and their views and opinions should not be ignored. Neonatal nurses have a wealth of experiential and personal knowledge which should be utilised and disseminated amongst their peers. The remaining chapters of this book are the first step in achieving this important goal.

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chapter 2

Chapter 2

Developmentally Focused Nursing Care

Contents

Introduction

The term 'developmental care' is used to describe those interventions which support and facilitate the stabilisation, recovery and development of infants and families undergoing intensive care, and beyond, in an effort to promote optimal outcome. The theoretical basis for our current understanding begins with the wide body of evidence that demonstrates that **low birth weight** (LBW) infants are at increased risk of medical complications, growth deficits, neurological impairment and a range of more subtle cognitive and behavioural difficulties (Fasting 1995; Hack *et al.* 1995; Powls *et al.* 1996; Tin *et al.* 1997). Whilst the incidence of major handicap is reducing and the survival of **very low birth weight** (VLBW) infants is steadily improving, there remains an urgent need to explore strategies which have the potential to ameliorate the side-effects of treatments necessary for life support.

During the last trimester of pregnancy, the fetal brain develops at a greater rate than at any other stage of life. This critical growth process consists of neuronal migration, synaptogenesis and arborisation, and myelinisation (Volpe 1995). Healthy brain development depends upon subtle influences from the ecologically appropriate uterine experience, therefore the prematurely extrauterine fetal infant is vulnerable to abnormal influences from the neonatal intensive care unit (NICU) environment. This frequently results in poorly modulated behaviours in the autonomic, motor and state systems which manifest themselves through unstable, delayed or abnormal systems development. Illness and extreme prematurity exacerbate the infant's vulnerabilities, however deficits in neurobehavioural performance at term age are noted in healthy preterm infants as well as those requiring prolonged intensive care (*Huppi et al.* 1996).

Irrespective of illness and treatments, the environment of care is itself a source of stress for preterm infants and their families, who are particularly vulnerable to the adverse conditions facing them (Miles *et al.* 1991; Gardner *et al.* 1993; Padden and Glenn 1997). Parent-infant relationships can be influenced by supportive interventions which will have an impact on outcome beyond the events of the neonatal period, and which could be seen as crucial to the success of neonatal intensive care. Thus developmental care is viewed as an expansion of neonatology in which evolving infant and family systems interface with the biological, environmental and psychoemotional risks of preterm birth.

Theories on developmentally focused care

Developmental support programmes have been guided by different theoretical models, summarised by Wolke (1991) thus:

- Programmes that counteract neonatal sensory overload or deprivation.
- Programmes that aim to prevent faulty maternal-infant attachment.
- Programmes that aim to help parents to resolve the emotional crisis of preterm delivery.

- Programmes that help parents to be more sensitive and responsive to their infant's cues and improve maternal-infant interaction.
- Compensatory programmes which are aimed at infants with developmental deficit or chronic illness.

More recently, current strategies based on the synactive theory of development (Als 1982, 1986) promote systematic assessment of infant behaviour with an increasing emphasis on parent teaching and support based on infant individuality (Als and Gilkerson 1997). A range of possible strategies and interventions are devised to meet individual need, and to support individuals' developmental goals. An important consideration within this framework is the recognition that the preterm infant is not a dysfunctional term infant but is competent for his or her stage of development within the uterine environment.

One theoretical approach views the infant in NICU as being over-stimulated compared to the natural uterine environment and proposes minimal handling and reduced sensory input, via the environment at large and the individual cotspace. Another approach views the infant as being deprived of appropriate sensory input, and additional stimulation is provided in an effort to improve outcome. Some studies have used a single intervention—auditory (maternal voice, heartbeat sounds), tactile (massage, stroking), visual (mobiles, facial presentation), vestibular (oscillating cot, waterbed, rocking)—whilst others have implemented multi-modal interventions using a combination of strategies in an effort to emulate the fetal environment. Other studies have investigated the impact of environmental manipulation, postural support, therapeutic touch, parent-infant interaction coaching and various models of psychoemotional support.

Als (1986, 1996) recommended a systematic review of behaviours in order to recognise individual infants' strengths and weaknesses and thus modify the context of care. The theoretical model suggests an emerging continuum of subsystems development, from which underlying behaviours indicate the current functioning of the subsystems. Through such assessments, an overall picture of the infant's developmental goals is constructed and care is planned and delivered to promote stability and development of the subsystems, thus reducing stress and supporting the infant's efforts. This approach to intervention requires a flexible and creative nursing team who are able to deliver individualised care according to infant cues. Such programmes have consistently demonstrated improved outcome compared to matched control groups (e.g., Mouradian and Als 1994; Als *et al.* 1994, 1996). However, some of the studies were of time lag design, which may have involved other changes in medical or nursing practice over the study period. Samples were relatively small and other variables may account for the differences, but an accumulation of small-scale studies suggests important benefits in terms of reduced severity of chronic illness, improved neuromotor outcome and reduced health care expenditure. Larger-scale studies, taking into account medical risk and the context of nursing care, are required, as such interventions require considerable additional resources. Nevertheless, developmentally focused practice development should

proceed on the basis of evaluation of infant status and the monitoring of the local population over time.

Studies of the effects of the neonatal environment have revealed many sources of hazard for the vulnerable preterm infant. Wolke (1987) undertook a systematic review of the ecology of the NICU and concluded that infants were not under- or over-stimulated but rather inappropriately stimulated. There was evidence of a mismatch between the type, intensity and patterning of stimulation and the infant's developmental status. This highlighted the need to go beyond general interventions and modifications to the environment, and look more closely at the behaviours of individual infants in an effort to meet their developmental goals. These goals will vary with individual circumstances, such as illness, treatments, gestational and chronological maturity as well as the environment of care.

Interpreting the findings from developmentally focused studies is difficult due to the variations in sample size and distribution, the wide range of interventions and the different short- and longer-term outcomes. Some studies have reported adverse reactions and others have shown encouraging short-term effects, although it has been suggested that these could be part of an observer effect (Wolke 1991), that is, benefits from the extra attention on the part of researchers or nursing staff. Although many studies are small-scale and took place before the 'surfactant era', there is nevertheless an accumulation of evidence to suggest that early developmental intervention shows immense potential to diminish the severity of the side-effects of intensive care. Individualised, tally focused care utilising systematic behavioural assessment (e.g., Als 1982, 1986, 1993, 1999; Als *et al.* 1996), due to the contingent and flexible nature of interventions, has the potential to improve outcome still further, with commensurate reduction in health care expenditure.

The majority of intervention and outcome studies in NICU are North American, therefore it is important to interpret them carefully for use in the UK system. There are numerous differences with, for example, medical resources, the organisation and delivery of nursing care, and the nature of family support. Visiting families in the North American tertiary NICU often face many additional difficulties due to the long distances from local support networks, and space for accommodating mothers and other family members is often far from optimal.

The developmental needs of preterm infants are complex, as they attempt to balance their lack of fetal experiences with the hazards of treatments necessary for survival. Their environment is highly inappropriate, as their experiences are unable to match the normal sensory requirements of the developing human brain, provided first by the uterus and secondly by the breast, with the sensitive and nurturing contact of the mother. In this sense, almost every activity associated with newborn intensive care could be described as a developmental issue.

The feto-maternal and maternal-infant relationship is also in deficit and requires consideration from a developmental perspective. Maternal role development depends upon self-esteem and mastery of natural mothering behaviours (Rubin 1984; Mercer 1985) and these are unlikely to proceed appropriately in the threatening circumstances of preterm birth and the ensuing period of intensive care (Mercer and Ferketich 1994;

Goulet *et al*. 1998; Reid 2000). Family-centred models of care have considerably improved the situation of parents, who were often excluded from caregiving and care planning decisions, but there is still much that can be done to support the development of healthy relationships which are crucial to the success of newborn intensive care and the goal of optimal outcomes in the longer term. Parental education about how to support their infant's emerging developmental agenda is increasingly regarded as an essential component of developmentally focused care. By understanding preterm infant behaviour, parents can observe more closely the individuality of their infant, become able to 'reset' their expectations and thus offer more infant-led support.

Brazelton (1973) were amongst the first to note term infants' abilities to be active participants and social partners, who affect caregiving behaviours, and to recognise that they were highly organised for their stage of development. Als (1982) observed that preterm infants, although less organised, were similarly competent for their stage of development. She proposed a dynamic model for preterm infant behavioural organisation which identified subsystems which interact with each other and are influenced by the infant's internal and external environment. This is known as the synactive theory of development (Als 1982) and this model provides an important basis with which to understand preterm infant behaviour, assess subsystems functioning and thus identify the emerging developmental goals. Observations can be made in the naturalistic setting, before, during and after a caregiving procedure. This enables nurses who offer some degree of continuity of care to deliver contingent handling based on the evidence of their observations. Developmentally focused care can therefore proceed to identify stress responses, note the sources of such stress and suggest ways in which this can be minimised. Similarly, an assessment of subsystems functioning can note the emerging competencies and developmental goals of the infant, and care may be planned in ways that support the infant's efforts, thus providing positive feedback for those efforts. Stable functioning in one subsystem will have a stabilising impact on other subsystems, thereby supporting the emergence of new developmental goals. This approach to developmental care has the advantage of being specific to the observed infant and is supportive of infant individuality. This improves the scope for facilitative parent teaching, and can have an impact on the way parents understand the individuality of their infant and how they can offer support which promotes stability and development. Parental teaching and learning opportunities are thus more specific and responsive to their infant's needs and contingent handling skills are promoted. In this way, parent-infant interaction can be facilitated by a better understanding of preterm infant behaviour.

In order to incorporate developmentally focused care, practitioners must have some knowledge of preterm infant developmental theory and be able both to assess behaviours and to interpret their meaning in the context of internal and external factors. In addition, it is essential that practitioners are able to determine and deliver an appropriate plan of care, have the resources to deliver the required strategy and the ability to evaluate its effectiveness.

This is best done through repeated systematic observations which review the functioning of the infant in the context of his or her medical status and treatments, gestation and environment of care, and could be incorporated into a general nursing assessment. With increased understanding of developmental theory and systematic behavioural assessment, the emphasis of developmental care moves towards a more individualised and holistic approach, rather than the general introduction of developmentally supportive initiatives, such as modifying the environment or the provision of positioning aids. Nevertheless, both approaches are important; there are many initiatives of a general nature which will facilitate developmental stability, but individualised assessment will highlight the emerging agenda for the infant and enable care to be delivered according to infant cues. Advocacy is a fundamental principle of nursing care, and the ability to communicate effectively with clients is an essential component. Communicating with preterm infants is particularly difficult as they are almost powerless to inform us of their needs. Greater recognition and understanding of their sources of stress and comfort can facilitate responsive and nurturing care, which will serve to strengthen the role of nurse as advocate to the most vulnerable members of society.

The following themes identified from the literature and various practical initiatives are likely to support the goal of improved medical and developmental outcomes for infants undergoing intensive care, and enhance parent-infant interaction and subsequent psychosocial relationships.

- Behavioural assessment.
- The reduction of environmental hazard.
- The promotion of contingent handling.
- Postural support.
- Family-centred, developmentally focused models of care.

Behavioural assessment

Als (1982, 1986, 1993, 1999) advocates a regular systematic behavioural assessment in order to deliver contingent care that supports the infant's developmental goals. Brazelton (1973) demonstrated the full-term infant's capacity for participation in his relationship with others, affecting his social environment and his emerging developmental agenda. This model presented the newborn's functioning as an emerging continuum of organisation with hierarchical tasks accomplished within caregiver-infant interactions. Term newborns have stable autonomic and motor systems function, and rapidly acquire stable and distinct state system organisation. Sleep and drowsy states predominate, but the term infant can achieve quiet and active alert states as well as fussy, irritable and lusty crying states. They can be readily consoled and satisfied and are able to 'shut out' stimulation for rest and sleep. Note the lack of reaction to noise of a full-term infant who is deeply asleep, or the increasingly insistent cry of the infant who needs attention.

The emerging task of the term newborn is increasing alertness, with growing differentiation and responsiveness. An active and responsive social partner is essential to support this development, which should be rewarding and stimulating for both partners, and supportive of an increasing interest in the external world. Preterm infants, in contrast, whilst driven by the similar goals of stability and organisation, are at a stage of development which is more concerned with their internal world. They have immature systems organisation, and their behaviours are consequently more diffuse and less stable. Subsystems stability develops with increasing maturity, but is also supported by caregiving and handling that is appropriate for the infant's current and emerging developmental status.

The autonomic subsystem, demonstrable through respiratory, cardiovascular and digestive function, is unlikely to have reached a level of robustness to enable stable functioning without support, and this stability is likely to be under duress when challenged by handling, environmental disturbance, pain and illness. Stability and organisation in all other subsystems depends upon autonomic stability, and in this model it can be viewed as the basis for more complex, hierarchical developmental tasks.

The motor system, demonstrable through muscle tone, posture and movements, is the second emerging subsystem. Preterm infants have reduced muscle bulk and power, and an immature central nervous system, which results in poor mobility and an inability to counteract gravity in order to support their own developmental needs. The contrast with term infants' motor function serves to remind us of some of the goals of support in the motor system of preterm infants. The term infant's flexed, rounded posture and organised, purposeful movements are in stark contrast with the preterm infant. Consider the flattened and extended posture, flaccid muscle tone, inability to move towards or away from stimuli and the tremulous and diffuse movements. Postural support, containment and the provision of self-regulatory aids to support stability and organisation in the motor system will be discussed separately.

Sleeping and waking states represent the level of maturity and intactness of the central nervous system, the infant's relationship with the external environment and his or her ability to regulate and stabilise underlying internal systems. At a gestational age of 24 weeks, cycling between sleeping and waking can be identified by EEG in some infants (Hellmstrom-Wetas *et al.* 1991) and by 28 weeks, infants clearly exhibit distinct sleeping and waking states (Holditch-Davis 1990). However, prior to 36 weeks, there is poor coordination between states, and with decreasing gestation, behaviours that demonstrate particular states are increasingly difficult to identify, even by trained observers. State transitions are diffuse, erratic and more easily influenced by internal and external stressors. Preterm newborns spend greater amounts of time in light sleep or drowsy states, and have difficulty in achieving deep sleep. Quiet alertness, when infants are socially responsive, is rarely observed in NICU conditions, and active alert states are usually generated by the caregiving regime, which often results in hyperalertness and arousal followed by systems collapse and exhaustion. Preterm infants are unable to make the ordered, organised transition between states, and state stability is easily challenged by

handling and the environment of care. Note the infant in light sleep who, upon handling, immediately becomes hyperalert and agitated with extensor postures and 'panicked' facial expression. Note also the difficulty in achieving a quiet, alert state with responsive and animated facial expression. The observer has the additional problem of being unable to detect distinct states due to the infant's immature behaviours and the hostile environment in which the infant is unlikely to give their best performance.

Sleep-wake states

There are several sleep-wake scoring systems, based on careful observations of term and preterm infants (Table 2.1). Brazelton and colleagues developed a scale to be used as part of the Neonatal Behavioural Assessment Scale (NBAS; Brazelton 1973; Brazelton and Nugent 1995). The score is used to determine predominant states, transitions between states and the quality of alertness. It has the advantage of being widely used in research and clinical practice, and is relatively easy to use due to the distinct definitions. However, owing to the small number of states described, it fails to capture the indeterminate and transitory qualities of preterm infant behavioural states.

Thoman's revised state scoring system (1990) is more sensitive, and has inter-rater and test-retest reliabilty with preterm infants (Holditch-Davis 1993). There are ten states which differentiate state-related behaviours in preterm infants and those with perinatal complications. This system is more difficult to learn and training is not readily available, but it has been shown to have predictive validity with later developmental outcome.

Anderson's behavioural state scoring system (ABSS) was devised following earlier work with preterm infants and was used to demonstrate a relationship between heart rate, energy consumption and behavioural state (Ludington 1990). Gill *et al.* (1988) used the scale to show the effects of non-nutritive sucking on behavioural state. There are few published studies using this scale, so its reliability and validity are not well established; however, it was constructed specifically for preterm infants and was based on extensive observations of their behaviours. Although similar to other scales, it is more complex as it takes into account physiological parameters, and thus is derived from a different theoretical base (Holditch-Davis 1993). It is likely to be a useful instrument for quantitative studies into the relationships between interventions and infant state, but its use as a clinical instrument is not yet clearly defined.

Als (1982) defined behavioural state in greater detail in the Assessment of Preterm Infant Behaviour (APIB), which was a modification of the NBAS (Brazelton 1973). This scale more closely describes the state-related behaviours of preterm infants in response to their internal and external environments, and can be used with infants of very low gestational ages: The scale has been expanded to account for the immature and diffuse nature of emerging preterm state-related behaviours. As the scale is based on the NBAS, it is more familiar to researchers and clinicians, but training and

Table 2.1 Sleep-wake assessment scales

Brazelton (1973)	*Thoman (1990)*	*Anderson (Gill et al. 1988)*	*Als (1982)*	*Reid (2000)*
1 Deep sleep	1 Quiet sleep	1 Quiet sleep, regular respiration	1A Very still deep sleep	1 Very quiet sleep, regular respiration
2 Light sleep	2 Active–quiet transitional sleep	2 Quiet sleep, irregular respiration, slight movement	1B Deep sleep	2 Quiet sleep, irregular respirations, slight movement
3 Drowsy	3 Active sleep	3 Active sleep, irregular respiration, movement	2A Light sleep	3 Restless sleep, unsettled, some movement
4 Alert	4 Sleep–wake transition	4 Very active sleep, whole body movement	2B Noisy light sleep	4 Drowsy, inattentive, some movement
5 Considerable motor activity	5 Drowsy	5 Drowsy	3A Drowsy with activity	5 Quiet awake, calm, focused attention/interaction
6 Crying	6 Daze	6 Alert, slight movement, fixated eyes	3B Drowsy	6 Restless awake, irregular respirations, suck searching, some movement
	7 Alert	7 Quiet awake, no movement	4A(L) Quiet awake or 4A (H) Hyperalert	7 Hyperalert
	8 Non-alert waking	8 Awake, some movement	4B Bright alert	8 Fussing, grunting, increased movement
	9 Fuss	9 Awake, whole body movement	5A Active	9 Crying, facial grimace, tongue or jaw tremor
	10 Crying	10 Fussing, prolonged exhalation	5B Considerable activity	
		11 Crying	6A Crying	
		12 Hard crying, clenched fists	6B Lusty crying	

supervision in its use are essential. The advantage of this system is that it also assesses autonomic and motor function as part of a holistic assessment and care planning programme, where state functioning is an important consideration. According to the synactive theory of development (Als 1982, 1986), stabilty and organisation in autonomic and motor function can support and facilitate state system stability, thus enabling the infant to engage with the external world or settle into better quality sleep states. Sleep-wake state is more difficult to assess because of the subtle definitions described, nevertheless it is essential to recognise the current functioning of this subsystem in order to protect the infant from stressors and to facilitate and support emerging competencies.

A modified sleep-wake assessment has been developed by one of the authors (Reid), which has practical validity and which has 85 per cent inter-rater reliability with experienced nurses in three neonatal units. Although it is less sensitive than other preterm sleep-wake assessments, it can be readily utilised in general practice with minimal training and supervision. This assessment has been taught to neonatal nursing students on the specialist practitioner programme at the University of Central Lancashire. It has been shown to be a useful instrument in supporting the change process and promoting developmentally focused care in seconding units.

The most vulnerable infants undergoing intensive treatments often maintain a pattern of maladaptive arousal followed by periods of exhausted collapse. There are few resources available for adaptive arousal, that is, when the infant is in a quiet, alert state and attending to low-key social contact. In addition, infants cannot achieve deep, restorative, undisturbed sleep because of immature state regulation. Quiet alert and deep sleep states require facilitation from contingent handling and environmental manipulation.

The three subsystems of autonomic, motor and state-related functioning provide a basis for assessment of preterm infant behaviour, which supports and enhances medical and nursing care. Two additional subsystems, the attentional-interactional and the regulatory subsystems, identified by Als (1982, 1986), are the final components of the synactive theory of development. Attention and interaction are assessed when the infant is in alert state, and regulation is an assessment of the infant's success in achievement of a balance in the other subsystems. Each subsystem can be described independently, but it is the processes of subsystems interaction, or synaction combined with the infant's interaction with his or her environment which completes the developmental theory. This model describes a hierarchical continuum of stability, organisation and development in the underlying subsystems which enables the infant to achieve more complex tasks as stability is achieved, helped by resolving illness, maturity and contingent handling. With experience and reflection, practitioners can begin to assess the attentional-interactional and regulatory abilities of infants, but careful assessment of the autonomic, motor and state subsystems can provide sufficient knowledge of an individual's developmental agenda to determine appropriate interventions in most practical situations.

Behaviour

Behaviours fall into two categories: stress or avoidance signals, where the stimulation is beyond the infant's capacity to assimilate, and comfort or stability signals, where the behaviours demonstrate organisation or self-regulatory activity. Comfort signals inform the observer of the infant's current stable functioning and point to the next level of developmental goals (Table 2.2).

Behaviours should be assessed before, during and following handling activity. It is important to take note of the context of the intervention, including the responses of the infant to caregiving and the influences of the environment. A developmental review should therefore consist of observations of infant behaviour, an assessment of the current level of functioning and recommendations that support stability within the infant's immediate environment of care. The assessment should take place approximately every 7–10 days, although when significant changes in behaviour are noted, these should be recorded on an ongoing basis in the shift report, and plans reassessed accordingly. Wider developmental issues, such as modifications to the environment at large, education of staff and parents, and institutional awareness, all have an impact on the ability to integrate developmentally focused care, but careful, systematic observation is pivotal to its success.

The NICU environment

The birth of any infant presents a dramatic change in environment. Consider the fetal environment: consistent temperature, containment, gravitational support, attenuated light and noise and subtle nutritional, hormonal and psychological influences. However, the full-term infant is ready for the outside world, and quickly adapts with increasing interest in it.

The preterm infant is in double jeopardy as he or she is unprepared for extrauterine life and has to face the particularly hostile environment of the NICU. The greater the degree of prematurity, the more hazardous the environment will be in order to deliver the critical care the infant requires. The infant is exposed to painful and aversive experiences, there is a lack of patterning in handling and an absence of care which responds to the infant's emerging behavioural organisation. The NICU environment is complex and generally over-stimulating, which creates a state of sensory overload and maladaptation. Thus, the preterm infant is frequently and persistently aroused by the environment, particularly noise and light, or by frequent non-contingent or aversive handling events. This state of arousal is compounded if the infant is naked and hindered by flattened postures or the use of equipment such as endotracheal tubes and IV splints, which are uncomfortable and restrict mobility. It is ironic that the infants least able to signal their distress or protect themselves are the ones most likely to be subject to the most invasive treatments.

***Table 2.2* Stress and stability signals**

Stress or avoidance signals	*Stability or comfort signals*
Autonomic system	
Respiratory pause, tachypnoea, gagging, gasping or sighing	Stable heart rate, respiratory rate, oxygenation and colour
Colour, vascular and visceral changes	Tolerance of enteral feeds
Tremor, twitch or startle	
Posseting, vomiting or hiccough	
Bowel strain	
Cough, sneeze or yawn	
Motor system	
Flaccidity of trunk, extremities or face (gape face)	Smooth, well-modulated movements
Hypertonicity – extension of legs, arms, head and neck, fingers, trunk arching, facial grimace, tongue extension	Relaxed postures and tone, with increasing flexion towards midline
Hyperflexion – trunk, limbs, fists, feet, neck	Mobility and efficient self-regulatory activity, e.g. hand and foot clasping, hand to mouth activity, grasping and handholding
Protective manoeuvres – hand on face, salute, high guard arm position	Suck searching and sucking
Frantic, diffuse activity	
Fixed, stereotypical postures	
State system	
Diffuse, oscillating sleep–wake state, whimper or high-pitch cry	Clear, robust sleep states
Strained or irritable fussiness or cry, lack of consolability	Clear, robust rhythmic cry
Eye floating or staring, lack of facial expression, strained or panicked alertness	Ability to self quiet or to be consoled
Active averting	Alert, intent or animated facial expression
	Focused, wide-eyed alertness

Adapted from Als (1982, 1993)

Studies have consistently demonstrated the harmful effects of excessive noise (Long *et al.* 1980; Strauch *et al.* 1993), light (Blackburn and Patteson 1991) and handling (Gorski *et al.* 1990; Cooper Evans 1991), for example fluctuations in heart rate, respiratory rate, blood pressure, motor and state systems stability.

Noise hazard

Sudden noise hazards provoke stress reactions in infants and thus cause instability in the emerging subsystems (e.g., Zahr and Balian 1995). Arousal from noise and activity levels can therefore cause problems with physiological stability in the more fragile infants, and prevent the more mature infants from achieving relaxed sleep and alertness. Constantly raised noise levels, such as machine and equipment noise, background voices and footsteps, can also cause the infant to be aroused and under duress, hindering systems stability, recovery and development. There remains also the possibility of damage to the developing cochlea, as the levels of noise that cause hearing loss in preterm infants is unknown.

Light hazard

Bright lighting conditions in the NICU appear to cause arousal, as changes to lighting conditions affect sleep-wake state (Blackburn and Patteson 1991). Preterm infants are unable to shut out stimulation for rest and sleep and are unable to achieve wide-eyed alertness unless environmental conditions are modified. If sleep-wake state is carefully observed under dimmed lighting conditions, it becomes apparent that this environment can improve the quality of sleep and promote alert states. Under bright and constant lighting, infants are likely to be aroused autonomically and motorically and remain in transitional states with closed eyes, neither deeply asleep nor available for social contact.

A view that the NICU environment cannot be significantly modified prevails. However, there are many practical ways in which noise, lighting and activity can be modified without making structural changes, but they work best in conjunction with better understanding of infant behaviour. The general atmosphere of a unit can be gradually modified as practitioners become more aware of infant reactions, but this requires a reflective and creative approach. Medical and nursing staff need to be aware of the potentially harmful effects of the environment at large, and have some insight into factors that support more stable functioning. Infants who are particularly vulnerable should be placed away from sources of high activity and noise. Equipment manufacturers are taking into consideration incubator and alarm noise, and bins with softly closing lids are available. Telephones and nursing stations should be located outside the immediate clinical vicinity. Noisy footwear should be discouraged in favour of soft-soled shoes. Large, sparsely furnished rooms tend to be more resonant but these can be modified by the use of thick curtaining, clinical grade carpeting and noise absorbent ceiling tiles. Soft furnishings have the advantage of appearing more home-like and less clinical, which will help to alleviate parental anxieties (Miles *et al.* 1991; Padden and Glenn 1997). Smaller nurseries with four to six cots per room tend to protect infants from general activity levels and can provide a more intimate and private atmosphere

Reflective exercise

Observational reflective exercise of an infant in NICU
Observe the reactions of infants in different environmental conditions. Note the circumstances which support the infant's efforts to quieten and relax or reach alertness. Observe infant behaviour during periods of high activity and note the stress responses. Compare state system stability in relation to the environment of care.

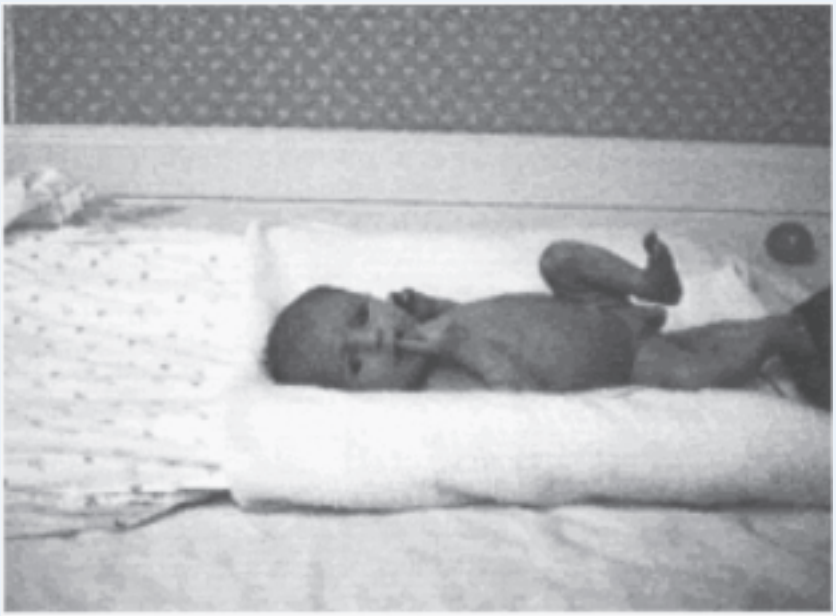


Figure 2.1 **A recovering infant aged 32 weeks in a quiet, alert state, available for social contact**

for parents and staff. A separate admission/resuscitation area may prevent other infants from being subjected to frequent bursts of activity. Many units have experimented with a 'quiet' period with variable degrees of success. Activity, noise and lighting are reduced in order to enable infants to rest and sleep. A gradual lengthening of this period may be possible if infant behaviour is noted to improve as a result of the reduced activity. The quality of sleep and alert states should be monitored, as well as physiological parameters which suggest more stable autonomic functioning. Improved motor system stability may also be observed: tremor, startle, frantic, diffuse or extensor activity should be replaced by more relaxed postures, tone and movements with self-regulatory activity.

The lighting of clinical areas is an important environmental consideration. Practitioners need to balance the need for soft lighting to promote rest and sleep and support alertness, with the need for close observation of the infant. Bright, overhead lighting is inappropriate for the needs of fragile preterm infants, who are unable to shut out the stimulation if they remain unprotected. Dimmer switches should be fitted to overhead lights, to modify lighting conditions. Spotlights or anglepoise lighting can be utilised to illuminate a particular cotspace, which prevents other infants in the nursery from being exposed. Wall-mounted uplighters can provide ambient lighting whilst improving the clinical atmosphere and appearance of the nursery.

There are inevitable limits to the extent of environmental modification, particularly if it is not possible to make structural changes. It is possible, however, to focus on the immediate cotspace to reduce the impact of the clinical environment in vulnerable infants. Incubator hoods or blankets can be used to absorb noise and attenuate lighting conditions. Thick, soft blankets and quilts around the mattress have a dual purpose of promoting comfort and absorbing incubator and tubing noise.

The provision of auditory stimulation in the form of softly modulated voice when the infant is receptive can help to promote alerting. Parents have described seeing their infant respond to their voice by turning, eye opening, calming or becoming animated as a powerful emotional event, which helps them to feel closer to their infant (Padden and Glenn 1997). Other forms of auditory stimulation such as musical toys can be inappropriate, particularly if left inside the incubator to be switched on by staff who are not vigilant about contingent stimulation.

Handling

Handling is the most direct source of stress for infants in the NICU; it is a wellknown hazard to which experienced nurses are well attuned (Gorski *et al*. 1990; Peters 1992; Zahr and Balian 1995). The majority of handling episodes are inevitably unpleasant, stressful or painful, and for the most vulnerable infants, these experiences dominate their existence. It is not surprising therefore that they remain aroused, anticipating the next insult, unable to proceed with their developmental agenda. The more robust infants make efforts to regulate themselves by grasping, sucking and boundary searching, but the more fragile infants are unable to mount the resources needed to stabilise their behaviour or protect themselves.

Contingent care

Handling can also be the most effective source of comfort and pleasure. Human contact, particularly parental contact, can be both stimulating and stabilising provided that it is appropriate and the surrounding environment is conducive. The introduction of positive handling experiences is important

at every stage of the infant's progress, but it is important to monitor their tolerance. Even low-key, gentle handling can cause distress if not delivered in the context of the environment or with consideration to the schedule of care. For example, it may be unreasonable to expect an infant to respond to parental contact following practical caregiving such as a nappy change or feeding, when resources are depleted. Yet between the caregiving schedule, the infant may be lying quietly alert and socially available. Parents' expectations of social contact generally revolve around practical caregiving, but this may not be the most appropriate time for promoting alert and animated interaction. Lighting, noise and activity levels as well as infant preparedness must be taken into consideration prior to social handling if parent-infant interaction is to be optimised. Parents should be encouraged to spend time observing their infants, to become accustomed to their individuality, as an important first step towards contingent interaction. With guidance, many parents will develop observation skills which support practical caregiving, and similarly, parents who are unable to provide direct care can still have an important role to play, being better equipped to advocate for their infants. Parents may become more sensitive to their infant's needs, which is a sound basis for better quality interaction. It may also help to improve maternal self-esteem.

Handling stress can be reduced by incorporating measures to stabilise the infant's status and minimising the risk of overloading their capacity to assimilate events. Infants frequently demonstrate less stress responses in contained, flexed postures as motor stability is achieved and associated autonomic instability reduced. Autonomic and motor stability can be supported by hands-on containment, concave nesting with deep boundaries or swaddling and by maintaining flexed, midline postures and head support whilst performing cares. These techniques provide greater opportunity for the infant to tolerate the intervention without aversion, stress or total systems collapse. In the most fragile infants undergoing hazardous interventions, it may be necessary for two people to perform the intervention—one to maintain flexed containment and one to deliver the treatment or care. This approach to handling can have a marked effect on systems stability, and reflecting on the comparative outcomes of different handling techniques is a worthwhile exercise.

Autonomic stability can be supported by gently introducing the handling episode through voice and soft touch, in order to prevent immediate stress responses. This may also help to prevent the infant remaining in a constant state of partial arousal or exhaustion.

The environment and schedule of care are also important for the more mature and robust infants, for example prior to and during feeding activity. The organisation and energy required for feeding success are considerable, and infant efforts can be supported by ensuring that they are in optimal readiness. Any source of stress, for example vigorous handling or background activity, can provoke failure as their behavioural organisation fails to cope with the additional demands.

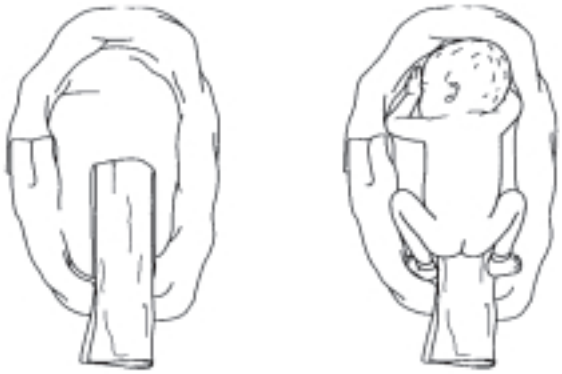
In order to support the infant's responsiveness to social handling, it is essential that we schedule care which is realistic for infants' abilities and which supports

parents' needs. This will often require teaching parents to adjust their expectations and to understand their infants' need for low-key, unimodal interaction.

Postural support

Preterm infants are unable to counteract the effects of gravity, and without support will develop stereotypical head, shoulder and hip flattening which, in turn, leads to a lack of mobility (Downs *et al*. 1991; de Groot *et al*. 1995). This results in an inability to engage in self-regulatory behaviours such as exploration of the face and mouth, hand and foot clasping, boundary searching and flexion and extension of the limbs. Thus persistent flattened postures cause a cycle of reduced mobility, inhibiting opportunities for self-regulation and developmental exploration (Fay 1988; Jorgensen 1993; Hunter 1996). Motor system development may be delayed and postural problems frequently persist into childhood, causing yet more lost opportunities for experiential learning (Davis *et al*. 1993). The development of crawling, walking and fine motor skills may be directly influenced by mobility in the neonatal period (Fay 1988).

(a)



Prone 1:
Softly rolled sheet or blanket positioned in a complete circle. One smaller softly rolled sheet placed over the sheet circle and cover sheet, folded to support pelvic and thoracic lift. Arms and shoulders can be elevated to improve lung function, or fixed and tucked under the thorax. An additional cover may be needed to tuck under the nesting sheet. This serves to draw the nest closer into the infant, supporting flexed containment.

(b)

Prone 2:
Nappy roll length-wise under the body from head to hips. This may require additional rolls or blankets across the baby. The head can be supported at an oblique angle, if tolerated.

(c)

Supine 1:
Soft blanket or sheet rolled into a nest encourages flexion of lower limbs, brings shoulders forward and keeps the head in midline. If this continues round the contours of the head it may promote comfort. A small degree of neck flexion, if tolerated, can provide greater stability.

(d)

Supine 2:
Supine quarter turns can be utilised to vary position and reduce head flattening.

(e)

Sidelying:
One firmly rolled blanket in a 'U' shape. May need to be supported by tucked covers. Note the opportunity for tactile and visual stimulation in this position.

***Figure 2.2* Postural support (a,b) prone position; (c,d) supine position; (e) sidelying position**
Source: Redrawn from positioning chart provided by the Edinburgh Sick Children's NHS Trust, courtesy of Maureen Grant, Superintendent Physiotherapist

Furthermore, infants who are unable to summon protective manoeuvres or maintain flexed postures, thus being more exposed to environmental hazard, may remain in autonomic arousal at a cost to their energy and oxygen expenditure and systems stability. The goals of postural support are therefore multidimensional:

- to support physiological/autonomic stability;
- to prevent abducted, rotated postures of shoulders and hips and flattening of the head;

- to prevent pressure damage from persistent stereotypical or favoured positions;
- to promote mobility and motor systems stability and development;
- to facilitate protective manoeuvres and self-regulatory behaviours.

Physiological stability is the prime goal of nursing the acute or chronically ill infant and postural support should be considered not only for its intrinsic value but also for optimising autonomic and physiological stability.

It is essential to support the infant in a range of positions in order to promote optimum outcome. Flexed, midline containment in side-lying and supine postures is ideal, and some form of hip and shoulder elevation in prone positions will help to reduce flattening. Various commercial products are available for swaddling, nesting and stabilising positions (e.g., Jorgensen 1993; Young 1995) but 'home-made' resources can be equally effective (see Figure 2.2 above).

Equipment

Many infants benefit from a soft head boundary. Head containment often helps to soothe and calm. When in supine postures, the head can be supported to enable occipital lying, the only position which helps to reduce the incidence and severity of head flattening (Cartlidge and Rutter 1988). This can be achieved by the use of a soft roll or small sheet closely contouring the head, but loose enough to ensure some mobility. The use of neck rolls should be avoided as they may damage the delicate vessels at the back of the neck and may cause neck extension. The sheet can be fixed in position by gently tucking it under the shoulders. This elevation will help the shoulders to fall naturally into midline and support mobility of the upper limbs. Cushioned mattressing and/or water pillows will provide a 'nesting' effect, the gentle indentation supports flexion and containment, and reduces the gravity effect on pressure areas (Fowler *et al.* 1997). Fluid-filled cushioning, however, may cause vestibular stimulation, and water mattressing may not provide sufficient stability for mobilisation. Careful observation of subsystems stability and development will guide carers in the provision of postural support which is appropriate for the individual infant.

Prone position

Prone positions are frequently utilised when infants are undergoing respiratory support or recovering from respiratory illness (Mendoza *et al.* 1991; Heimler *et al.* 1992) as there is improved oxygenation and ventilation in this position. As infants recover, prone positions are normally replaced in accordance with the recommendations for reducing the risk of sudden infant death syndrome (SIDS) by supine or side-lying postures (Department of Health 1991). In prone postures, gravity has its greatest effect. The head is always on one side or the other, and hips are forced into abduction and rotation as the infant cannot maintain elevation or lower limb flexion under the pelvis. Shoulders are elevated or forced

into the mattress, particularly if there is any pelvic elevation and the mattress is flat. Although the lower parts of the limbs can mobilise, and there will be some abdominal and pelvic squirming, mobility is considerably reduced without postural support in the prone position. If the mattress is elevated, by approximately 30 degrees, to aid respiratory function, it can reduce the pressure effects on the shoulders, neck and head. However, it also forces the infant to slide down the cot where lower limbs are cramped against the fixed cot boundary. Support around the buttocks can help to fix the infant in a stable prone position.

Pelvic elevation has been advocated by Downs *et al.* (1991) to prevent hip abduction and rotation and promote lower limb flexion. However, care must be taken to ensure that the infant's weight is not transferred to the femur and knee as this may cause other problems with hip development (Dunn 1991). Hip slings have been used to fix the lower body in position and provide some degree of pelvic elevation, but they do not completely prevent hip abduction. Small pillows or rolls can be placed under the infant's pelvis and trunk to elevate the hips, and to a lesser extent the shoulders and neck. It is important to ensure that the elevation does not cause respiratory embarrassment, that the infant appears relaxed and comfortable, the weight of the body is evenly distributed and the limbs are mobile. The lower limbs should be flexed, adducted and tucked under the pelvis or pelvic support.

Supine position

Although supine lying with midline occipital support is the position that best promotes mobility, it is associated with decreased ventilation and increased energy expenditure and is therefore more frequently utilised in the mature and stable infant (Martin *et al.* 1979; Masterson *et al.* 1987). However, these studies were undertaken with infants in exposed and unsupported supine postures; it is therefore important to re-evaluate this evidence in the light of improved developmental knowledge. It is entirely possible that modified supine postures actually improve oxygenation and energy expenditure as infants are able to utilise their motor system to engage in self-regulatory and protective behaviours and thus reduce autonomic stress. Even if there is a cost to energy expenditure, the increased opportunity to mobilise and explore may be worthwhile as a developmental intervention. Careful assessment of the individual infant will determine its effectiveness; as with any position change, it is important to continue observation until the infant is stable and comfortable.

This position requires some degree of 'nesting' around the infant boundary in order to maintain head, shoulders, pelvis and limbs in midline flexion. A small degree of hip and shoulder elevation from the boundary will promote limb mobility, support midline alignment and facilitate self-regulatory behaviours such as hand to mouth activity. This can be achieved by a combination of nesting into soft bedding and the use of close, flexible boundaries around the infant's entire body. The lower limbs should be supported in flexed postures, but as limb mobility is facilitated in this position, opportunities for stretching and extending should be provided. Often limbs

can be found draped over the boundary, but this does not mean that the infant no longer needs boundary support. Whilst utilising some degree of shoulder and pelvic support, they should be supported well enough to flex and extend at will. The effects of gravitational pressure are more evenly distributed in the supine midline position, and the pressure on the occipital region should ensure a more rounded head shape.

Side-lying position

Side-lying postures tend to minimise hip and shoulder rotation and abduction. Limbs should be in adduction and flexed towards the midline axis. This posture can be maintained by swaddling in soft, flannelette sheeting. The infant's back should be rounded, and may require some boundary support from a small rolled sheet or towel.

It is essential that boundaries are flexible to closely follow the contours of the body. Anteriorly, the infant may benefit from the placement of a soft toy or filled silicone glove within reach of the hands but away from the face. This will provide an opportunity for grasping and tactile stimulation and may encourage flexion towards the object. Side-lying can also be used to treat unilateral lung disease, with better oxygenation being achieved by positioning the 'good' lung uppermost (Heaf *et al.* 1983).

Attention to detail in positional care is crucial to the dual goals of prevention of postural deformity and the facilitation of self-regulation and mobility. It is important to ensure that side-lying, prone and supine postures are utilised and position changes are planned to incorporate a varied range of positions, including half turns and intermediate postures, where appropriate (see Figure 2.2). It is also essential to assess the effectiveness of the intervention by ensuring that the infant tolerates the new position and that it supports both mobility and comfort.

The importance of behavioural assessment and developmental goal-setting cannot be over-emphasised, as it enables the practitioner to monitor and evaluate the effectiveness of a range of developmental strategies. The ability to refer to systems functioning helps to validate this approach.

Developmental models of care

In order to fully utilise developmental knowledge, it is essential to incorporate theoretical concepts and practical skills into the model of care. This will ensure that experienced practitioners convey their values to less experienced team members, and developmental practice is regarded as an essential component of neonatal nursing care. Effective documentation will ensure that care planning decisions are based on careful assessment, and implementation is continued over shift changes. Issues such as mobility, sensory, comfort and communication needs are thus considered to be as important as other nursing and medical needs, and developmental considerations become fundamental to neonatal care. As these issues are

incorporated into practice, it is important to evaluate the outcomes, for both individuals and populations. Documentary evidence of the effects of the implementation of a particular strategy, such as reference to more stable functioning, provides the justification for its continuation in a plan of care. Audit of longer-term outcomes such as ventilator days, days on oxygen, days to full feeds and days to discharge, may provide the evidence which will support further investment and practice development in a wide range of strategies and interventions.

The support of parent-infant relationships is perhaps the most important developmental intervention of all, as it is this relationship which will have the greatest impact on long-term developmental outcome (Hack *et al.* 1995; Lauch *et al.* 1997). Developmental models of care must therefore place the emotional and educational needs of parents at the centre of practice. This means going beyond the practical caregiving approach, and providing structured psychoemotional support and contingent handling skills development. Parents should be encouraged to contribute actively to care planning decisions, and be fully informed and supported as partners in care. In addition, mothers of infants undergoing intensive care are known to suffer from particular psychoemotional difficulties associated with loss of self-esteem, guilt, anger and blame (Affonso *et al.* 1992; Padden and Glenn 1997). Many may need help to express these feelings in order to recover and proceed with the tasks of caring for their infant, whilst at the same time resuming daily responsibilities. Communicating with parents is clearly an important aspect of nursing care, and a developmentally focused model should ensure minimal levels of communication are routinely incorporated and documented, for example written guidelines and information, teaching plans, interaction support, crisis interviews and discharge planning. Semi-formal interviews with parents can reveal many underlying difficulties, but these are unlikely to be revealed if communication consists of opportunistic conversation at the cotside, with the multitude of distractions and lack of privacy (Padden and Glenn 1997). This model of care therefore advocates prearranged meetings with parents on a regular basis, where information-giving, decision-making and evaluation can take place with nurses who are familiar with both the infant and the family.

Parents may experience difficulties in expressing their communication needs, as they often submerge or camouflage their emotional difficulties as the focus shifts from the pregnancy and birth to the progress of their infant (Affonso *et al.* 1992, 1993). An acknowledgement that negative emotional reactions are normal and that parents will need help to adjust is a useful first step towards effective communication.

Supporting parents in their interactions with their infant must take into account their abilities and expectations. Most parents have little or no experience of prematurity and need to learn new skills, in order to establish their parental role. Those who have previously experienced preterm birth, rather than being better able to cope, appear to be even more devastated if it happens again (Padden and Glenn 1997) and therefore warrant careful support. Young, unsupported primiparous mothers are particularly vulnerable

and need help to establish their identity and develop their maternal role under threatening conditions (Zabielski 1994). Whilst encouraging close physical contact with their infant, clarification of their role requires negotiation based on an accurate assessment of both their capabilities and knowledge, and their infants needs. Developmental care provides a focus for such negotiations, constructing a dialogue based on a partnership between professional caregivers and parents. The practical hassles and time pressures associated with visiting NICU and attempting to resume daily responsibilities are considerable and frequently underestimated by NICU staff (Padden and Glenn 1997). Efforts to improve the quality of time spent in NICU and reduce the pressures experienced are likely to reduce the stress associated with separation and hospitalisation. Caring for siblings and other family members is frequently cited as a responsibility which conflicts with the mother's need to be close to her new infant. Time spent expressing milk and travelling to and from the unit are also perceived by mothers to be highly stressful (Padden and Glenn 1997).

Practical support in such instances is an important means of improving the quality of visiting time and subsequent parent-infant interaction: for example, the provision of a supervised crèche for siblings, playpens in clinical areas for very young infants and help with transport to and from the unit. Double-pumping facilities for mothers expressing their milk will halve the time spent expressing, making more time for direct infant care.

As well as time pressures, parents frequently cite lack of space and privacy as an obstacle to interaction with their infant. Where possible, interactions should take place out of sight of other parents and staff, ideally behind screens or in separate nursing rooms or cubicles. These factors tend to be taken into consideration when breast feeding is attempted, but privacy and intimacy are rarely considered for those who are bottle feeding or who are holding or cuddling their infants.

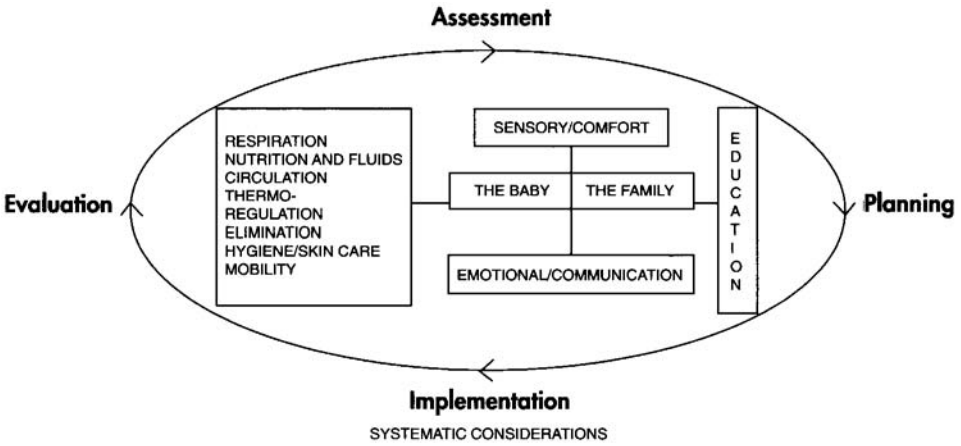


Figure 2.3 **Family-centred developmental model of care**

It can be seen that infant and family are at the centre of the model depicted in Figure 2.3. However, some of the considerations apply to the infant, while educational issues clearly apply only to parents and other relatives or friends. Sensory and comfort needs and emotional and communication needs can apply to both. All should be assessed to see whether they require intervention, then planned, implemented and evaluated according to individual requirements.

The model does not have to differentiate between a need, problem or potential problem if they are regarded as systematic considerations (see Figure 2.4). All

Respiration	Rate, movements, respiratory status, colour
Circulation	Skin perfusion, apex beat, pulses, blood pressure, haematological status, signs of constriction/oedema
Fluids/nutrition	Fluid balance, biochemical status, tolerance of enteric feeds, feeding readiness, feeding performance, feeding plan, information on lactation and expression
Thermoregulation	Peripheral/central/environmental temperatures, stability of temperatures, humidity, frequency of assessment, application of heat conserving/losing measures
Elimination	Output assessment, bilirubin status, stool chart, urine tests
Hygiene/skin care	Pressure areas, IV sites, eye/mouth care, wound care, dressings, nappy care, skin protection, lotions, infection screens
Mobility	Tone, postures, reactions to handling. Passive movements, position change, aids to self-regulation, postural support, mobility and self-regulatory activity
Sensory/comfort	Rest and sleep, environmental protection, contingent stimulation, tolerance of handling, promotion of social interaction, intimate contact and privacy. Pain relief and sedation. Maternal postnatal care needs
Emotional/Communication	Visiting/residential arrangements, social support and responsibilities, access arrangements, financial or social difficulties. Information needs, communication chart, care planning discussion. Religious beliefs and practices, ethnic or cultural beliefs or practices. Parent to parent support, key worker support, psychoemotional assessment, referral to specialist or voluntary sources. Primary care team communication
Educational	Teaching programme identified, agreed, actioned and evaluated. Written supporting material. Interview schedule. Resources for discharge, follow-up arrangements, access to self-help groups and specialist centres

Figure 2.4 **Examples of systemic considerations within the family-centred developmental model of care**

aspects must be assessed, and the appropriate strategy devised to meet the need, deal with the problem or prevent a potential problem from developing.

Documentation to support the developmental aspects of the model can be devised in the same way that clinical assessments and plans are devised. Charts can record sleep-wake state, postural support, parent interaction and teaching plans. Care plans can record behavioural assessment, interventions and evaluations. Developmental progress can thus be submitted for audit in order to evaluate the effectiveness of developmentally focused care over time and make resource decisions that will influence the future direction of practice development.

Conclusion

Although research has demonstrated the potential to improve outcome and reduce health care expenditure, there is no prescriptive approach. There are few conclusive solutions that apply in general, although modification to the environment is likely to benefit all infants and parents experiencing intensive care. Creative and reflective practice development, based on the available knowledge of developmental care, should enhance the nurse's role in promoting optimal outcome for infants and families experiencing neonatal intensive care.

The values and beliefs of the discipline of neonatal nursing are congruent with an expansion of developmental care. Issues such as advocacy, interpersonal therapeutic care and the nurturing of fragile lives and relationships are central to the nurse's role. Neonatology is a complex discipline as it works at the frontiers of biomedical technology, often in stressful and under-resourced circumstances. However, developmental interventions have the potential to enhance the effectiveness of neonatology by supporting traditional medical and nursing care.

The infant and family are in a state of crisis or disequilibrium and require the basic human skills of empathy and effective communication. The balance between technology and humanity, art and science, is difficult to negotiate, but success is nowhere more essential than at the beginning of a disadvantaged life.

An understanding of developmental theory and knowledge of behavioural assessment will serve to promote a creative approach to individualised, holistic, family-centred care that reflects the fundamental values and beliefs of neonatal nursing.

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Chapter 3

Resuscitation of the Newborn

chapter 3

Contents

Introduction

Resuscitation in the newborn period is a relatively frequent occurrence. Saugstad (1998a) suggests that 3–5 per cent (4–7 million) of the 140 million newborn infants born worldwide each year need some kind of resuscitation at birth. Extrapolating this data to the UK population suggests that an average of 30 000 infants will require some intervention at birth. Whilst most of these infants will respond to good airway and ventilatory management, a small proportion will go on to require further interventions of cardiac massage and, in extreme situations, drug therapy.

Neonatal nurses, as either the lead practitioner or assistants, are often involved in the resuscitation of 'high risk' infants. In order to comply with Code of Professional Conduct regarding accountability (UKCC 1992), it is imperative they have the requisite knowledge and understanding of the physiology of the asphyxial process to be able to rationalise interventions that may need to be undertaken. This chapter examines the current available literature to establish what is effective practice in the resuscitation situation. Whilst it will focus upon delivery room management, the reasoning and techniques can be applied in the intensive care unit situation.

Infants who require resuscitation at birth fall into two broad categories: those who have undergone a period of hypoxic stress in utero, and those who are prone to hypoxaemia in the immediate postnatal period due to inadequate pulmonary development, airway obstruction or congenital malformations. Significant hypoxia will lead to asphyxic tissue damage, so anticipation of the need for resuscitation prior to delivery is of great importance.

Asphyxia

Asphyxia before, during or after birth is an important cause of perinatal mortality and morbidity (Williams *et al.* 1993).

Asphyxia is literally defined as 'without pulse', but Levene's definition of 'impairment of placental or pulmonary gas exchange resulting in hypoxia, hypercapnoea and acidosis' (1995:405) is perhaps more reflective of the perinatal situation, as it is this series of events that leads to circulatory compromise and collapse.

Relative oxygen depletion is part of the normal process of labour, with the healthy fetus being equipped with several protective mechanisms that will prevent hypoxic damage to vital organs. The high affinity for oxygen of fetal haemoglobin supports the flow of oxygen to the tissues. This mechanism allows for oxygen extraction at tissue level to increase by almost 100 per cent during hypoxaemic events as it shifts the haemoglobin oxygen dissociation curve to the right (decreased affinity), which enhances oxygen extraction delaying the development of hypoxic damage (Bocking *et al.* 1992; Talner *et al.* 1992). The fetal heart rate is a major determinant of cardiac output. It is four times greater than that of an adult per kilogram body weight, as it is the combined output of right and left ventricles, with 50 per

cent of the output directed to the placenta (Cohn *et al.* 1974). The combination of these mechanisms allows for a wide safety margin of adequate oxygen delivery during labour.

In short duration hypoxaemic events, the heart rate of the mature fetus will fall, but as it is associated with an increase in arterial blood pressure, myocardial contractility is increased and cardiac output is maintained. This is due to vascular resistance changes in the fetal gut and carcass, which divert the blood supply to the heart, brain and adrenals (Cohn *et al.* 1974). This autoregulatory process is modulated at cellular level via metabolic feedback regulation of the calibre of the arterioles and capillary sphincters (Grainger *et al.* 1975, cited in Talner *et al.* 1992). The heart and brain are efficient at autoregulation and can maintain their blood flow over a wide range of perfusion pressures and oxygen contents. Hypoxaemic events of this type are intermittent during labour, and the ability of the fetus to quickly redistribute oxygenated blood is very important.

When there is no stress, the fetal and newborn heart is working at close to capacity merely to satisfy normal demands of tissues and growth (Talner *et al.* 1992), therefore any increased output requirement to satisfy sub-optimal tissue oxygen demand cannot be sustained for long and myocardial contractility will fail. So whilst the fetus can make circulatory adjustments to compensate for the rigours of labour, under severe conditions these adaptive mechanisms will be overwhelmed, the shunting of the blood towards vital organs and cerebral oxygen delivery will reduce as the blood pressure and cardiac output fall.

The ability of an organ to maintain aerobic metabolism is dependent on the amount of energy (**adenosine triphosphate, ATP**) needed to maintain functional activity, the amount of oxygen delivered to the tissue and the amount of oxygen extracted by the tissue. The variation in response during labour is related to maturity, with immature fetal tissue being generally more robust. The smaller body mass to placental size gives a larger oxygen reserve to the premature fetus (Greene and Rosen 1995). As the ventricular pressure falls there is a corresponding decrease in cerebral blood flow (CBF). During severe asphyxia CBF is directed to the brain stem rather than the cerebrum, which increases the likelihood of damage to the cerebrum and cortex (Williams *et al.* 1993). When organ blood flow and oxygen delivery are severely compromised, increased tissue extraction of oxygen will not be able to compensate fully for the decrease in oxygen delivery. This will result in aerobic metabolism becoming compromised, and anaerobic metabolism (glycolysis) becoming a supplementary mechanism to maintain cellular energy stores.

Anaerobic glycolysis produces approximately one-fifteenth of ATP produced aerobically and is consequently a short-term emergency measure. In this state glucose can only be oxidised to pyruvate and lactate. As the lactate accumulates in the blood and tissues there is a gradual progression towards a metabolic acidaemia. ATP is required to maintain the ion gradient of sodium and calcium across the cellular membrane. Failure of this mechanism leads to an influx of sodium and calcium into the cells, which

allows for an influx of water, leading to intracellular swelling. This acute swelling creates cytotoxic cerebral oedema, which may contribute to brain injury.

The sequence of events that occur following intrauterine hypoxia has been well documented since the work of Dawes in the 1960s (which is recommended reading for all involved in neonatal resuscitation) and will not be further described. As birth asphyxia is arguably the most common cause of perinatally acquired severe brain injury in the full-term infant (Levene 1995), practitioners working within the field of neonatal care have a responsibility to be able to ameliorate its potentially devastating effects by prompt and skilled resuscitation. This is true also in management of the preterm infant who, if neglected in the first few minutes of life, is more likely to succumb to hypoxic and thermal stresses that will undoubtedly adversely affect that infant's outcome.

Anticipation

On arriving in the delivery room at least four things should be ascertained prior to the delivery: the gestation of the infant, any known problems (for example, exomphalus), the presence of multiples, as more assistance will be required, and any use of maternal narcotics. If time permits, a more detailed history can be taken (for example, parity, previous perinatal morbidity or mortality, maternal illness), but the above will enable a plan to be formulated quickly.

Wherever possible, the parents should be spoken to regarding what is likely to happen following the delivery (for example, immediate removal of the infant to the resuscitation platform), so that they are aware of the situation and prepared as much as possible as to why these interventions may be necessary. This is not always possible, of course, when an infant is born unexpectedly compromised. Neonatal resuscitation events are unique in that there are usually other persons in the immediate vicinity who are not directly (practically) involved with the situation. Whilst debate may remain around the presence of relatives in adult resuscitation scenarios and accident and emergency departments (Mitchell and Lynch 1997; Stewart and Bowker 1997), the situation is not up for discussion during delivery room resuscitation events, as the parents are most certainly present and witnessing the events as they occur. This has implications for the professionals' demeanour and the support of the parents both during and after the resuscitation event. The resuscitation situation within the NICU can be further compounded when other parents are in the vicinity. This is particularly pertinent when the parents of the 'collapsed' infant are not present. This situation requires thought and sensitivity surrounding the issues of support and reassurance of the witnesses, whilst working within the remit of patient confidentiality, and ultimately the support of the actual parents when they are informed of events.

The equipment and environment in the delivery room should be also prepared so that everything that may be required is close to hand and infant heat loss is

minimised. Whilst it may appear obvious that equipment is checked, and be sufficient for the task in hand, failure of this simple procedure is implicated in a proportion of neonatal deaths reported annually in the UK (Maternal and Child Health Research Consortium 1995).

Careful attention to thermal homeostasis cannot be overemphasised in a resuscitation event. Whilst practitioners are mindful of its importance, this intervention is often only partly achieved, with the infant being partially dried, with wet hair, and left in contact with damp towels, whilst other interventions take precedence.

Thermal stability must be maintained. The intrauterine environment is approximately 1.5 °C greater than the maternal temperature. A newborn infant's temperature is approximately 37.8 °C (range 37–39 °C) (Mann 1968, cited in Rutter 1992). Heat losses by convection, radiation and evaporation are high. Very low birth weight infants are at an even greater risk of cold stress due to their unfavourable surface to mass ratio. The core temperature of a wet, asphyxiated infant can drop by 5°C in as many minutes (Milner 1995). Cold stress is associated with hypoxia and acidosis, factors which inhibit surfactant production in the newborn and should, obviously, be avoided. (See Chapter 4 for events following heat loss.) The room should be warm (approximately 25° C, if possible), with doors and windows closed to prevent draughts and convective heat loss. A switched on radiant heat source and several warmed towels should be immediately available for use following the delivery (see pp. 77–9).

ABC(D and E) of resuscitation

As soon as the infant delivers, start the clock and assess the situation. This assessment process is crucial.

Most healthy mature infants have spontaneous onset of respiration by 10 seconds (Milner 1995), therefore no further assistance is required other than keeping warm by careful drying, followed by skin to skin contact with the mother, with any exposed surfaces covered by dry, warm towels. This point is made to reinforce the point that many infants, even those in the 'high-risk' categories, are born in good condition and require minimal interventions. Research indicates that the presence of a paediatrician at such deliveries increases the incidence of unnecessary intubation (Kroll *et al.* 1994), a procedure that is not without potential complications.

An infant born in less than optimal condition requires further assessment. Place this infant onto a warmed padded surface underneath a radiant heat source, and dry off. Immediately remove the wet towels and cover extremities, especially the head, as it accounts for 25 per cent of the infant's surface area and is consequently a site for massive heat loss (Daze and Scanlon 1981). This intervention has the dual effect of reducing heat loss and providing tactile stimulation, which may be enough to induce gasping and onset of respiration.

Airway

Airway management is the next consideration, if the infant has failed to cry or is not breathing.

The infant should be in the supine position, with the head in a neutral or slightly extended position. The use of a 2 cm thickness pad under the shoulders may be helpful in achieving and maintaining proper head position (Zideman *et al.* 1998).

If the infant is gasping, but there is no improvement in colour, or the heart rate is less than 100, gentle oropharyngeal suction is indicated. Aggressive pharyngeal suctioning can delay the onset of respiration (Milner 1995), cause laryngeal spasm and vagal bradycardia (Codero and How 1971).

Suction should be controlled from three standpoints, the *pressure* exerted should not exceed 100 mmHg (13.3 kPa), the *depth* of catheter insertion should not exceed 5 cm from the lips, and *time* should not exceed 5 seconds (BMJ Working Party 1997; Zideman *et al.* 1998).

The rate, rhythm and depth of respiration now need assessing.

Breathing

The spontaneously gasping infant who has central cyanosis and/or a heart rate around 100 beats per minute may require facial free-flow oxygen, by mask, funnel or cupped hand. Whilst this intervention will provide cold stimulus for breathing to occur, it can lead to significant cooling which is counter-productive.

Apnoea, pallor or a heart rate less than 100 indicates the necessity for positive-pressure ventilation via a mask. If a self-inflating bag system is utilised, the bag volume should be 500 ml. Bag volumes less than 300 ml rarely produce ventilation greater than the anatomical dead space (Field *et al.* 1986), so are inadequate in creating an adequate functional residual capacity (FRC). This type of system also requires a pressure-limiting device pre set to 30 cm H_2O, to avoid inadvertent over-distension of the lungs leading to pneumothorax. This limiting device should, however, have the facility to be over-ridden, as some infants may require generation of greater pressures to achieve appropriate tidal volumes and FRC. Slow compression of the bag is required for the first five breaths so that an inflation time of around one second is achieved. This technique is said to achieve a better formation of FRC (Vyas *et al.* 1981), although this is not easily achieved with some systems.

T-piece resuscitation systems have had a resurgence within neonatal resuscitation in both delivery room settings, with many resuscitation platforms having an integral system, and within the NICU, with the development of free-standing portable systems. With these systems the first inspiratory breath may be held for up to 3 seconds, with a subsequent five breaths being held for 1–2 seconds. Following establishment of respiration with these first few slow breaths a regular rate of 40–60 per minute should be established.

The T-piece system should also have pre-set pressure limits in order to prevent over-inflation, and the development of air leaks. Satisfactory inflation pressures may stimulate the infant to make spontaneous inspiratory efforts by invoking Head's paradoxical reflex (Milner 1995).

Many of these systems also have the ability to give positive end-expiratory pressure (PEEP). The application of PEEP, at resuscitation, may have several benefits for the premature infant. It is thought not only to optimise alveolar expansion and conserve and prolong the effectiveness of surfactant (Wyszogrodski *et al.* 1975; Froese *et al.* 1993), but also attenuate the potential damaging effects of high inspiratory pressures (Carlton *et al.* 1990).

Whichever system is being used, a soft edge mask, of an appropriate size, should be used to maintain a good seal around the nose and mouth, and prevent trauma to the infant's eyes or skin.

Currently it is still recommended to use 100 per cent oxygen in newborn resuscitation (BMJ Working Party 1997; Chameides and Hazinski 1997; Zideman *et al.* 1998), despite the potential damaging effect that this may have. The term 'oxygen-free radical disease' has been introduced into neonatology in recent years, with the main emphasis being its association with retinopathy of prematurity, bronchopulmonary dysplasia, necrotising enterocolitis and persistent ductus arteriosus (Saugstad 1998b) in the premature population. There are, though, other considerations to be made in the use of high concentrations of oxygen during resuscitation of both premature and term infants.

Lundstrom *et al.* (1995) measured cerebral blood flow on preterm infants resuscitated with either room air or 80 per cent oxygen. They reported a persistent cerebral vasoconstriction in the high oxygen group which, they concluded, may make the brain more susceptible to hypoxic episodes or ischaemia which may increase the risk of cerebral damage in the newborn period.

In the term asphyxiated population, high oxygen concentrations may also be damaging due to hypoxia reperfusion injury. Animal studies suggest that restoration of blood supply containing a high concentration of oxygen is more detrimental than restoration of the circulation alone, as it appears to create radical oxygen metabolites which create further pathological changes (Rootwelt *et al.* 1992; Raivio 1996).

Following these findings, is it justifiable to continue to give high concentrations of oxygen to newborns? Ramji *et al.* (1993) suggest that room air is no less effective in resuscitation of asphyxiated newborns than 100 per cent oxygen, and Svenningsen and colleagues (1989) have long suggested that 30–40 per cent oxygen will appropriately treat the preterm infant in the delivery room.

Whilst the prevention of hypoxia must remain a high priority during neonatal resuscitation, the indiscriminate use of oxygen needs careful consideration in order to prevent potential long-term adverse sequelae.

Most infants will respond favourably to mask ventilation (Arya *et al.* 1996), but a proportion will require further intervention of endotracheal intubation,

due to extreme prematurity, severe asphyxia, altered anatomy or poor response to bag and mask ventilation.

Mask ventilation is contraindicated in infants with diaphragmatic hernia (Harjo and Jones 1993), therefore in antenatally diagnosed cases a practitioner skilled in endotracheal intubation should be in attendance (see p. 289).

Circulation

The heart rate should be assessed. This is most easily achieved in the delivery room situation by palpation of the base of the umbilical cord. The most frequent cause of an inadequate heart rate is ineffective ventilation. Once this is established most heart rates will rise. A rate of less than 60 beats per minute, or less than 80 beats per minute despite effective ventilation, requires prompt initiation of cardiac compressions.

There are two techniques described for cardiac compression in the newborn. The optimal technique is for the operator's hands to encircle the thorax, with the thumbs, pointing **cephalad,** placed side by side over the lower third of the sternum (David 1988; Menegazzi *et al.* 1993). The sternum is then compressed to approximately one-third of the thorax depth (Chameides and Hazinski 1997), at a rate of 120 beats per minute ((Burchfield 1993; Zideman *et al.* 1998). It is important that this rate is not exceeded, as too rapid cardiac compression will not allow for the relaxation phase of the procedure, which will hinder its effectiveness. Each compression should have an equal pressure and relaxation phase. To be most effective the compressions should be interposed with a ventilation breath every third compression, as simultaneous chest compression may hinder ventilation. This 3:1 ratio will give 120 cycles per minute, for example, 90 cardiac compressions to 30 breaths.

The alternative technique is to place the index and middle finger on the lower third of the sternum and press at the same depth and rate as previously described. This technique may be more appropriate in the extremely low birth weight population due to the size of the thorax, but it is recognised as being less effective in creating coronary artery perfusion pressures and mean arterial blood pressure (Menegazzi *et al.* 1993).

Compressions should be briefly stopped every 30 seconds to check actual heart rate. Once the infant's spontaneous rate is above 80 and rising, compressions may stop.

In Perlman's 1995 study, chest compression (or drugs) as part of neonatal resuscitation was administered in only 0.12 per cent of 30 839 newborn infants (cited in Ginsberg and Goldsmith 1998), but in the unlikely event that the infant's heart rate is still not improving following compression, drug therapy needs to be considered.

Drugs

Drugs are not a first line action in neonatal resuscitation and should only be considered when one is certain that the endotracheal tube is appropriately placed, the chest is moving, ventilation with 100 per cent oxygen is under way, and the heart rate is still not increasing despite cardiac massage.

Drug therapy during resuscitation is one of the most controversial areas, with arguments raging as to which are the most appropriate and most effective agents, dosages, routes of administration and speed of delivery. These controversies are, in part, due to many of the drug dosages being extrapolated from adult data. Also, when the anatomical and physiological differences in the neonate are considered, for example the presence of lung fluid, right to left shunts, and the susceptibility of the neonatal brain to haemorrhage, these extrapolations may be inappropriate (Ginsberg and Goldsmith 1998). Indeed, the use of drugs at all in certain groups of infants may in itself be inappropriate, as their necessity seems to be associated with an extremely poor outcome (Sims *et al.* 1994).

This section will attempt to rationalise which drugs are the most effective in neonatal resuscitation.

Adrenaline

Adrenaline (epinephrine) is used to stimulate the failing heart during resuscitation in both adults and infants. It is an endogenous catecholamine which has both alpha- and beta-adrenergic effects. Its major benefit during resuscitation is the alpha-mediated vasoconstriction which increases the aortic diastolic pressure increasing the coronary perfusion pressure, and myocardial blood flow (Burchfield 1993).

The route of administration is via the endotracheal tube or the central intravenous route. Lindemann (1984) was the first to suggest endotracheal instillation in the delivery room and neonatal intensive care setting. Utilising the same dose as for the intravenous route (0.1 mg/ml of 1:10 000) he reported the positive response of return to normal heart rate within 5–10 seconds of instillation. Previous to this, adult work had suggested a ten-fold increase in the dose to gain net effect if the tracheal route was to be used (Roberts 1978, cited in Burchfield 1993). In the neonatal population, the theoretical risk of intracranial haemorrhage, especially in the highly vascular germinal matrix of the preterm infant, following the acute hypertensive response to the drug, excludes this dose increase from routine recommendation (Nadkarni *et al.* 1997).

Mullett *et al.* (1992) suggest that there may be a delay in diffusion of tracheal adrenaline during the first week of life due to thicker epithelial linings of the respiratory bronchi, alveoli and pulmonary vascular walls, suggesting that infants collapsing after the first week of life may respond more effectively to tracheal adrenaline than those immediately after birth. The tracheal route should be used as a first route of administration in any given situation, to 'buy time' whilst vascular access is being gained for repeated doses, if required, or as a route for

other drugs that may need to be administered (see p. 303). If the first dose of adrenaline does not have the desired therapeutic effect then a second dose can be given after 3–5 minutes.

As metabolic acidaemia and hypoxia will attenuate the action of adrenaline (Preziosi *et al.* 1993), the failure of repeated doses may be due to the acidotic state of the infant. Ideally the base deficit should be determined by blood sample analysis, as is the case in the intensive care setting, but in extreme situations, for example, prolonged resuscitation in the delivery unit where the infant is not responding to the above interventions, the use of buffering agents needs consideration.

Buffers

During cardiopulmonary collapse, gaseous exchange in the lungs ceases whilst cellular metabolism continues in an anaerobic environment. This produces a combination of respiratory and metabolic acidosis. As previously discussed, acidaemia decreases myocardial contractility, diminishes blood pressure by vasodilatation and decreases the heart's responsiveness to catecholamines. The correction of this state, then, is not only rational, it is imperative if recovery is to achieved. Whilst management of the respiratory component is, without question, good ventilatory support, the management of the metabolic component is at the very least contentious!

Spencer *et al.* (1993) suggest that it is difficult to determine infants with severe metabolic acidaemia clinically, and that by one hour of age a pH as low as 6.99 will be corrected spontaneously by the infant's own compensatory mechanisms.

Koster and Carli (1992:143) also claim the best buffer agent is 'the return to a spontaneous circulation'. Continued cardiac depression following good ventilation and adrenaline administration may indicate that acidaemia is a contributory factor and that a more expedient return to normal pH is indicated rather than hoping the infant will 'self correct' if left.

The use of alkaline buffers for correction of metabolic acidosis has a long tradition in cardiopulmonary resuscitation and several drugs are available for clinical use. Wherever possible, the base deficit should be ascertained. This may well be achievable within a resuscitation situation within the NICU but this is not usually feasible within a delivery unit.

Bicarbonate

Sodium bicarbonate has long been the most commonly used buffer for correction of metabolic acidosis of differing aetiologies, as well as acidosis seen at cardiac arrest (Bjerneroth 1998).

In the normal situation carbon dioxide entering the blood as a result of metabolism in the tissues initially exists as a simple solution. Some of it though will react with water to form carbonic acid, which dissociates to hydrogen and

bicarbonate due to the action of carbonic anhydrase in the red blood cells. Most of the carbon dioxide in the body is transported this way. For example:

$$H_2O + CO_2 \Leftrightarrow H_2CO_3 \Leftrightarrow H^+ + HCO_3$$

The reverse set of reactions occurs in the lungs. As the carbon dioxide level declines, bicarbonate ions re-enter the red blood cell and bind with hydrogen to form carbonic acid which is then cleaved by carbonic anhydrase to release carbon dioxide and water for elimination during expiration. This maintains the correct ratio of hydrogen and bicarbonate in the blood to maintain the acid base balance.

$$H^+ + HCO_3 \Leftrightarrow H_2CO_3 \Leftrightarrow H_2O + CO_2$$

Several authors (Howell 1987; Burchfield 1993; Hein 1993; Bjerneroth 1998; Ginsberg and Goldsmith 1998) cite Ostrea and Odell's work of 1972 when describing the use of bicarbonate in correcting metabolic acidosis. They describe it as functioning as a physiological buffer only in an 'open system', in which the carbon dioxide created can be transported to the lungs and blown off. Thus, in inadequate ventilatory situations (during resuscitation) a 'closed system' occurs and the carbon dioxide created shifts the equation to the left, leading to a worsening of the acidaemia. In addition, the tissue and venous side of the circulation probably acts also as a 'closed system', in that carbon dioxide cannot be appropriately eliminated due to poor tissue perfusion from the diminished cardiac output (Hein 1993). Thus, bicarbonate will only serve as an effective buffer in balancing the hydrogen-bicarbonate in the presence of adequate ventilation, otherwise the metabolic acidosis will be replaced by respiratory acidosis. Moreover, since the increased carbon dioxide diffuses more rapidly into the cells than the bicarbonate, paradoxically it may actually worsen intracellular acidaemia. Therefore indiscriminate administration of bicarbonate during resuscitation may make matters worse (Howell 1987).

Other potential complications associated with bicarbonate administration are within its hypertonicity. Undiluted bicarbonate contains 2000 mOsmol and 1 mEq of sodium; as a consequence it is highly implicated in the development of hypernatraemic states and intraventricular haemorrhage in the preterm infant (Howell 1987; Ginsberg and Goldsmith 1998).

Trishydroxyaminomethylmethane (THAM, TRIS)

Other buffering solutions may be considered. Trishydroxyaminomethylmethane was initially suggested by Gomori in 1962 (cited Bjerneroth 1998) for pH control. An organic buffer, THAM is a weak base that acts as a proton acceptor which increases pH and reduces carbon dioxide (Bowman and Rand 1980: 28.25). Whilst it was regarded as a promising alternative to bicarbonate it has been recognised as having several toxic effects, including hypoglycaemia, severe

vasospasm leading to phlebitis and thrombosis, and with extravasation injuries resulting in necrosis. More importantly, it has been demonstrated that it induced arterial vasodilatation, decreasing aortic diastolic pressure and drastically lowering coronary artery perfusion, thereby reducing the success in cardiopulmonary resuscitation (Kette *et al.* 1991, cited in Bjerneroth 1998). In the light of these findings, THAM cannot be recommended during acute emergency resuscitation situations.

Tribonat

In 1994 the first clinical trial of Tribonat was published by Herlitz *et al.* Tribonat, a mixture comprising mainly of THAM and bicarbonate, was designed to overcome the previously reported disadvantages of both solutions. The study suggests that Tribonat is an effective buffer that does not have the side-effects of its parent compounds. Whilst no studies have to date been undertaken in the newborn population it is an area that could benefit from further clinical trials.

The correction of metabolic acidaemia with buffers is undoubtedly controversial but the current recommendations remain with the use of bicarbonate.

Slow intravenous administration (over two minutes) of 1–2 mmol/kg of a 4.2 per cent bicarbonate solution is justified to correct metabolic acidaemia in well-ventilated and oxygenated infants who are unresponsive to adrenaline (Burchfield 1993; BMJ Working Party 1997; Zideman *et al.* 1998).

Colloid and crystalloid

The use of albumin during resuscitation has become a common practice to 'correct acidaemia'. Roberton (1997) reviewed the use of albumin in neonatal resuscitation and suggests that albumin has little value in the correction of acidaemia *per se,* unless it is due to volume depletion. He further states that it is 'physiologically unsound', and that it may actually do further harm by overloading an already failing myocardium. Dixon *et al.* (1997) reported that whilst both albumin and bicarbonate raised the pH and reduced base excess, bicarbonate appeared to have a superior effect. If volume is deemed necessary, a further question is raised as to whether colloid or crystalloid should be used.

In 1998 Schierhout and Roberts published a systematic review of colloid versus crystalloid solutions in critically ill patients which suggested an increased mortality of 4 per cent in the colloid-treated group. This generated a 'knee jerk' reaction in many neonatal units that colloid should not be used, despite the assertion that no neonatal trials were included!

Albumin is used to redistribute water within the body rather than just replace it (Tomlin 1997) and probably still has a place in restoring circulating volume in

infants with hypoalbuminaemia. In the acute situation the use of crystalloid in the form of isotonic saline has been shown to be as effective as colloid in raising the blood pressure (So *et al.* 1997), suggesting that the infants require volume rather than protein load. Roberton (1997) further reminds of the complications of volume overload in the preterm infant with its association with bronchopulmonary dysplasia, necrotising enterocolitis, patent ductus arteriosus, and intraventricular haemorrhage, so careful assessment of its need and calculation is obviously important.

The use of colloid or crystalloid should, therefore, be used judiciously in resuscitation and reserved for volume replacement where there is evidence of acute blood loss, pallor persisting following oxygenation, poor pulses with an adequate heart rate and a poor response to otherwise adequate resuscitation attempts. The recommended volume is 10–20 ml/kg of whichever fluid is selected (BMJ Working Party 1997; Zideman *et al.* 1998) and given over a short time frame.

Dextrose

Whether to use dextrose during resuscitation of an asphyxiated infant is yet another unresolved issue, due to inconsistencies in research findings. Glucose is the predominant metabolic fuel for the immature brain, with hypoglycaemia being associated with poorer developmental outcome (Lucas *et al.* 1988). During anaerobic metabolism glucose stores are rapidly turned over, so it would appear sensible to administer intravenous glucose during prolonged resuscitation events to prevent the development of hypoglycaemia. Lundy *et al.* (1987), however, reported an increased mortality and morbidity (in dogs) when 5 per cent dextrose was administered during resuscitation, which supported the assertions of Myers and Yamaguchi (1977) that glucose infusion prior to severe hypoxicischaemic events exacerbated brain injury in the mature brain. Vannucci and Mujsce (1992) suggest that an elevated glucose level prior to an asphyxial event appears to be protective. All of these studies are of little benefit to the argument as to whether glucose should be given pre asphyxial injury or not! Studies of glucose administration post asphyxial injury are equally contradictory, with benefits and damage being reported equally (Hattori and Wasterlain 1990; Sheldon *et al.* 1992).

However, anatomical and physiological differences between the mature and immature brain also seem to have a bearing on this situation. Levene (1995) suggests the immature brain handles glucose differently to the mature (adult) brain owing to differences in the blood-brain barrier, with its rate of consumption of glucose and enzyme release restricting anaerobic metabolism of glucose to concentrations of lactic acid which are not neuro necrotic. It is clear that hypoglycaemia following asphyxia is not beneficial, but the administration of high concentrations of dextrose solutions during resuscitation cannot be recommended as good practice, and a judicious approach should be taken in its prescription until evidence exists to the contrary.

Extras

Naloxone (Narcan) is, as its trade name suggests, a *narcotic an*tagonist, and subsequently has no role in resuscitation of the hypoxic, asphyxiated infant. Its only use is in the reversal of opiate analgesia that has been given to an infant directly, or to a fetus via its mother. At birth an infant with a good heart rate and respiratory depression may benefit from a dose of 100 µg/kg. Naloxone should NEVER be given to an infant of a known (or suspected) opiate-addicted mother, as the withdrawal effects may be severe.

Calcium gluconate has been given in the past during resuscitation as it increases myocardial contractility and excitability. It is also a potent constrictor of the coronary vessels and may invoke asystole if given rapidly. It is no longer recommended in neonatal resuscitation.

Atropine is used to block the inhibitory effects of the vagus nerve, thus speeding up the heart rate. Whist this may be of benefit in avoiding reflex bradycardia during intubation and surgical procedures, it has no place in correction of hypoxia-induced bradycardia in neonatal resuscitation settings

Meconium stained liquor

Meconium stained liquor occurs in 10–20 per cent of pregnancies at term (Halliday 1992). Whilst this incidence is high, the incidence of **aspiration** is said to be 5 per cent of infants in the USA (Wiswell and Bent 1993) and 0.2 per cent of infants in the UK (Greenhough 1995). Meconium in the liquor may be an 'innocent' finding due to fetal maturational processes, often associated with post-term deliveries and often described as 'thin' meconium. It may, however, be 'thick' meconium, which is viewed as a marker of fetal hypoxia, the hypothesis being that in utero hypoxia increases intestinal peristalsis and relaxation of the anal sphincter tone.

Meconium passage is rare in preterm deliveries, and its apparent presence before this time should be viewed with caution as it may be representative of different pathology, such as listeria infection.

Meconium stained liquor, whether thin or thick, requires the presence of someone who is competent at intubation at the delivery in case this intervention is required, remembering that thin meconium may become thick during the latter stages of labour. Irrespective of whether the meconium staining is considered to be thin or thick, the oropharynx should be suctioned prior to the delivery of the infant's shoulders (Cleary and Wiswell 1998). Manoeuvres such as cricoid pressure, epiglottal blockage and thoracic compression to prevent inhalation, are not recommended as they are not scientifically tested, and are potentially dangerous as they can cause vagal stimulation, trauma and deep aspiration due to chest recoil when the encircling hands are released. The further management of the infant following delivery is dependent on its condition.

The infant who then comes out vigorous and crying is probably best left alone, as forcing cord visualisation and/or intubation is more likely to cause trauma or bradycardia and not improve the outcome (Linder *et al.* 1988).

The infant who is obviously compromised, floppy, with no respiratory effort and low heart rate, requires immediate intervention of direct vision of the vocal cords, suction and intubation to remove any particulate matter. These interventions should take place under a radiant heat source to curb massive heat loss, but *prior* to drying and stimulation in order to prevent invoking any reflex gasping respiration. Repeated intubation and suction may be necessary in order to clear the airways prior to instigation of IPPV. The heart rate will determine how many times this can be undertaken before oxygenation by IPPV should commence.

Whilst, in severe situations, meconium is probably aspirated in utero, due to hypoxic fetal gasping, this 'dual suctioning' regime, suggested by Carson *et al.* in 1976, has reduced the incidence of meconium aspiration syndrome (MAS) and brought about a significant decline in mortality (Wiswell and Bent 1993). The infants who create more controversy are the ones who fall between these two extreme situations. In these instances, astute clinical assessment is required and if the infant is less than vigorous and occasionally gasping, visualisation of the cords and aspiration is probably judicious, prior to IPPV, as the frequency of adverse complications from this procedure appears to be low (Wiswell and Bent 1993).

There is no evidence to suggest that saline lavage is beneficial in the acute management of MAS as it may dilute the meconium, creating easier dispersal throughout the lung and worsening lung function (Cleary and Wiswell 1998). The use of surfactant as a pulmonary lavage is being investigated as a potential early treatment option as it increases retrieval of meconium and increases oxygenation (Revak *et al.* 1997).

Meconium aspiration syndrome remains an important cause of neonatal mortality and morbidity (Greenough 1995). As a consequence, its active and sometimes rather aggressive management continues to be justified.

See p. 104 for further management.

Evaluation of resuscitation

The prognosis of the term infant following an asphyxial insult is difficult to determine in the delivery unit and can only really be predicted when the infant has been carefully assessed on the neonatal unit, when the degree of hypoxic ischaemic encephalopathy has been determined.

Even infants born apparently dead can be successfully resuscitated and have a normal outcome. Casalaz *et al.* (1998) reported a 36 per cent intact survival rate in a series of 42 unexpected stillbirths, a further 16 per cent are reported to have 'equivocal outcome' with mild motor development problems but with no developmental delay. Prediction of outcome in asphyxiated infants is of obvious importance to parents, and the information needs to be given as clearly and honestly as possible. The information given to parents can be more clearly and accurately given if the appearance of the infant at birth and resuscitation manoeuvres are clearly documented. The use of the numerical 'Apgar' score alone is of little value. Infants with the same scores

can have vastly differing aetiologies and therefore vastly different outcomes. Full written descriptors should accompany the scoring system if it is to be of any clinical value. Following a resuscitation event, however minor or extensive, the interventions undertaken should be carefully and accurately documented. This should provide for continuity of care by enabling the detection of problems at an early stage, and allow for improved communication and dissemination of information between members of the health care team (UKCC 1998).

Within documentation the use of the term 'asphyxiated at birth' should be avoided, as an infant who fails to breathe spontaneously immediately at birth may not have undergone a hypoxic-ischaemic insult and use of the term may cause controversies in the long term, both medically and legally (Donn 1998).

In the very low birth weight population the incidence of resuscitation increases to 80 per cent (Leuthner *et al.* 1994). Several studies indicate that infants in this category who require the full range of interventions, including cardiac compressions and drugs, in the first few days of life have a very poor prognosis, and use of such interventions may signal a cessation of active management (Lantos *et al.* 1988; Sood and Giacoia 1992; Sims *et al.* 1994; Barr and Courtman 1998). These works have to be taken on balance and on an individual basis. Ginsberg and Goldsmith (1998) suggest that vigorous attempts at resuscitation are mandated in all but a few situations, as a wait-and-see approach results in an infant with further damage from cold stress, hypoglycaemia, hypotension and, most importantly, hypoxia. The term infant who requires prolonged resuscitation is also more likely to sustain neurological injury and adverse sequelae and a guarded prognosis should be given until full neurological assessment has been undertaken.

How long should resuscitation continue?

Most hospitals will have their own policies to determine when resuscitative efforts should be abandoned. Recommendations suggest that resuscitation efforts should be discontinued if an infant has no cardiac output after 10–15 minutes, or if no respiratory effort is made by 30 minutes, when other factors for respiratory depression have been eliminated, for example, opiate depression or neuromuscular disorders (Levene 1995; Zideman *et al.* 1998). The final decision to abandon resuscitation should be made by the most senior neonatologist or paediatrician available.

Conclusion

Resuscitation events around the time of birth and within the NICU are relatively frequent, but practitioners must not become complacent because of this and feel that they are competent to practise effectively in given situations. Studies (Broomfield 1996) suggest that deskilling occurs over time and that mandatory updating sessions should be undertaken by all members of the team in order to improve practitioner skills and infant outcomes.

Case study: management of a delivery unit resuscitation

You are asked to attend the delivery of a 26-week gestation infant. What are your immediate actions prior to the birth?

A girl weighing approximately 900 g is delivered, and the liquor is heavily bloodstained. The infant is pale, limp and gasps once.

Q.1. What is your response to this situation?

Following IPPV, the heart rate is less than 80 beats per minute.

Q.2. How should you manage this?

You are asked to prepare to administer drugs to this infant.

Q.3. Which drugs are safe and of most use in this infant?

Q.4. What is the most likely outcome for this little girl?

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chapter 4

Chapter 4

Management of Thermal Stability

Contents

Introduction

The neonate has to initiate thermal control at birth. When born early, the physiological pathways necessary to commence this are not fully established; nor are there sufficient reserves in the premature infant to maintain thermal stability without compromising other body systems. To manage this situation successfully, the neonatal nurse must have a thorough knowledge of the systems involved in maintaining thermal stability and the means of assisting the infant in that vital transition time after birth, especially when the birth has been difficult or early. The role of the neonatal nurse is crucial in limiting heat loss at birth and in establishing a suitable environment in which the infant can be nursed. This caring begins before the infant is delivered by ensuring adequate facilities are available in the delivery suite, that equipment is ready for use to speed any resuscitative measures and that adequate warmth is provided (see p. 47). The nurse also has the responsibility to provide the right environment within the unit, ready to receive the infant after delivery. Without a thorough understanding of the systems of thermoregulation and the likely stage of development, dependent on the **infant's gestational age,** the nurse is not empowered to make these choices. The neonatal nurse is at the 'front-line' when it comes to thermal management of infants and it is one of the few domains of therapy over which nursing can claim to have a firm grasp.

Embryology

The fetus becomes viable (with intensive support) at 24 weeks, as the lungs become capable of supporting gas exchange. The nervous system has matured enough to direct breathing movements and assist in the control of body temperature. **Myelination** of the peripheral nerve fibres starts in the late fetal period assisting in the transmission of nerve impulses, such as the reaction to cold stimulus. The fore brain divides into the epithalamus, thalamus and hypothalamus at 5–6 weeks. It acts as a relay station to conduct impulses to and from the cerebral cortex. Hypothalamic function matures between 5 and 35 weeks' gestation and is involved with the regulation of temperature control, as well as regulating the hormones of the pituitary gland, which in turn secretes thyroid stimulating hormone (TSH) which can be detected at 10–12 weeks (Moore and Persaud 1993).

The hormonal mechanisms of thyroxine (T_4) and Triiodothyronine (T_3) are present from 22 weeks and by term are similar to maternal levels. Thyroxine stimulates enzymes related to glucose oxidation and in this way is connected to heat production (Marieb 1995). The thyroid gland also secretes type II iodothyronine 5' deiodinase, which converts T_4 to T_3, and is active by 25 weeks. T_4 levels remain low until 30 weeks because these enzyme systems are incomplete (Blackburn and Loper 1992a).

Brown adipose tissue (BAT) has been found to contain mitochondrial uncoupling protein thermogenin—the rate-limiting component of heat

production, which is present in increasing amounts between 25 and 40 weeks (Housetek *et al.* 1993). Moore and Persaud (1993) state that brown fat is being formed at 20 weeks, primarily found in the root of the neck and in the perirenal region. Biopsy of the brown fat of four preterm infants aged 25–27 weeks' gestation demonstrated that it is fairly well differentiated and thermogenetically active at this stage of development (Zancanaro *et al.* 1995). The adrenal glands are involved in thermoregulation because heat loss is controlled by the release of noradrenaline. The adrenal medulla arises from the neural crest and is therefore part of the sympathetic nervous system (Marieb 1995). The fetal adrenal gland is, by 4 months' gestation, larger than the kidney (Challis and Thorburn 1976).

The fetal adrenals secrete cortisol in response to adrenocorticotrophic hormone (ACTH) by day 50–60. This response is lost in mid-gestation but returns near term (Challis and Thorburn 1990), which is an important fact to remember when the infant is delivered before term.

Mechanisms of heat gain

The newborn infant has limited capabilities to produce heat by shivering, and when the infant is born early, poor muscular development means the neonate has no means of changing position to preserve heat. Likewise, although the infant has more sweat glands than an adult, the ability to use them as a form of heat reduction is limited as they are regulated by the hypothalamus via the nervous system, which is poorly myelinated (Moore and Persaud 1993).

The primary source of heat in the newborn is non-shivering **thermogenesis,** which involves the utilisation of brown adipose tissue to produce thermogenesis.

Vasoconstriction

Where there is a layer of subcutaneous fat, peripheral vasoconstriction can result in some reduction in heat loss, especially in full-term infants. However, in the very preterm, this layer is very thin and therefore there is little or no reduction in heat loss through vasoconstriction (Okken 1995).

Thermal receptors

Unmyelinated nerve endings penetrate the basal layer of the epidermis. These contain numerous mitochondria, which provide energy for a temperature-sensitive Na^+/K^+ pump, which changes the cold stimulus into an electrical signal. Deep body thermistors are sited in the pre-optic area and the anterior hypothalamus, which contain warm- and cold-sensitive cells (Bruck 1992).

In the infant the hypothalamus reacts to cold stimulus by causing vasoconstriction of the cutaneous blood vessels via the sympathetic nervous

system, though this is limited, plus an increase in the metabolic rate by releasing noradrenaline, and an enhancement of thyroxine release (Marieb 1995).

Non-shivering thermogenesis

Thermogenesis is initiated by three different mechanisms: cutaneous cooling, oxygenation and separation from the placenta. The cold receptors in the skin stimulate local noradrenaline release from the sympathetic nervous system to the brown adipocyte receptors. An increase in oxygen content of blood with increased flow is needed for the initiation of the system. Separation from the placenta when the cord is cut plays a pivotal role in maximising non-shivering thermogenesis (Gunn and Gluckman 1989). The mechanisms of non-shivering thermogenesis include the secretion of noradrenaline, the release of thyroxine and the metabolism of brown adipose tissue (see Figure 4.1).

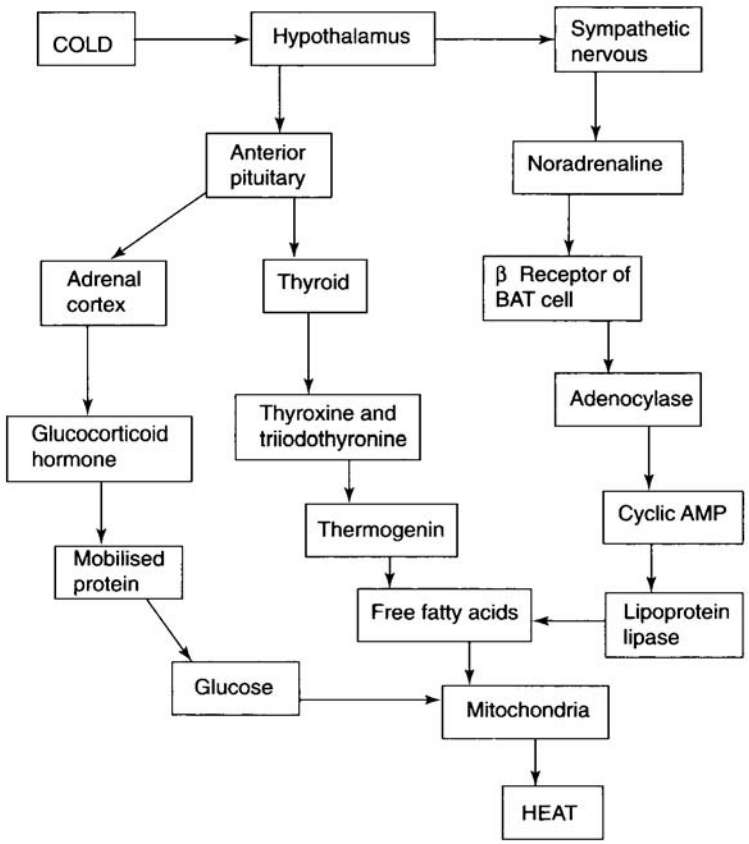

Figure 4.1 **The mechanisms of non-shivering thermogenesis**

Noradrenaline

Non-shivering thermogenesis is controlled by the release of noradrenaline from the sympathetic nervous system. Infants born before term have a substantially reduced adrenal medulla, which leaves them potentially adrenal-deficient. Cord blood catecholamines are low and the infant's ability to withstand cold stress is reduced. Noradrenaline binds to beta1-receptors increasing cyclic adenosine monophosphate (cAMP) by releasing adenosine triphosphate (ATP) from adreno cyclase. This increase in cAMP causes the release of kinase which, in turn, leads to a breakdown of the triglycerides in BAT which releases fatty acids to be combusted by the mitochondria (Nedergaard and Cannon 1992). The fatty acids are readily available on the outer membrane of the mitochondria (Nichols and Locke 1984).

The thyroid

Thyroid stimulating hormone (TSH) is released by the anterior pituitary, which is triggered by thyrotropin releasing hormone from the hypothalamus. Thyroid hormone consists of thyroxine (T_4) and triiodothyronine (T_3) which is produced by the conversion of T_4 in the liver and peripheral tissues. Thyroid hormone stimulates the enzymes concerned with glucose oxidation and binds to plasma proteins and target tissue receptors. Falling levels of thyroxine trigger TSH release (Marieb 1995).

Free fatty acids, combined with thermogenin, alter the membranes of the mitochondria to accelerate heat production. The amount of thermogenin increases with gestation, being only 29.4 pmol/kg at 25 weeks and 62.5 pmol/kg at term (Housetek *et al.* 1993). It is therefore apparent that even if there is sufficient T_4 at 25 weeks, the available thermogenin to convert it is low. Immediately after birth there is a sudden rise in serum thyroid hormone concentrations. The dissipation of these levels is dependent on the maturation at birth, calorie intake and cooling relative to the extrauterine environment. The levels of thyroid stimulating hormone do not appear to vary in sick or healthy infants, whereas the levels of T_4, free T_4 and T_3 are significantly lower in sick infants (Chen 1994).

Brown adipose tissue (BAT)

The majority of brown fat is located around the neck, between the scapulae, across the clavicle line and down the sternum. It also surrounds the major thoracic vessels and pads the kidneys. Brown fat is well suited to the production of heat energy as there are multiple fat vacuoles within the cytoplasm. Brown fat cells contain a nucleus, glycogen and the mitochondria that are used for energy production. The mitochondria are numerous and provide energy for metabolic conversion (Brueggemeyer 1993). Brown fat contains a high concentration of stored triglycerides, a rich capillary network and is densely innervated with nerve endings. The presence of thermogenin means that when fat is oxidised

heat is produced rather than energy (Sauer 1995). The substrate for BAT is thought to be free fatty acids and converted glucose. Micheli (1995) suggests that glucose is the main energy substrate in the very low birth weight infant, providing it has entered the cells. Glucose cannot enter the cells if there are low amounts of amino acids, and where these are reduced, insulin production is also lowered. This leads to the glucose transporters being down-regulated, ultimately leading to glucose intolerance and intracellular failure. In Sauer *et al.'s* study (1994) of substrate utilisation in the first days of life it was apparent that only part of the glucose infused on the first day is oxidised and that half the energy expenditure is provided by fat oxidation by days 7 and 28.

The presence of glycerol and fatty acids which are both metabolic products of lipolysis are good indicators of non-shivering thermogenesis (Power 1998). Some fatty acids enter the blood and provide metabolic fuel for a more prolonged thermogenesis.

Gluconeogenesis

Non-shivering thermogenesis is dependent on a source of energy from glucose and fatty acids; therefore, if the body is unable to generate new glucose, this source of heat production will be affected. Gluconeogenesis is the process that converts proteins and lipids into glucose for utilisation. Corticotrophin released from the anterior pituitary stimulates the release of glucocorticoid hormone from the adrenal cortex, to free proteins within the cells for metabolism into glucose. In the preterm or small-for-dates infant the stores may be depleted, thus inhibiting gluconeogenesis. Hormonal levels may also be inadequate, such as TSH and ACTH, which influence thyroxine and the adrenal cortex, which assist in gluconeogenesis (Brueggemeyer 1993).

Insulin

Insulin acts in the liver and muscles to increase glycogen synthesis and in the adipose cells it increases glucose uptake and the conversion of carbohydrate to fat. It decreases lipolysis and increases the uptake of free fatty acids. There is rapid **glycogenolysis** after birth subsequent to falling levels of insulin. There is a response from glucagon stimulating the sensory nervous system to release catecholamine which frees hepatic cAMP. Surges of glucagon and cAMP help to change the activity of the liver from glycogen to glucose production (Blackburn and Loper 1992b).

Thermoneutrality

This is defined as the environmental temperature at which minimal rates of oxygen consumption or energy expenditure occur (Roncoli and Medoff-Cooper 1992). In setting standards for neutral temperature, Sauer *et al.* (1984) describe the temperature at which infants can maintain a core temperature at rest of

between 36.7 and 37.3 °C with a change of only 0.2–0.3 °C per hour from the core and skin temperatures.

Mechanisms of heat loss

As well as understanding the physiology of heat production, it is important to be aware of the means external to the infant by which heat is gained or lost. Insulation will reduce any transfer of heat. There are two forms of insulation —internal and external. Internal insulation is provided by the layer of subcutaneous fat, which starts developing from 26 to 29 weeks' gestation. Fat is a poor conductor of heat and its depth will contribute to its effectiveness. The smaller infant has had less chance to develop this layer of insulation. External insulation is provided by the still air boundary and coverings (Thomas 1994). Transfer of heat between the environment and the infant occurs by conduction, convection, radiation and evaporation. The amount of heat transfer is influenced by the surface area of the infant and the proportions of the body in direct contact with the mattress/clothing (Sedin 1995). The amount of heat lost from convection, radiation and evaporation can exceed the infant's metabolic production, and where this occurs, the infant will remain cold in a warm environment.

Conduction

Conduction involves the transfer of heat from one object to another when they are in contact with each other, such as the infant being placed on a cool surface (Brueggemeyer 1993). Thus it is important to ensure that the delivery room is provided with a warm area with a source of radiant heat on which warmed linens are placed. This practice should be adhered to in the nursery, with particular care paid to weighing scales and X-ray plates, which, though the infant is in contact with them for a short time, induce thermal instability.

Convection

Convection involves heat loss due to the movement of air at the skin surface. The amount of loss will depend on the speed of the airflow, the air temperature and the surface area of the infant that is exposed (Merenstein and Gardner 1989). It is therefore important to remember that draughts from air conditioning and open doors as well as facial oxygen are likely to increase heat lost via convection.

Radiation

Radiation is the transfer of heat energy from the exposed surface of the infant to the surrounding surfaces. It is proportional to the difference

between these surface temperatures but independent of the temperature and speeds of the intervening air (Rutter 1992). When the wet infant is delivered in a cool delivery room the amount of heat lost through radiation from the infant's skin is high and attention must be paid to drying and warmth.

Evaporation

Evaporation is the insensible water loss from the skin's surface and the respiratory mucosa (Roncoli and Medoff-Cooper 1992). Mature infants have the capacity to increase their evaporative heat loss in response to a warm environment by sweating. Under normal conditions in a term infant evaporative heat loss is about a quarter of the resting heat production. However, the preterm infant has much higher evaporative losses as a consequence of transepidermal water loss, which is up to six times higher per unit surface area in an infant of 26 weeks' gestation (Rutter 1992).

Heat exchanged through the respiratory tract

Expired air is more humid than inspired air. This results in an evaporative loss of water and heat from the respiratory tract. There is also a small amount of convective heat transfer. As a result of the alternate inspiratory warming and expiratory cooling of the air, the convective heat exchange depends on the temperature of the inspired air (Sedin 1995).

Surface area

The newborn infant has a large surface area compared to its mass. There is an imbalance in the smaller neonate between the heat-producing ability (mass) and the heat-loss potential (surface area). This large surface area to body mass requires a high calorific intake to support temperature balance (Thomas 1994).

Immature skin

The infant of 26 weeks has developed a keratinised stratum corneum but the epidermis is only two or three cells thick. It is thought that transfer from the intrauterine aquatic environment to the external atmospheric environment stimulates and accelerates the maturation of skin (Kuller *et al.* 1990). Rutter (1989) considered that at whatever gestation the infant is born, by the time the infant is two weeks, the skin is similar to an infant born at term. More recent work has shown that in the very immature infant the development of a fully functional stratum corneum can take significantly longer than four weeks (Kalia *et al.* 1998). Histological analysis has shown that epidermal development is complete in utero at 34 weeks' gestation and infants of 30–32 weeks have a

barrier function comparable to adults. Preterm skin is more gelatinous and transparent than at term.

The stratum corneum, the outer horny layer of the epidermal barrier, conserves the body contents, resists noxious agents and protects against trauma. The immaturity of this layer means that the risks of percutaneous absorption of drugs or chemicals is increased. Immaturity also means that the skin is permeable to gases, allowing for the passive diffusion of oxygen in and carbon dioxide out along a concentration gradient (Rutter 1996).

Gestational age has a profound effect on transepidermal water loss in infants less than 30 weeks. It can reach as high as 100 g/m^2 per hour in a 24-week infant on the first day of life (Nachman and Esterley 1971). The skin of a preterm infant comprises up to 13 per cent of its body weight compared to 3 per cent in an adult.

Transepidermal water loss (TEWL)

TEWL is a physical process dependent on the epidermal barrier, the temperature, air speed and humidity. A high air speed can increase TEWL in low birth weight infants and yet when 100 per cent humidity is added, TEWL is stopped. Radiant warmers increase the TEWL by a factor of 0.5–2.0 (Rutter 1989).

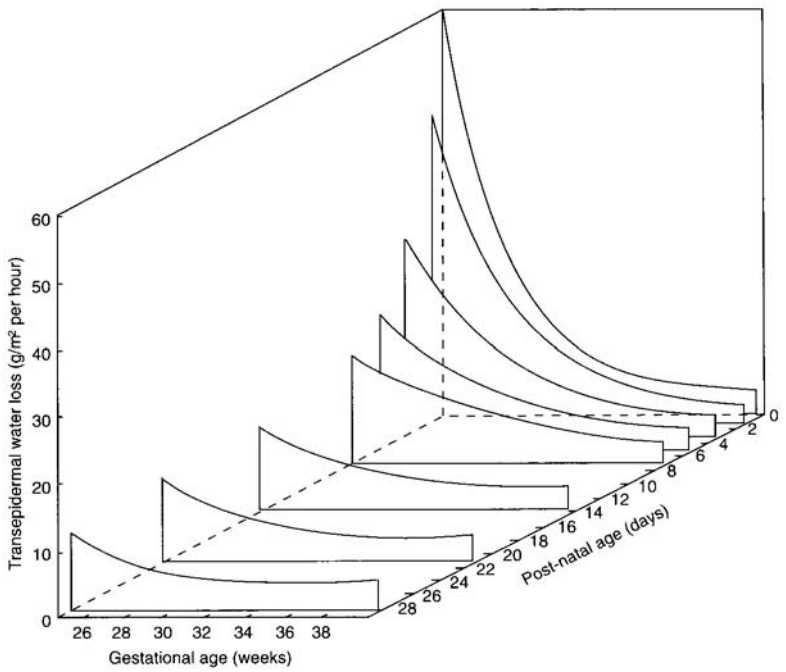

Figure 4.2 **The regression of transepidermal water loss at different postnatal ages**
Source: Hammarlund *et al.* 1983 with permission of the Scandinavian University Press

TEWL can be as much as five times higher in infants born at 25 weeks compared to those born at term (Sedin *et al.* 1983) (see Figure 4.2). Agren *et al.* (1998) showed that at 24–25 weeks' gestation, when nursed in 50 per cent ambient relative humidity (RH), TEWL was still high immediately after birth and decreased slowly but at a slower rate than more mature infants.

In 1985 Baumgart demonstrated that infants of 31 weeks' gestation (and who already had a developed stratum corneum) who were nursed under radiant heat lost most of their heat through convection, exceeding the loss from evaporation. These losses greatly exceeded the rate of metabolic heat production.

Doty *et al.* (1994) developed a model for predicting TEWL in which it was found that the predicted losses were more accurate at <28 weeks, indicating the thin layer of the stratum corneum. For each millilitre of water lost there are 560 calories of heat removed, thus rendering the very immature infant susceptible to hypothermia. Water loss diminishes with post-natal age as well as the loss of heat through evaporation (Sedin 1995).

Respiratory water loss

Sjors *et al.* (1994) studied respiratory water loss and oxygen consumption in full-term infants who were self-ventilating in air, in incubators set at 33 °C and 27.5 °C in a relative humidity of 50 per cent. This showed that not only did these infants' peripheral temperatures drop, their oxygen consumption rose and the level of carbon dioxide production increased, but that the respiratory water losses increased in lower air temperatures before there was a drop in the core temperature. Respiratory water losses in preterm infants are higher, which is thought to be due to the faster rate of breathing in the preterm infant. Full-term infants have equal losses of water from skin and respiratory tract, whereas the preterm infant loses more water through the skin (Riesenfeld *et al.* 1995).

Thermal instability

Hypothermia

The sick newborn infant is more prone to hypothermia, especially where respiratory disease is involved. Non-shivering thermogenesis requires the presence of oxygen to metabolise the brown fat. If the infant suffers a degree of hypoxia, the limited ability to produce heat is further diminished. Preterm and small-for-gestational age infants are at increased risk from hypothermia because of the high surface area to body mass ratio, low amounts of subcutaneous tissue, brown fat and glycogen stores and an immature central nervous system (Merenstein and Gardner 1989).

In a study of temperature changes during the first day of life in a busy district general hospital, it was found, not surprisingly, that it was the

low birth weight or preterm infants who became hypothermic (Johanson and Spencer 1992). Lyon *et al.* (1997) concluded that infants less than 1000 g have poor vasomotor control but start to develop vasomotor tone in the first few days of life, at which point the measurement of central-peripheral temperature difference assists by early indication of cold stress. It has been shown that in a group of term infants born small for gestational age, the reaction to thermal stimulation in skin blood flow during the first few days of life was absent; however, this is present in the term and preterm infant (Jahnukainen *et al.* 1996). It appears that the cardiovascular responsiveness of growth-retarded infants is absent in the first few days of life, which might impair their ability to meet stress (that is, changes of ambient temperatures).

Cold stress

Infants lose heat during birth, resuscitation and transportation. Cold stress affects oxygenation by increasing the pulmonary artery resistance and reducing surfactant production. Poor perfusion causes an increase in anaerobic metabolism leading to a worsening acidosis. Acidosis itself will increase the pulmonary artery pressure, decreasing the amount of flow through the lungs leading to hypoxia (Lyon and Pushner 1995). Surfactant production decreases and its ability to act as a surface tension lowering agent is impaired if the temperature drops below 35 °C (Gandy and Roberton 1987), which will give rise to atelectasis, thereby worsening the hypoxia.

The extra utilisation of glucose because of an increased metabolism can lead to hypoglycaemia, which worsens the acidosis and reduces energy available for growth. An increase in acidosis can lead to the displacement of unconjugated bilirubin from albumin binding sites causing an increase in the risk of kernicterus (Lyon and Pushner 1995) (see Figure 4.3).

Loughead *et al.* (1997) reviewed a sample of 100 very low birth weight infants at a tertiary centre and found that 45 per cent of them were hypothermic on admission and that this group of infants were significantly more likely to be acidaemic than normothermic infants.

Prolonged hypothermia with its resultant poor cardiac output and flow to the central nervous system also has an effect on the intestinal blood flow. It can cause prolonged ischaemia to the gut, which can lead to the development of necrotising enterocolitis (Powell *et al.* 1999). Pulmonary haemorrhage can be a complication of hypothermia due to left ventricular failure and damage to the pulmonary capillaries leading to fluid and cells leaking from the alveoli (Gandy and Roberton 1987) (see p. 107).

Neonatal cold injury

This occurs after a period of extreme hypothermia (below 32 °C), where a small drop in the temperature will cause a profound metabolic change. The infant

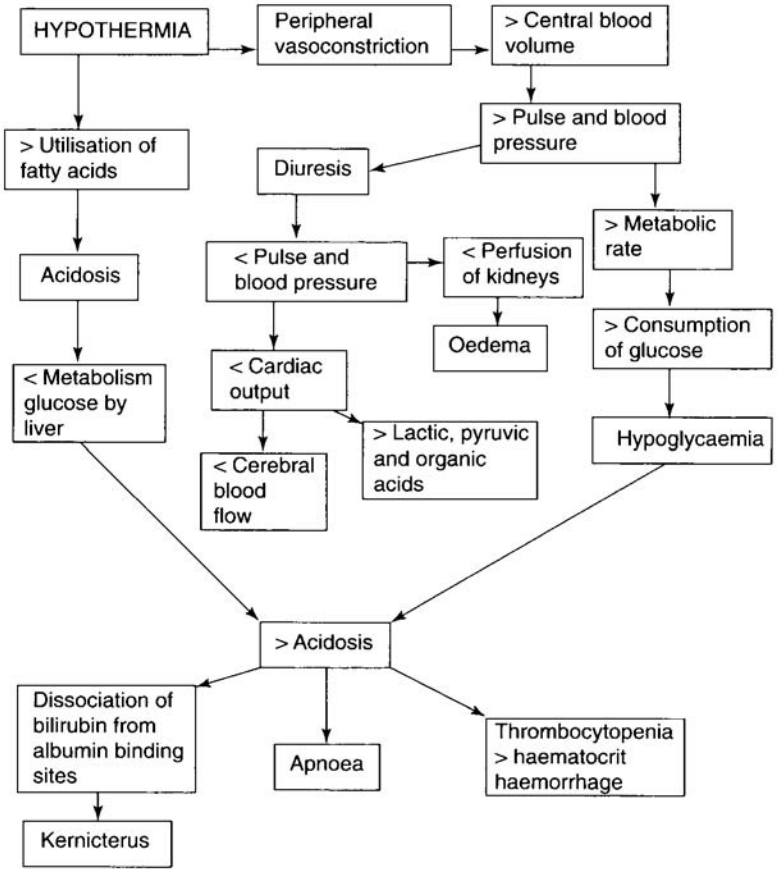

Figure 4.3 **The effects of cold stress**
Source: Based on data from Brueggemeyer 1993

appears a bright red colour due to the dissociation of haemoglobin at low temperatures (Klaus and Fanaroff 1986). The consequences of profound hypothermia are a cascade of acidosis, hypoxia, clotting disorders leading to pulmonary haemorrhage and shock with decreased cardiac output (Gomella 1994).

Re-warming in severe hypothermia

Historically, it was thought preferable to undertake gradual re-warming of the deeply hypothermic infant. Bauer and Versmold (1995) report on studies carried out by Tafari and Gentz (1974), Racine and Jarjouri (1982) and Kaplan and Eidelman (1984), which indicate a similar or decreased mortality when using rapid re-warming (>2° increase per hour). Tafari showed that those infants who received 20 ml/kg of normal saline before re-warming by whatever method had a better outcome. Racine and Jarjouri (1982) reported

that during one very cold winter 38 infants were admitted with a rectal temperature of less than 33 °C. Re-warming by the gradual method had a lethality of 70 per cent, whereas in those who received rapid re-warming, the death rate decreased to 33 per cent. Thompson and Anderson (1994) reported the case of a profoundly hypothermic 5-hour-old infant in cardiac arrest who had been in a freezer for approximately 4 hours. She had a temperature of 16.2 °C. Active internal and external warming methods were used and by 3 hours her temperature was 30.5 °C. At 49 minutes into the resuscitation she was in sinus bradycardia and by 53 minutes she moved both upper arms. By 4 months of age she had a normal neurological assessment.

One of the most successful methods of re-warming used in paediatrics has been the use of an extracorporeal circulation (ECMO) (see p. 121) as this provides rapid internal warming, allows volume replacement and oxygenation; however, this procedure is only available in a limited number of centres (Corneli 1992).

Hyperthermia

Hyperthermia in the neonate is unusual and except when the infant has a pyrexia due to sepsis, it is most likely due to inappropriate environmental settings. That is why it is important that all sick infants should have their temperatures closely monitored. If there is sepsis then there will be a large difference in the core and peripheral temperatures. Overheating can make the infant become less active or, in the case of the term infant, restless (Brueggemeyer 1993). The same factors that are responsible for hypothermia can also give rise to hyperthermia—large surface area, limited insulation and limited ability to sweat (Thomas 1994). Sweating can occur in the term infant in response to overheating, which is most readily seen on the forehead and temple (Rutter 1992). Hypotension can occur secondary to the vasodilatation, and dehydration follows an increase in insensible water loss (Merenstein and Gardner 1989) (see p. 72).

Management of thermal stability at delivery

In utero the infant temperature is at least one degree higher than that of the mother due to heat exchange via the placenta. Therefore, the drop in ambient temperature at delivery is even more marked when the wet infant is delivered into a cool environment.

The healthy infant

The healthy newborn infant is faced with a substantial drop in environmental temperature at birth and will react by increasing heat production. However, if left naked and wet in a temperature of 25 °C, the infant's heat loss will exceed heat production. This healthy infant, if dried and wrapped or dried and placed

on the mother's abdomen covered in a blanket in a room temperature of 26 °C, will be able to maintain its temperature adequately (Christensson *et al.* 1992). In a study on the effect of post-delivery care it was found that 85 per cent of the infants in an observation study had temperatures lower than 36 °C at 2 hours of age and 50 per cent still were below 36 °C at 24 hours, with 14 per cent of these still under 35 °C. In the groups where interventions (kangaroo, oil massage or plastic swaddler) were undertaken after drying, it was discovered that all three interventions were found to be equally effective. Overall only 38 per cent of infants had temperatures less than 36 °C at 2 hours and only 18 per cent were still below 36 °C by 24 hours, with none below 35 °C (Johanson *et al.* 1992).

Delivery by cesareon section

It is known that infants delivered by section are more likely to encounter problems with respiration and thermoregulation because they have not undergone the processes associated with vaginal delivery. To test the hypothesis that healthy full-term infants delivered by section have more difficulty establishing homeothermic status compared to their vaginally delivered counterparts a comparative study was undertaken. This showed that axilla and skin temperatures were significantly higher in the vaginal deliveries and that overall they were slightly warmer in the first 90 minutes after birth (Christensson *et al.* 1993).

The asphyxiated infant

In the case of the potentially asphyxiated infant, who has the potential for being more vulnerable to heat loss, further measures will be required. A pre-warmed radiant heat source is ideal for short-term use during resuscitation, but where needed for longer periods, close monitoring of the temperature is essential (Mayfield *et al.* 1984) (see p. 44).

Meconium stained liquor

It is important to remove all wet towels from contact with the infant and then place it in warm towels under a radiant heat source. However, any drying should be withheld until the status of aspiration has been ascertained. Covering the infant with a warm dry towel should suffice until the airway is cleared (see p. 56).

The low birth weight infant

This group of infants face many problems (Figure 4.4) and are the most likely to become hypothermic because most delivery rooms are not an optimal thermal environment for them. Commonly the room temperature is low, there may be

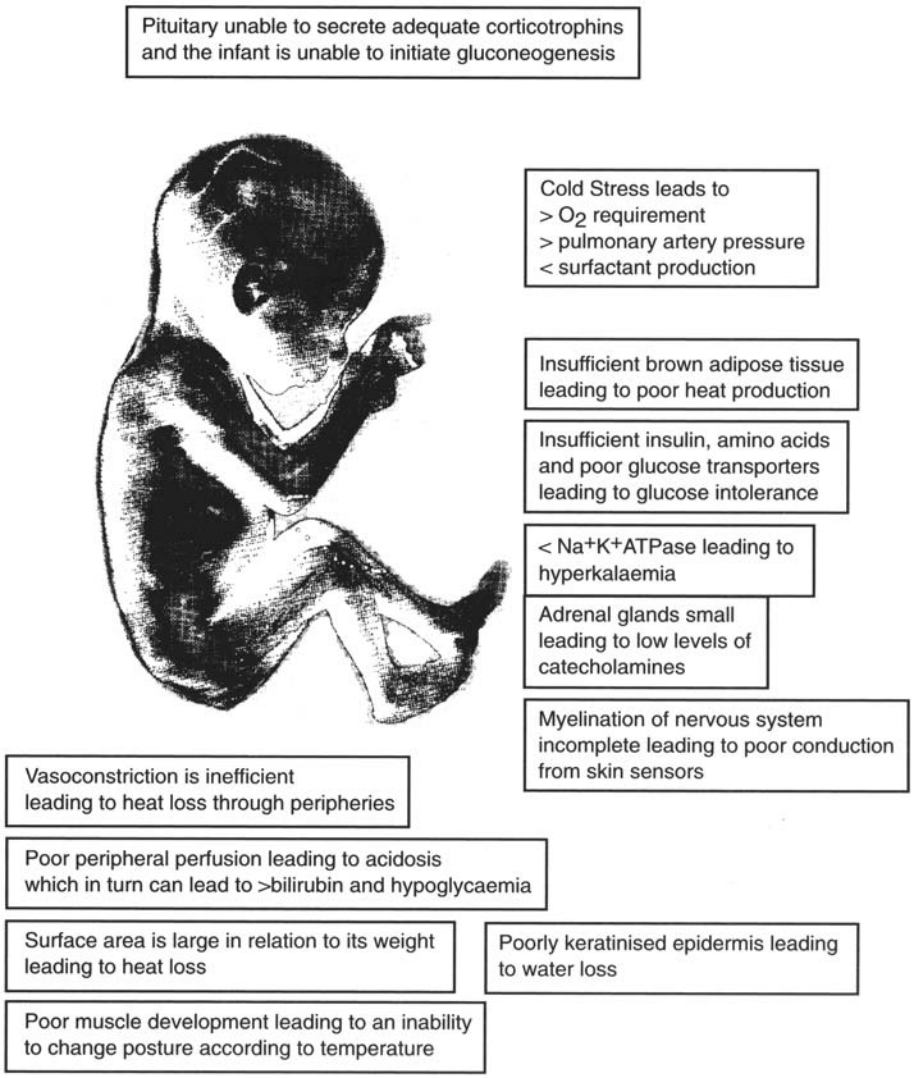

Figure 4.4 **The thermal problems facing a very immature infant**

draughts which increase convective heat loss, the air surrounding the infant has low humidity and there is usually no source for warmed gas (Bauer and Versmold 1995). The body temperature of an exposed 1 kg infant can fall at the rate of 1 °C every 5 minutes (Rutter 1992).

Extra care should be taken to ensure that the radiant warmer is set to its maximum and that warmed towels and a hat are available to limit heat loss. The relatively large surface area of the head can be a major source of heat loss, which can effectively be managed by putting on a warm hat (Koch 1995). Ensuring that all windows and doors are shut at the time of delivery will reduce the air current passing over the infant. If the

resuscitation period is prolonged, covering the infant with a sheet of polythene will reduce the TEWL until the infant is able to be placed in an incubator with humidity.

Another factor to consider with the low birth weight infant is the length of time that he or she is exposed to room temperature humidity (RTH) during the resuscitation and transfer to the NICU. Data have shown that although the monitored parameters of oxygen saturation, heart and respiratory rate remain stable during this period, there is a change in either dynamic pulmonary compliance or conductance after 5 minutes, mainly due to large airway reactivity. It is possible that long-term exposure to RTH with increases in airway resistance and resistive work of breathing might add to problems of fatigue and diminished weight gain (Greenspan *et al*. 1991).

As soon as possible the infant should be transferred to a warmed transport incubator. It is important to allow the parents to see and if possible touch their baby but this will be dependent on the condition of the infant.

It is important to remember that it is not only low birth weight infants that can become hypothermic at delivery. Those term infants who are born following asphyxia are at especial risk as well as the infants born with congenital abnormalities such as gastroschisis or exomphalos (see p. 362).

Management of thermal stability in the NICU

Once the infant has been transferred to the NICU it is vital that the correct thermal environment is provided. The neonatal nurse has a prime responsibility to ensure that heat loss is minimised and that the thermal conditions are stable for the infant. In order to provide this there are various well-developed warming devices that are at the nurse's disposal but it is important that the equipment is used appropriately. A thorough knowledge of the equipment available to maintain thermal stability will help the neonatal nurse make the right choices.

Radiant warmers

Following on from their early use in the delivery room, with their ability to limit heat loss and their ease of access, radiant warmers have now moved into the intensive nursery. Baumgart (1995) describes a radiant warmer as 'a bed platform set at waist height, with sufficient area for delivering neonatal intensive care and openly exposed to the room air', with, suspended above it, a radiant warming module, which provides infrared heat directly onto the infant's skin. These usually have the adaptability of either being used in servo control mode or heat settings can be changed manually.

Radiant warmers limit heat loss during interventions because of ease of access and rapid radiant warmer responsiveness (Seguin and Vieth 1996). During the stabilising period the infant may be subjected to many interventions. Adequate heating needs to be provided during this time, remembering that sterile towels and heads can effectively block the radiant heat source. If hypothermia is worsening a period where the infant can recover will be necessary. An unfavourable effect of a radiant warmer is that it increases the potential for increased insensible water loss and increased metabolic rate as the infant tries to produce thermal neutral conditions (Baumgart 1995). This is more often seen in the low birth weight infant of low gestational age who has a poorly developed stratum corneum. It is essential to provide supplemental humidity and shielding to counteract water loss and prevent hypernatraemic dehydration.

Metabolic rates are higher under radiant heat due to the increased rate of evaporative and convective losses. Therefore, although an open radiant warmer can warm an infant more quickly, the heat losses in the low birth weight infant are much higher and overall more fluctuant (Koch 1995).

Incubators

Incubators have the advantage of providing an enclosed space in which the infant is protected from external noise and the excesses of handling. Most modern incubators are double-walled, which reduces the heat lost through radiation from the infant. Modern incubators are able to surround the infant with a curtain of heat, and endeavour to maintain warmth even with the doors open. It is also easier to administer humidity safely and effectively through an incubator with purpose-built delivery systems. Modern incubators also have the ability to supply servo-controlled oxygen and usually have an integral oxygen monitor. Access to the infant has improved with new designs to the portholes and doors, which are usually cushioned to reduce noise impact. It is possible to control the temperature by three devices:

- Servo-controlled skin probe—if the infant's temperature falls, additional heat is provided until a target temperature is reached. Recent incubators have a skin sensor detach alarm to prevent overheating in case the probe becomes dislodged.
- Air temperature—the air temperature of the incubator is raised or lowered depending on the measured temperature of the infant.
- Air temperature probe—this hangs near the infant and maintains a constant set temperature leading to less temperature fluctuation (Gomella 1994).

A radiant hood warmer can now be added to incubator roofs to act independently of the incubator's main heat source. This device can increase skin temperature and decrease radiative heat loss in both term and preterm infants and yet not increase evaporative or convective loss (Sjors *et al.* 1997).

Radiant warmers vs incubators

There appears to be no universal approach as to whether low birth weight infants are nursed in radiant warmers or incubators. Seguin and Hayes (1997) carried out a survey of regional practice, which showed a lack of uniform approach to the methods of providing warming facilities.

There has been some concern over increased handling associated with the use of radiant warmers. In Davenport's (1992) study on hand washing, there were more interactions between nurses and infants in radiant warmers than those in incubators. LeBlanc (1982) carried out a comparative study where infants were nursed alternatively one day in an incubator and one day under a radiant warmer. The results showed that oxygen consumption was 8.8 per cent greater under radiant heat. LeBlanc concluded that an incubator provided a better thermal environment for small preterm infants. Very preterm infants exert little thermoregulatory control and variations in TEWL are a major factor in determining the appropriate thermal environment (Wheldon and Hull 1983).

Seguin and Vieth (1996) studied infants (ranging from 24 to 31 weeks with a mean birth weight of 913 g) under radiant warmers. All infants were covered in a plastic heat shield with supplemental humidity. Radiant heat did limit heat loss during interventions but in order to achieve these interventions the humidity was removed, which in its turn increased the TEWL. Evaporative heat loss in infants born after 26 weeks is three to five times higher than in the infant born at 31–32 weeks (Sauer *et al.* 1984). An ambient temperature of 37–39 °C may be necessary to minimise heat loss through convection and radiation.

Mok *et al.* (1991) studied 25 preterm infants for temperature changes during care interventions and showed that the core temperature dropped by 0.7 °C and the peripheral temperature by 1.3 °C. The difference widened by 0.6 °C after interventions.

Infants nursed with the air control mode of incubator have a more stable environment and show less variance in abdominal and peripheral temperatures than those nursed in servo-control mode as the response time of the incubator is too slow (Ducker *et al.* 1985). Servo-control is not used in some units because of the associated risks of dislodgement of the probe (if there is not a probe alarm on the incubator), direct heating or cooling of the probe and any masking of temperature changes due to hypothermia or fever. Boyd and Lenhart (1996) state that small infants expend a large amount of energy with servo-control in order to maintain temperature, whereas the air control method allows the infant to use energy resources for growth rather than heat production.

Humidity

Humidity has been shown to reduce skin water loss and improve the maintenance of body temperature (Harpin and Rutter 1985). In contrast, infants nursed without humidity are known to become hypothermic in very high incubator

temperatures. A frequent method of coping with TEWL is to increase the fluid load in order to maintain normal serum sodium (Na) concentrations. However, this is known to precipitate sequlae such as patent ductus arteriosus (Bell *et al.* 1980), necrotising enterocolitis (Grosfeld *et al.* 1996), intraventricular haemorrhage (Simmons *et al.* 1974; Han *et al.* 1997) and can increase the incidence of bronchopulmonary dysplasia (Tammela and Koivisto 1992). Preterm infants may lose up to 13 per cent of their body weight as TEWL in the first day of life when nursed in 50 per cent humidity and their losses can still be significant at 4 weeks of age (Sedin *et al.* 1985).

Older incubator systems relied on passive humidification, which led to airborne aerosols onto which bacteria could grow. Historically, because of the increased risk of pseudomonas and other bacteria (Verissimo *et al.* 1990) associated with the use of humidified incubators, the practice was halted, which increased difficulties of maintaining the thermal environment in the neutral range. Today most incubators are equipped with an active source of humidity, which heats and evaporates water separately from the circulating air and then adds it to the incubator canopy. Servo-control allows for precise levels of humidity and, because it is an active system, when the portholes are opened the recovery response is minimal as the vapour is continuously added to the circulating air (Marshall 1997). Ducker and Marshall (1995) undertook an investigation of a modern humidification system by adding pseudomonas to the water bottles. At no stage did the organism get into the incubator itself. Frembergen (1991) gives a thorough explanation of how the system works in his article on safe humidity systems.

Providing humidity via an incubator is a relatively easy task; however, the same cannot be said when trying to provide the environment using a radiant warmer. Kjartansson *et al.* (1995) studied the comparative rates of evaporation in term and preterm infants in incubators and radiant warmers. This study showed that the evaporation rates of the preterm infants were five times higher than in the term infants when nursed in an incubator at 50 per cent humidity and four times higher when nursed under radiant heat.

Units use a variety of methods to establish a micro-environment providing humidity, which involve plastic tunnels, polythene sheeting, bubble wrap, etc., usually with an adapted ventilation humidity system. This can limit the radiant heat transfer to the infant and therefore require more heat generation. It also makes it difficult to have simple access to the infant. Every time the coverings are removed for observation and attention, the infant is subjected to large swings in TEWL as the infant is suddenly subjected to direct heat and high convective heat loss (Marshall 1997). Such systems make it very difficult to establish a constant level of humidity or measure it. It requires a flow of 10 litres and a humidifier set at 38 °C to achieve a maximum of 74.5 per cent RH (Seguin 1997). Seguin also points out that when infants are nursed under radiant heat with additional humidity, it is important for practitioners to be fully aware of both the system's capabilities and its limitations. Guidelines for the humidity settings used in incubators will vary from unit to unit. A general guide should be the gestation of the infant; if less than 30 weeks the infant would require 80 per cent for 10 days. This

level of humidity would be required for longer in the case of the very preterm infant of less than 25 weeks where the stratum corneum will be taking longer to mature.

Skin

The very preterm infant has skin that is liable to break down easily and can also absorb substances. Many methods have been investigated to cover the skin in some way to reduce water loss. It has been shown that a paraffin mixture applied to the skin of preterm infants can cause an overall reduction in water loss by 40–60 per cent (Rutter and Hull 1981). A later study showed that twice-daily application of preservative-free ointment significantly reduced water loss in the first 6 hours of life. It also apparently reduced bacterial colonisation in the axilla and there were fewer positive blood and CSF cultures (Nopper *et al.* 1996). However, practical application of any topical mixture is limited in the infant who needs intensive support with lines and monitoring, and great care is needed to find a substance that would be unable to be absorbed through the skin.

A further approach to reducing water loss is to cover the skin with a semipermeable membrane. A study by Donahue *et al.* (1996) showed that semipermeable dressings caused no skin damage compared to those areas left exposed. However, this did not appear to make a significant difference to fluid or electrolyte status. There is a concern over applying such dressings in that they may harbour or encourage bacterial growth. Strickland (1997) found that there was an overall increase in coagulase-negative Staphylococcus where such dressings were used (see p. 263).

Monitoring lead electrodes and their adhesives that are used to care for these small infants can easily cause trauma, and great care is needed to avoid damage when they are removed. Lund *et al.* (1997) demonstrated an increase in TEWL after removal of adhesive plastic tape and the pectin barrier, though a period of 24 hours produced a recovery. There was less trauma associated with the removal of hydrophilic gel.

Heated water-filled mattresses

Heated water-filled mattresses (HWM) have become a useful adjunct to caring for the healthy preterm infant in the nursery. They can help mothers overcome their anxieties about their infants more easily and encourage bonding, compared to the experience of mothers of infants nursed in incubators (Sarman *et al.* 1993). It can also be better for the infants themselves. Sarman (1992) showed that resting oxygen consumption and heart rate were lower and the time to cooling of the foot during cares was also reduced in those infants on an HWM compared to those nursed in an incubator. HWMs have also proved useful in re-warming preterm infants who were able to achieve normal temperatures within the first day and remained normothermic in subsequent days (Sarman *et al.* 1989). Heated water pads have also been used in conjunction with a radiant heat source for

very low birth weight infants and it was found that this reduced conductive heat loss (Topper and Stewart 1984).

Thermal status during transport

Many studies have shown that a significant feature of neonatal transfers is the problem of hypothermia in the low birth weight infant (Hood *et al.* 1983; Vails *et al.* 1986; Hals *et al.* 1990; Smith *et al.* 1990; Lundstrom *et al.* 1993). Bowman and Roy (1997) showed that the incidence of hypothermia in the infant on arrival of the specialist neonatal team has been reduced from 22 per cent to 7 per cent over a period of 20 years. There is, however, a constant rate of 3 per cent hypothermia seen in these infants on arrival at the base hospital. Bowman stresses that these infants require continual monitoring of their temperature and a review of environmental conditions to optimise status both prior to and during transport. Bowman's figures appear similar to those of Gunn and Outerbridge (1978), who reviewed the results of infants transported to the NICU in Montreal. These figures showed that 25.2 per cent of infants were hypothermic at pick-up but had warmed by arrival, with only 3.1 per cent with a temperature of less than 35 °C.

Holt and Fagerli (1999) showed that when the retrieval team attended the delivery, the after-transfer temperature was significantly higher, indicating that an early request for the team to attend the delivery may impact on the outcome. Moving the sick or small neonate involves some inevitable heat loss due to moving between incubators at both ends of the journey, which is not easy with the very sick, much monitored infant. Ambulances are rarely as warm as the units that the infants are transferring from. Therefore the transport incubator must have an efficient heating system set up which is kept pre-warmed and ready for use (see Chapter 16).

If transferring a surgical or very low birth weight infant, heat and water loss can be limited by the use of a polythene bag in which the infant can be placed—as long as it is secured away from the neck—especially in an unventilated infant. A warmed blanket over the infant's abdomen secured under the mattress will not only limit heat loss but add to stability from vibrations whilst in motion.

A probe should be securely placed in a skin-to-mattress site (intrascapular) and if possible peripheral temperature should be monitored as well. Where possible, any existing hypothermia should be corrected before setting off on the return journey. Ventilation systems are not usually humidified on transport incubators, which may expose the infant to another source of heat loss. Neither do transport incubators themselves have humidification systems and the small infant will be at risk of high evaporative heat losses. There is no even distribution of inner wall temperature, which can lead to heat loss through radiation (Sedin 1995). Some transport incubators have the facility of double-walling, which helps reduce heat lost by radiation. If the ambulance heating is on maximum and there is still a degree of hypothermia, covering

the roof of the incubator with a blanket will help reduce some of the heat loss.

Temperature measurement

Continuous monitoring

All sick and small infants should have their temperatures measured continuously. A range of electronic devices are available to measure temperature. These use thermistors relying on changing electrical resistance and the conduction properties of metal when heated. There are also a variety of probes which can be used and many studies have been carried out to find the most appropriate sites to record temperatures (Mayfield *et al.* 1984; Bliss-Holtz 1993; Thomas 1993; Dollberg *et al.* 1994; Lemburg 1995; Simbruner 1995; Lyon *et al.* 1997). Lemburg (1995) concluded that the rectal temperature was of least value, as readings can vary as much as 3 °C (depending on the position of the instrument) and are also influenced by the temperature of the blood returning from the lower limbs.

Clinical assessment of hypovolaemia in sick, preterm infants is difficult and can be improved by monitoring core and peripheral temperatures (Lambert *et al.* 1998). Monitoring core and peripheral temperature (c-pT) can be particularly useful in giving an early indication of cold stress in this group, who have poor vasomotor control at birth (Lyon *et al.* 1997). Lemburg (1995) states that it is important to record a two-point measurement as less problems of care occur when using this continuous assessment. Low core temperatures can indicate significant thermal stress. A wide c-pT (>2–3 °C) is abnormal and might be caused by hypovolaemia, cold stress, catecholamine infusions, infection or patent ductus arteriosus (McIntosh *et al.* 1995). Mayfield *et al.* (1984) found that preterm infants had lower temperatures and these varied less with the site of measurement, suggesting a smaller core-surface gradient because of the comparative lack of thermal insulation by body fat. Therefore measurement of the temperature between the skin and mattress is nearly as accurate as other more frequently used methods.

Dollberg *et al.* (1994) investigated the use of zero heat flow temperature sites. The principle relies on the fact that any body has an internal heat-producing component and has a continual heat flow to its surface as long as the surface is cooler than the heat-producing component. Zero heat flux mimics the rectal temperature closely, and is suggested as an appropriate site for measuring core temperatures. Skin-to-mattress (intrascapular) temperature measurement was the most useful site for measuring the core temperature since it is non-invasive and there is no gradient to the oesophageal temperature (Lemburg 1995). Although an oesophageal reading would give an accurate core temperature there are at present no probes suitable for long-term use and it remains invasive. The use of catheters with thermistors attached placed in a central artery are coming

into practice in the NICU but this remains extremely invasive and only for use in the critically ill infant.

Taking axillary temperatures has been a method of choice in the neonate for some time. The difficulties with siting probes in this area are that the infant very often moves the arm and the preterm infant often lies in a relaxed posture with the arms above the head. This can lead to heat flux from the site of the probe and an inaccurate measurement. Axillary temperatures alone give a poor indication of brown fat activity and those infants that are compensating through non-shivering thermogenesis may not get the environmental support needed (Bliss-Holtz 1993).

Intermittent temperature recordings

Intermittent temperature recordings have traditionally been performed with a mercury thermometer, which is known to be hazardous and a practice that should be eradicated (Pazart *et al.* 1997; Smith *et al.* 1997; Thigpen and Sexson 1997). Recently, the infrared sensors have come into common use. These work on the basis that solid objects emit an intensity of infrared radiation, which corresponds to specific temperatures. Pyroelectric sensors detect the heat flow and give a very quick reading (Thomas 1993). This technology is used widely in tympanic thermometers in adults and paediatrics and has also been introduced for axillary use. The tympanic thermometer has been introduced for neonatal use but studies have shown that the measurements vary depending upon the environment in which the infant is nursed (radiant warmer, incubator or cot) (Hicks 1996; Cusson *et al.* 1997). Therefore it is important to assess the likely environmental effects when choosing the method of measurement and to pay particular attention to the size of the infant in which it is used.

The axillary infrared device has been specifically designed for use in neonates (O'Toole 1998) and appears to give an accurate, quick assessment of the axillary temperature but will require comparative studies. Rectal temperatures have been used for many years and considered the ideal core temperature in neonates, but the procedure is not without risks (Fonkalsrud and Clatworthy 1965; Merenstein 1970; Fleming *et al.* 1983) and it has been shown to not always reflect a true core temperature (Lemburg 1995).

Localised hypothermia following birth asphyxia

Hypothermia is usually viewed in a negative context, with neonatal nurses devoting a great deal of attention to keeping infants warm. Recent research (Thoreson and Wyatt 1997; Gunn *et al.* 1998) indicates, however, that for the infant who has received an asphyxial insult there may be a therapy that involves the use of localised hypothermia.

Following the resuscitation of an infant with birth asphyxia, in the past therapies have been supportive. There is now hope on the horizon

that localised hypothermia, if established within the first few hours of birth, could have a big impact on improving outcomes. The talk of a 'therapeutic window' means that within a certain time frame it may be possible to 'attenuate activation of the neurotoxic cascade that leads to cell death hours, days or months later' (Robertson and Edwards 1998). Reducing the brain temperature by 2–6 °C for 3–72 hours after resuscitation has been shown to reduce brain damage by 25–80 per cent in animal studies. It is thought that the protective mechanisms are a reduction in extracellular excitotoxic amino acids, reduced nitric oxide synthesis and an inhibition of apoptosis (Thoreson and Wyatt 1997). In the study conducted by Gunn *et al.* (1998) cooling was established by circulating water at 10 °C through a coil of tubing wrapped around the head for up to 72 hours whilst otherwise being nursed in a cool thermal environment (core temp 35.7 °C). It was found to be a safe and convenient method of quickly reducing cerebral temperature. Animal studies into the effects of hypothermia indicate an improved outcome (Gunn *et al.* 1997; Bona *et al.* 1998; Huang 1998). It is now necessary to set up clinical trials on a wider basis so that the value of localised hypothermia can be established and be brought into regular usage. However, establishing a trial could prove difficult, as there are many problems to be overcome, such as selecting the group of infants for the control group (Edwards and Azzopardi 1998).

Conclusion

The neonatal nurse needs to have a working awareness of embryology and physiology, especially when caring for the very preterm infant. In the infant born so early the systems that are involved in thermal regulation are limited and poorly functioning. Knowledge of the methods of heat production and the mechanisms of heat loss will enable the nurse to give adequate provision to manage the infant in a thermally stable environment. Prevention of heat loss at delivery is a major component of the nurse's role, especially where resuscitation procedures are prolonged. However, the nurse also needs to be aware that cold air acts as a stimulant to the initiation of respiration, therefore is it possible to achieve a balance? Choosing the thermal environment in which the infant is to be cared for, the appropriate monitoring of the temperature and the delivery of humidity as required are all part of the nurse's responsibilities and practice should be evidence-based. An awareness of current research should be incorporated into the care delivered, putting an end to 'traditional practices' that might not be appropriate. Investigating the use of incubators for preterm infants and delivering the correct amount of humidity for the correct length of time dependent on the gestation of the infant should be a prime topic and many articles are written on the subject. The nurse should be aware of why particular management is suitable for the infant being cared for, and be able to make appropriate changes based on sound knowledge.

Case study: effects of temperature instability in a preterm infant

An infant was admitted from an outlying unit. Owing to maternal hypertension, she had been born at 26 weeks and weighed 700 g. Following resuscitation she was admitted to the NICU with an axilla temperature of 34 °C.

Q.1. What are the potential reasons for this?

Q.2. What are the potential side-effects?

Q.3. What can be done to prevent this situation occurring?

By the time the flying squad team arrived the temperature was 36 °C axilla. At this point she was nursed under radiant heat, had adequate arterial and venous access, had a mean blood pressure above 35 mmHg, normoglycaemia with 10 per cent glucose IV and was ready for transfer. On arrival at the unit her temperature was 36 °C. As her general condition was good she required minimal handling in the next few hours. However, her temperature control was poor. She was nursed under a radiant warmer with the under-bed heating set at maximum. The warmer was used in the manual mode, initially set at 38 °C but later increased to maximum and initially she was covered with a plastic sheet for the first 2 hours but then placed in a humidified tunnel.

During an initial 4-hour period of observation there were seven incidences of handling, during which the humidity and covers were removed. At this time there were wide swings in the temperature between 36 °C and 38 °C (intrascapular). During a later 3-hour period of observation there were only two incidences of handling and the humidity was left in place. Her temperature remained fairly constant at 36.5–37 °C in an environmental temperature of 36.8 °C with added humidity.

Q.4. Why had these swings in temperature occurred?

Q.5. What could be done to manage this infant in a more stable environment?

Q.6. The following day her sodium levels were 146 mEq/l. What had caused the sodium to rise?

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Chapter 5

Management of Respiratory Disorders

Contents

Introduction

This chapter focuses on the management of infants with respiratory disorders. It will examine fetal and neonatal lung development as well as revisiting some of the common respiratory disorders with a view to applying this knowledge to the decision-making process in the management of these infants. The chapter will also explore methods of respiratory support used in the UK today as well as considering the potential impact of newer technologies on the management of infants with respiratory disorders. The concept of evidence-based care will be employed in this chapter as it is elsewhere in the book. However, it will become apparent that much of the practice in respiratory management is based on small-scale studies that lack the rigour of randomised controlled trials. For this reason, the authors have tried to reflect the variety of practices used and highlight the outstanding questions that remain to be answered in respiratory management of the neonate.

The development of the respiratory system (Figures 5.1, 5.2)

The development of the fetal lungs impacts directly on the pathophysiology of any respiratory disorder and influences the management of care. The development of the lungs is usually divided into five stages (Hodson 1998).

Stage 1 is the embryonic stage which starts at conception and ends at the seventh week of gestation. In this stage the laryngotracheal groove develops from the foregut and a septum begins to form to separate the trachea from the oesophagus. If the septum does not develop completely, then a tracheoesophageal fistula will result. The primitive bronchi also develop at this time.

Stage 2 of lung development lasts from week 8 to week 16 and is known as the pseudoglandular stage. During this time a network of narrow tubules develops—this will eventually become the part of the lung from the trachea to the terminal bronchioles. During this stage some of the connective tissues, including cartilage, muscle and blood vessels, start to develop.

Stage 3 lasts from weeks 17 to 27 and overlaps with stage 2. It is known as the canalicular stage. During this time a rich vascular supply proliferates and the capillaries are brought closer to the airway epithelium. Primitive respiratory bronchioles also develop.

Stage 4 is known as the terminal air sac stage and lasts from weeks 28 to 35. The terminal air sacs multiply and the surface epithelium of the air sacs thins, bringing the capillaries in closer contact with the airsacs.

The final stage, stage 5, is known as the alveolar period and involves the development of airsacs. It continues after birth and throughout childhood until about 8 years of age.

The epithelial lining of the lung consists mainly of two cell types—type I and type II pneumatocytes. The type I pneumatocyte covers almost 95 per cent of the alveolar surface while the type II pneumatocyte is more numerous but covers only

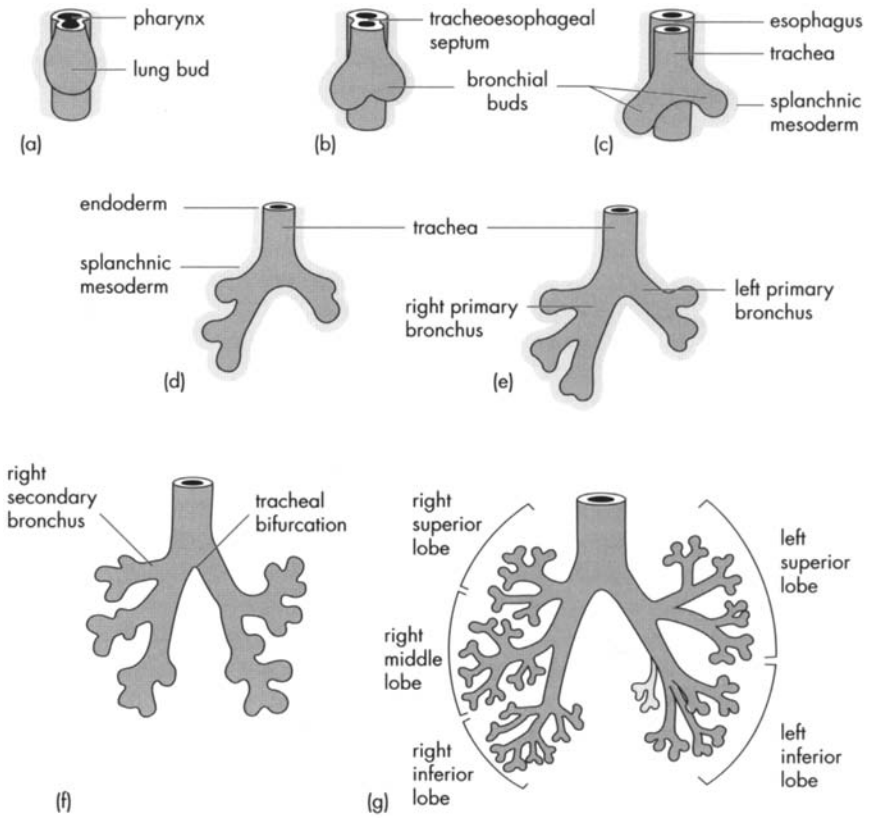


Figure 5.1 **Fetal lung development. Successive stages in the development of the bronchi and lungs: (a–c) 4 weeks; (d and e) 5 weeks; (f) 6 weeks; (g) 8 weeks**

Source: after Moore, K. and Persaud, T.V.N. (1993) *The Developing Human—Clinically Oriented Embryology,* 5th edn, Philadelphia: W.B.Saunders, with permission

5 per cent of the alveolar surface. It is believed that the type II pneumatocytes are responsible for surfactant production (Rooney 1998). Surfactant is an important factor in determining the surface tension in the lung. In particular, it prevents the lung deflating completely at the end of expiration. This facilitates gas exchange between the alveolar air space and the alveolar capillaries. The presence of surfactant also decreases the work of breathing. Although surfactant secretion is detectable between weeks 25 and 30, the amount of surfactant produced is insufficient to produce alveolar stability until the infant is between 33 and 36 weeks' gestation.

A series of enzymes and hormones regulate the synthesis and secretion of surfactant. Glucocorticoids, for example, appear to be able to accelerate the normal pattern of lung development—hence the reason for giving dexamethasone to women at risk of preterm labour (Crowley 1999). Catecholamines also appear to be able to increase the amount of surfactant in the lung. The catecholamine

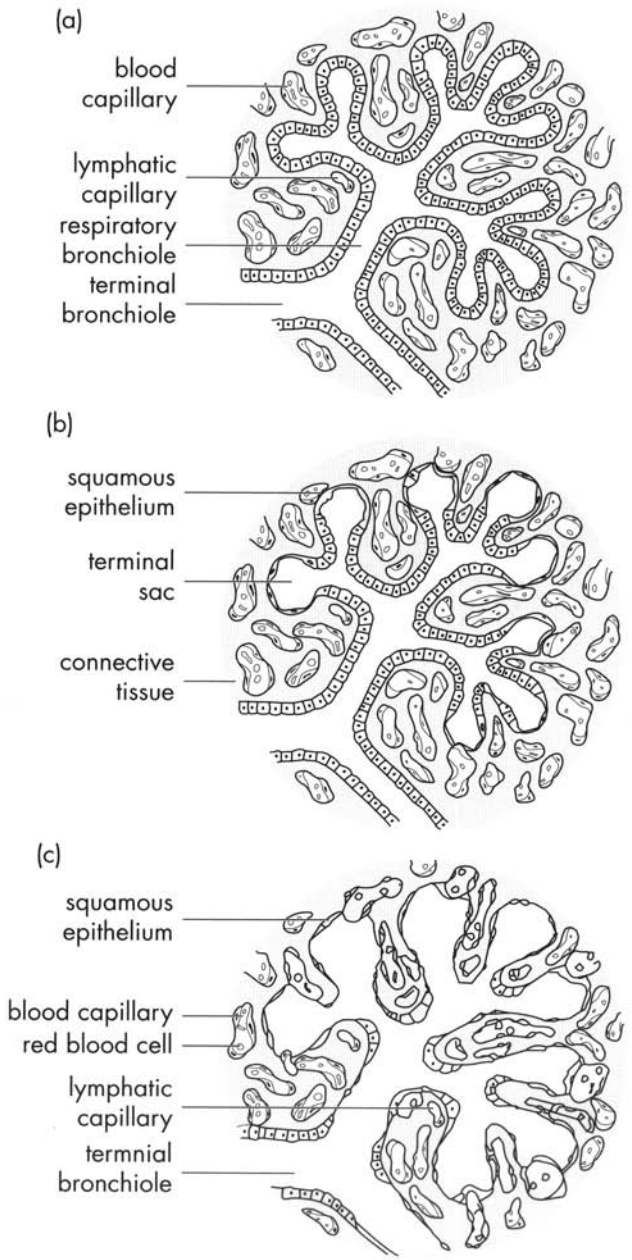


Figure 5.2 **Histological sections illustrating progressive stages of lung development: (a) Late canalicular period (about 24 weeks); (b) early terminal sac period (about 26 weeks); (c) newborn infant (early alveolar period)**

Source: after Moore, K and Persaud, T.V.N. (1993) *The Developing Human—Clinically Oriented Embryology, 5*th edn, Philadelphia: W.B.Saunders, with permission

response is increased in some infants under stress and may speed up the synthesis of surfactant, for example in SGA babies (Rooney 1998). Insulin, on the other hand, appears to inhibit surfactant synthesis. Infants of diabetic mothers are at increased risk of developing respiratory problems because of the lack of surfactant (Rooney 1998). Fortunately, the problems are usually transitory—particularly if the baby is born near term.

Hypothermia and acidosis are associated with the suppression of surfactant production and may hinder recovery (Gluck *et al.* 1972). In order to maximise the infant's chances of survival it is important to maintain the infant's temperature within a narrow temperature range (36.8–37.2 °C) and correct disturbances of acid base balance (see p. 74).

From the beginning of the canalicular stage the lungs secrete fluid which fills the alveoli and bronchial tubes. It appears to be important in cell maturation and development, as well as determining the size and shape of the developing lungs (Bland 1998). Fetal breathing movements can be seen from about 12 weeks' gestation. The strength and frequency of the movements increase as gestation increases. It is thought that fetal breathing, along with fetal lung fluid, assists in the development of the diaphragm and chest wall muscles. Conditions such as oligohydramnios are associated with a decrease in fetal breathing movements and can lead to pulmonary hypoplasia (Sherer *et al.* 1990).

Respiration in the neonate

The infant has a total lung volume of approximately 63 ml/kg (Davis and Bureau 1987). The tidal volume (the volume of gas inspired with each breath) is approximately 6–8 ml/kg, although it varies with gestational age. The functional residual capacity (the amount of gas remaining in the lung at the end of expiration) is established in the first few breaths. The functional residual capacity is low in infants with lung disease and this will compromise alveolar stability. The functional residual capacity increases as the lung disease resolves.

The amount of gas taken in by the infant depends on the respiratory muscles. The diaphragm is prone to muscle fatigue because of the lack of fatigue-resistant fibres (Davis and Bureau 1987). These increase as the infant grows postnatally. Preterm infants are particularly prone to diaphragmatic muscle fatigue. The compliant rib cage means that infants must work harder to move gas into and out of the lung and this means that they are more prone to developing lung collapse.

A baby born at 28 weeks' gestation or less will have very few alveoli and surfactant synthesis will probably be insufficient to ensure alveolar stability. The lack of surfactant causes alveolar collapse at expiration. The alveolar walls will still be quite thick, so gas exchange will have to take place across a thick wall leading to a decrease in the amount of oxygen in the blood. The diaphragm and the intercostal muscles will still be developing and this may mean that the infant's respiratory efforts will be inadequate to sustain life (Davis and Bureau 1987).

Respiratory distress syndrome (RDS)

RDS occurs because of a primary deficiency of surfactant (Avery and Mead 1959). It is a major cause of neonatal mortality and morbidity—although some of this is not directly caused by the pulmonary pathology but rather by the complications associated with extreme prematurity, such as intraventricular haemorrhage (see p. 156) and renal failure (see p. 224). The lack of surfactant causes diffuse atelectasis and high pressures are required to reinflate the lungs. The disease can cause ventilation-perfusion abnormalities and this, together with the movement of fetal lung fluid through the lungs, causes the erosion of the broncheolar epithelium and results in the formation of cell debris (Jobe 1998). The serum proteins in the lungs combine with fibrinogen to form hyaline membranes—hence the name hyaline membrane disease. The hypoxia associated with the condition can cause right-to-left shunting through the foramen ovale and left-to-right shunting through the ductus arteriosus. The altered respiratory physiology caused by the abnormal circulation contributes to a failure in transition from intrauterine to extrauterine life and this can lead to pulmonary ischaemia, which causes further damage to the developing lungs.

The onset of RDS occurs within 4 hours of birth. The infant may have an increased respiratory rate and may develop an expiratory grunt which is caused by forcing air past the partially closed glottis in an effort to retain some air in the alveoli at the end of each breath to prevent atelectasis. As the infant becomes more compromised, the effort of breathing increases and this may be manifested by nasal flaring, intercostal and sternal recession. Eventually the infant may become cyanosed and apnoeic. On auscultation breath sounds are diminished because of poor air entry.

The diagnosis of RDS can be confirmed by the history, blood gases demonstrating impaired respiratory function (lowering of the oxygen content, hypoxia, and increasing carbon dioxide levels, hypercarbia) and a chest X-ray that shows the classic 'whiteout' features of RDS. It may be difficult to differentiate between RDS and congenital pneumonia so it is common to give infants with the clinical signs of RDS prophylactic antibiotics until a negative infection screen is achieved (Greenough and Roberton 1996a).

The aim of treatment is to provide respiratory support until the type II pneumatocytes regenerate and start to produce surfactant. Infants with RDS are prone to developing a number of complications and care is planned to reduce the incidence and severity of many of the associated complications, such as intraventricular haemorrhage, necrotising enterocolitis (see p. 366) and chronic lung disease.

Respiratory complications are managed by providing respiratory support. Supplemental, warmed, humidified oxygen may be sufficient for infants with mild RDS. Babies with more severe forms of the disease may require nasal continuous positive airway pressure (CPAP) or mechanical ventilation. There is debate about the most effective form of management and a study is currently in progress to assess the effectiveness of nasal CPAP versus intubation and positive-pressure ventilation as a primary intervention in RDS (Subramanian *et al.* 1999).

Mechanical ventilation is usually the treatment of choice where there is a risk of the infant developing severe RDS. This is indicated by increasing respiratory rate, sternal and intercostal recession and altered arterial blood gases showing worsening hypoxaemia or hypercapnia. The use of high frequency ventilation (HFV) in RDS is discussed later in the chapter.

Exogenous surfactant therapy has been shown to reduce the severity of respiratory distress syndrome (Soll 1999a). It is also associated with a decrease in complications associated with RDS, including pneumothoraces. The OSIRIS trial (OSIRIS Collaborative Group 1992) demonstrated that early administration of exogenous surfactant was more effective than using it as a rescue treatment. However, questions remain about the most effective dosing regime (Soll 1999b). A systematic review of the effectiveness of the different types of surfactant by Soll (1999b) suggests that natural surfactants are currently more effective than synthetic surfactants in reducing the need for ventilation and reducing the number of pneumothoraces. However, there is little evidence available to suggest that these differences have any long-term benefit. However, surfactant studies are continuing and more information will become available as the infants mature and are followed up.

Administration of surfactant should be undertaken by experienced personnel and the manufacturer's instructions should be followed to reduce the possibility of untoward effects. The administration of surfactant is associated with a reduction in the need for supplementary oxygen and this is frequently accompanied by an alteration in ventilation requirements (Long *et al.* 1996). For this reason, someone who is experienced in ventilator management should be readily available when surfactant is being administered.

Infants with RDS may have a patent ductus arteriosus (PDA) (see p. 140) due to their immaturity and the intermittent hypoxia which they experience (Greenough and Roberton 1996a). Positive-pressure ventilation is associated with an increased incidence of PDA (Ahumada and Goldsmith 1996). This may be due to the pressures generated in the lung which cause the altered blood flow through the immature pulmonary tree and it may also be partly due to the hypoxia and associated pulmonary vasoconstriction (Jones and Deveau 1997). There is continuing debate over the management of PDA and this will be covered in more detail in a later chapter (see p. 141). Because of the poor tissue respiration, infants with RDS are prone to metabolic disturbances. In particular, they may become very acidotic. This may be corrected with good ventilator management but a persistent acidosis may require treatment with an alkali such as sodium bicarbonate or THAM. The following formula will reduce the base deficit by half:

$$\frac{\text{Base deficit} \times (\text{weight in kg}) \times (0.3)}{2}$$

It should be noted that different units may use a different formula but the aim of treatment is to bring about partial correction of the acidosis. Attempting full

correction of the acidosis is dangerous as this can lead to alkalosis. This should be administered slowly over 30–60 minutes as there is evidence that rapid infusion of base can cause cerebral vasoconstriction and lead to intraventricular haemorrhages (Levene *et al.* 1982).

Preterm babies are at risk of anaemia because of the shorter lifespan of fetal red blood cells and the repeated sampling which takes place during the acute phase of the illness (see p. 181). Maintaining a haemoglobin above 13 g/dl in ventilated infants will help reduce mortality so frequent blood transfusions are necessary (Roberton 1987). Hypoxia and infection increase the risk of infants with RDS developing clotting disorders such as disseminated intravascular coagulation (see p. 180), so clotting screens will be required at regular intervals.

The natural course of RDS is that the infant starts to recover after 48–72 hours as surfactant production increases and the need for ventilatory support decreases. However, problems associated with extreme prematurity may persist. Provided that associated complications such as intraventricular haemorrhages are avoided, the prognosis is good. However, infants who require prolonged ventilator support or who are less than 27 weeks' gestation are more likely to experience severe and persistent complications (Tin *et al.* 1997).

Pulmonary interstitial emphysema (PIE) is the result of air leaking from ruptured alveoli into the interstitial tissue and is strongly associated with the use of positive-pressure ventilation. It can adversely affect the pulmonary circulation and alveolar ventilation. The diagnosis can be suspected in a baby who is difficult to ventilate, with rising carbon dioxide levels and an increasing need for oxygen. However, the diagnosis is made on the basis of chest X-rays. These show cyst-like formations and linear radiolucencies. In severe cases, the lungs may appear like sponges. Treatment is difficult. An attempt can be made to reduce inflation pressures and high frequency ventilation may be useful, although evidence to support its use is limited.

Pneumonia

Pneumonia in the neonate can occur as a result of coming into contact with infective agents during the intrapartum period or acquired postnatally. Group B Streptococcus is one of the most common causes of acute neonatal pneumonia (Outerbridge *et al.* 1996) (see p. 269). The clinical features of pneumonia are similar to those in an infant with respiratory distress syndrome. The maternal history can assist in providing a differential diagnosis. A history of prolonged rupture of membranes, offensive liquor or maternal infection with Group B streptococcus should be regarded as suspicious.

Broad spectrum antibiotic treatment should be initiated as soon as an infection screen has been done. Supportive therapy—supplementary oxygen or ventilation—should be given according to the baby's needs.

Meconium aspiration syndrome (MAS) (Figure 5.3)

Meconium aspiration syndrome is a problem associated with term babies. Greenish liquor in preterm deliveries is more likely to be associated with infection by *Listeria* monocytogenes.

Meconium is made up of bile salts and epithelial cells from the fetal gastrointestinal tract. If peristalsis is stimulated in utero, by hypoxia for example, the anal sphincter may relax and the fetus will pass meconium into the amniotic fluid. While aspiration can occur at any time, the risk is greatest if the infant experiences intrauterine asphyxia. This results in gasping movements which will cause the meconium to pass from the amniotic fluid into the fetal lungs (Roberton 1996).

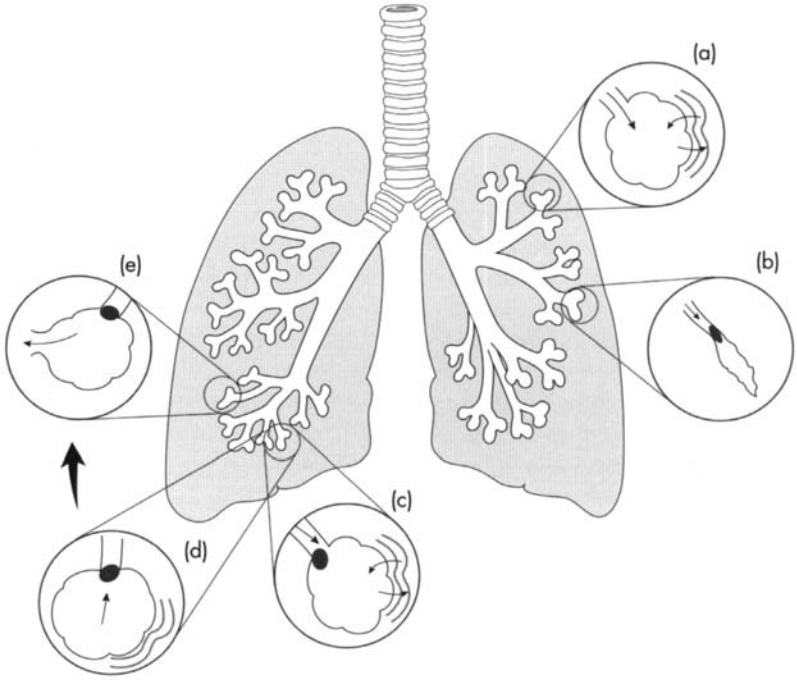
Following resuscitation (see p. 56), the infant may be profoundly hypoxic and auscultation of the chest will reveal rales and rhonchi. The chest may appear hyperinflated or barrel-shaped and the chest X-ray may show areas of hyper-expansion with patches of atelectasis caused by meconium blocking the alveoli. The infant may also show signs of respiratory distress, including tachypnoea, nasal flaring and sternal recession. In severe cases of MAS, the blood gases will show evidence of respiratory and metabolic acidosis. There may be a low PaO_2—even in high concentrations of inspired oxygen.

Infants with MAS may show circulatory changes. They are prone to systemic hypotension as well as persistent pulmonary hypertension of the newborn (PPHN). This is characterised by cyanosis and shunting of deoxygenated blood through the ductus arteriosus and foramen ovale. The rise in right-sided heart pressures causes a right-to-left shunt and this increases the hypoxia and thus the cycle of hypoxaemia and hypertension continues (see p. 136).

The aim of treatment is to correct the hypoxia and acidosis as this will facilitate pulmonary vascular dilatation (Greenough and Roberton 1996b). The infant may try to breathe against the ventilator, thus increasing the risk of pneumothoraces. To avoid this, sedation may be necessary in conjunction with a muscle depolarising agent such as pancuronium, to paralyse the respiratory muscles.

Infants who are heavily sedated or who have received muscle depolarising agents will be unable to move and are at risk of fluid retention and oedema, as well as pressure sores. Nursing care will include careful positioning to prevent undue pressure or unnatural positions of limbs and passive limb movements to encourage circulation. Eye care is important as the blink reflex will be suppressed by the muscle depolarising agents. Bladder expression may be required if the infant is unable to pass urine.

Oxygen is a potent pulmonary vasodilator and so high concentrations of inspired oxygen are used as the risk of retinopathy developing in infants born near term is low. Exogenous surfactant replacement therapy has been used in MAS but as yet there are no large studies to support its use (Auten *et al.* 1991; Khammash *et al.* 1993).



(a) Normal alveolar function and gaseous exchange
(b) Complete obstruction resulting in atelectic collapse
(c) Partial occlusion allowing gas in on inspiration
(d) Total obstruction of airway on expiration leading to gas trapping over distension and vascular impingement
(e) Over distension leading to rupture and air leak

Figure 5.3 **Schematic representation of the effects of meconium on pulmonary function**

Chemical pneumonitis can be a feature of MAS. This is caused by the presence of organic material in the alveoli and can lead to exudation of fluid through the damaged capillary walls, thus increasing pulmonary oedema while at the same time exacerbating systemic hypotension.

Infants with MAS are prone to metabolic disturbances. In particular they may become very acidotic. This may be corrected with good ventilator management but a persistent acidosis may require treatment with a base such as sodium bicarbonate. A respiratory alkalosis may also aid pulmonary vasodilatation so hyperventilation to reduce the $PaCO_2$ may also be indicated. Because infants with MAS and PPHN are often very unstable, it is important to keep any interventions to a minimum as handling may cause a fall in the PaO_2 and further exacerbate the pulmonary vasoconstriction. If there has been severe asphyxia then other pathology may develop. This includes neurological, hepatic and renal damage (Roberton 1996).

Infants with MAS and PPHN will require intensive monitoring of vital signs, including temperature, heart rate and blood pressure. Arterial blood gases should be checked regularly and oxygenation should be monitored constantly using an indwelling transducer or pulse oximetry and transcutaneous monitors. Although the baby may have pulmonary hypertension, the systemic blood pressure may be low because of the impaired cardiac output. This means that the baby is at risk of developing renal failure. Urinary output should be carefully monitored and the intake should be adjusted accordingly (see p. 218).

Drugs may be used to increase the systemic blood pressure and alleviate the pulmonary vasoconstriction to decrease the shunting of deoxygenated blood. Inotropes can be used to support the systemic blood pressure (Emery and Greenough 1993). These drugs assist by increasing the cardiac output. Vasodilators that act on smooth muscle can be used to dilate the pulmonary arteries (Goetzman *et al.* 1976). However, they also act systemically, so great care must be taken to avoid systemic hypotension. Epoprostenol, a prostacyclin-based drug, has also been used but there is limited evidence on its effectiveness (Northern Neonatal Network 1998).

If an infant with MAS and PPHN does not respond to conventional ventilator management, then alternative strategies, including high frequency ventilation and nitric oxide, can be considered. High frequency ventilation can provide more effective gas exchange at lower mean airway pressures and nitric oxide is a potent vasodilator which has a local, rather than a systemic, effect. Both of these approaches are discussed in more detail later in the chapter.

If the infant continues to deteriorate then extracorporeal membrane oxygenation (ECMO) should be considered. Results from the UK ECMO trial (UK Collaborative Trial Group 1996) show that ECMO is an effective form of treatment for MAS and PPHN. The mechanism of ECMO, along with the selection criteria, are discussed later in the chapter.

There is a very high mortality rate from MAS and PPHN. The prognosis for infants with MAS depends on the extent of hypoxia experienced by the infant and the development of neurological sequelae and persistent pulmonary hypertension. Infants with severe MAS may die or sustain significant neurological damage. ECMO has been shown to reduce the mortality and morbidity rate for severe MAS and this, together with the use of high frequency ventilation and nitric oxide, may further improve the outcomes of infants with MAS (UK Collaborative Trial Group 1996). There is growing evidence that MAS is associated with impaired lung function in infancy and an increased incidence of childhood asthma (Macfarlane and Heaf 1988).

Pneumothorax

Pneumothorax can occur spontaneously in a well term infant or may be associated with resuscitation, meconium aspiration syndrome, respiratory distress syndrome and positive-pressure ventilation due to the high inspiratory

pressures and asynchrony (Greenspan *et al*. 1998). The reduction in the number of pneumothoraces occurring in NICU has been attributed to the increased use of surfactant which means that ventilation rates and pressures can be reduced (OSIRIS Collaborative Group 1992). In a pneumothorax, air escapes from ruptured alveoli and collects between the parietal and visceral pleura. This causes the lung to become compressed and can cause severe respiratory embarrassment.

A pneumothorax should be suspected in any infant who is being resuscitated or is mechanically ventilated and who deteriorates suddenly showing signs of bradycardia and cyanosis. If the endotracheal tube is patent or has not been dislodged or disconnected from the ventilator tubing, then a pneumothorax should be suspected.

On auscultation, there may be decreased breath sounds on the affected side. Transillumination of the chest with a cold light source will show a translucent glow if a pneumothorax is present. This is a less reliable method in term infants. A chest X-ray will provide confirmation of the diagnosis. If the infant is in a state of collapse, emergency drainage of the pneumothorax will need to be undertaken based on the clinical features alone. A chest X-ray should be carried out as soon as possible after the procedure to confirm the diagnosis and the position of the drainage tube.

The aim of treatment is to remove the air to allow the lung to re-expand. This may be achieved by needle aspiration or the placement of a chest drain attached to an underwater seal drain (see p. 291 for technique). Term infants who are not seriously compromised by the pneumothorax can achieve lung re-expansion with high concentrations of supplemental oxygen (Greenough 1996).

If the infant is being mechanically ventilated and continues to fight against the ventilator, sedation or paralysis with pancuronium may be necessary. Hypotension is associated with pneumothoraces so inotropes may be required to maintain an adequate systemic blood pressure (see p. 139).

The outcome depends on the underlying lung pathology and the effect of the associated hypotension, but pneumothorax is now a rare occurrence in neonatal care and this has undoubtedly reduced neonatal mortality and morbidity.

Pulmonary haemorrhage

Pulmonary haemorrhage is caused by bleeding into the alveoli. It is usually found in infants who have other underlying lung pathology and are receiving positive pressure ventilation or it may be due to trauma as a result of suctioning. It has been reported as a side-effect of some exogenous surfactant replacement therapy (Ross Laboratories 1991). It is usually detected by the appearance of bright red blood in the suction catheter and may be accompanied by a sudden deterioration in the baby's condition. If the baby is not already intubated and ventilated then this would be instituted. A high positive end-expiratory pressure is used to achieve haemostasis by tamponading the bleeding. The rapid loss of

blood can lead to hypotension and clotting disorders. It is essential that a clotting screen is carried out as quickly as possible and any clotting disorders treated promptly.

The prognosis depends on the amount of blood lost. If there is a large bleed, then the most likely outcome is death. If the blood loss is small or controlled, then the eventual outcome will be determined by the underlying pathology.

Acid base balance

Disturbances of acid base balance can be more hazardous to the infant than the primary disease. All body functions are controlled by enzymes which are affected by small environmental changes. Acidity and alkalinity are especially important as they can disrupt the internal environment to such an extent that essential body functions are impaired. This can lead to disability and death if it is not corrected. Normal metabolism results in the production of acids which must be neutralised before being excreted from the body. There are three main methods of excretion—the lungs, the kidneys and haemoglobin.

Bicarbonate can combine with CO_2 to form carbonic acid. This is a weak acid which can be excreted via the lungs. It is converted to carbon dioxide and water and breathed out. The rate of removal of carbonic acid can be adjusted by altering minute ventilation. If the blood pH falls, the respiratory centre in the medulla of the brain detects the change and triggers an increase in the rate of breathing to 'blow off the carbon dioxide. The kidneys can excrete hydrogen ions and also conserve bicarbonate. Haemoglobin contains a protein buffer which neutralises acid—especially carbonic acid, which it converts to bicarbonate before releasing it as sodium bicarbonate into the plasma.

When considering blood gases, five values are usually measured:

- pH—the normal pH of arterial blood is 7.35–7.44.
- Partial pressure of oxygen (PO_2)—the normal PO_2 of arterial blood is 7–12 kPa.
- Partial pressure of carbon dioxide (PCO_2)—the normal PCO_2 of arterial blood is 4–6 kPa.
- Standard bicarbonate ($StHCO_3$)—the normal limits in arterial blood are 18–25 mml/l.
- Base excess (BE)—the normal base excess is +4 to -4.

Bear in mind that these are approximate values and the overall condition of the infant may mean that different values are accepted as 'normal' for a particular baby (Durand and Phillip 1996). For example, it is quite common to find infants with chronic lung disease with very high $PaCO_2$ values.

A metabolic acidosis occurs when there is an accumulation of acids or a loss of bicarbonate. Respiratory causes of metabolic acidosis include hypoxia. In this case, anaerobic metabolism will ensue. Lactic acid is a product of anaerobic

metabolism and this can cause a metabolic acidosis. This is common in infants with respiratory distress syndrome and those who are shocked and hypotensive.

The signs of a metabolic acidosis include: a fall in pH due to a rise in the number of hydrogen ions; a fall in StHCO$_3$ as bicarbonate is used up; a negative base excess as buffering agents are being used up; a normal *P*aCO$_2$. If the reason for the metabolic acidosis is hypoxia, then the *P*aO$_2$ will be low. An example of an arterial blood gas showing a metabolic acidosis would be: pH 7.24; *P*aCO$_2$ 4.5; StHCO$_3$ 16; *P*aO$_2$ 3; BE -8.

The treatment includes addressing the underlying cause. It may include increasing the *F*iO$_2$ to prevent anaerobic metabolism. Sodium bicarbonate should be given with extreme caution as it can lead to a metabolic alkalosis and it has been associated with intraventricular haemorrhage.

A metabolic alkalosis is due to increase in extracellular bicarbonate. The most common cause is inappropriate treatment with sodium bicarbonate. The signs are a rise in pH due to the loss in hydrogen ions; a rise in the StHCO$_3$; a normal *P*aCO$_2$ and *P*aO$_2$. An example of an arterial blood gas showing a metabolic alkalosis would be: pH 7.5; *P*aCO$_2$ 4.5; StHCO$_3$ 28; *P*aO$_2$ 8.4; BE +8. The treatment involves correcting the underlying cause. Prevention is better than cure.

A respiratory acidosis is due to an accumulation of carbon dioxide and is frequently seen in respiratory distress syndrome and meconium aspiration syndrome. It can also happen as a result of perinatal asphyxia. The signs are a fall in pH due to the accumulation of hydrogen ions and a rise in the *P*aCO$_2$ resulting in the formation of carbonic acid. The *P*aO$_2$, BE and StHCO$_3$ may be normal depending on the extent of the illness. An example of an arterial blood gas showing a respiratory acidosis would be: pH 7.14; *P*aCO$_2$ 8.4.5; StHCO$_3$ 20; *P*aO$_2$ 7.4; BE -4. The aim of treatment is to provide respiratory support to facilitate the excretion of carbon dioxide.

Respiratory alkalosis usually arises due to incorrect management of mechanical ventilation where excessive amounts of carbon dioxide are eliminated through the lungs. It can be dangerous, as a very low *P*aCO$_2$ can lead to a fall in cardiac and cerebral blood flow. The signs of a respiratory alkalosis are a high pH due to loss of hydrogen ions and a low *P*aCO$_2$ because carbon dioxide is being excreted via the lungs. An example of an arterial blood gas showing a respiratory alkalosis would be: pH 7.5; *P*aCO$_2$ 2.6; StHCO$_3$ 18; *P*aO$_2$ 7.4; BE -4. The treatment is to reduce the ventilation to allow the *P*aCO$_2$ to return to normal.

A mixed acidosis is usually the result of poor gas exchange which results in anaerobic metabolism. The signs are: a low pH due to the accumulation of hydrogen ions (particularly from the formation of lactic acid); a low *P*aO$_2$ because of poor lung function; a high *P*aCO$_2$ because of poor lung function; a low StHCO$_3$ as bicarbonate is used to buffer the carbonic acid and lactic acid produced; and a low BE as buffers are being used up. An example of an arterial blood gas showing a mixed acidosis would be: pH 7.24; *P*aCO$_2$ 6.8; StHCO$_3$ 16; *P*aO$_2$ 3; BE -8.

The condition is improved by improving gas exchange. This will increase the amount of oxygen available, thus reducing anaerobic metabolism, and also allow

carbon dioxide to be excreted via the lungs. A buffer may need to be given to counteract the effect of lactic acid production.

Measuring blood gases gives an indication of the adequacy of alveolar ventilation. However, tissue perfusion cannot be measured by blood gas analysis as it is dependent on other factors such as blood pressure and this should be remembered when assessing the condition of the infant (Durand and Phillip 1996). However, sometimes a blood gas value may appear to conflict with the clinical condition of the infant.

Errors in blood gas measurement can occur if the blood is above or below 37 °C. Dilution with heparin will lower the levels of carbon dioxide and increase the base deficit without affecting the pH. Air bubbles will cause a fall in the carbon dioxide levels and a rise in the oxygen levels. Delay in analysing the sample can cause the blood to continue to consume oxygen. This may cause an artificially low *P*aO$_2$ to be recorded by the blood gas analyser.

Decisions about the clinical management of the infant should not be based on blood gas results alone. Instead they should form part of the clinical profile of the infant to assist in assessing if and how the respiratory management should be altered.

Apart from intermittently measuring blood gases, oxygenation can be monitored continuously. Different methods can be used depending on the condition of the baby and the availability of equipment.

Indwelling transducers can be inserted into an umbilical artery. The catheter tip has a transducer which measures the partial pressure of oxygen in the vessel in which it is located. In the case of an umbilical arterial catheter, this will be aortic *P*aO$_2$. The transducer is connected to a monitor and gives a continuous readout. It is often possible to take samples of blood from the catheter and this can be used to calibrate the monitor. Calibration is necessary because the monitor is bombarded with red blood cells, the transducer becomes coated with a thin layer of protein and this can cause the monitor to display an artificially low reading.

In the case of an umbilical arterial catheter, it is important to avoid positioning the catheter opposite the renal, mesenteric and coeliac arteries as it can reduce the blood flow to organs supplied by these vessels. The position of the catheter should be checked on X-ray and a careful record should be kept of urine output. The abdomen should be observed for signs of distension that might indicate the development of necrotising enterocolitis.

Arterial catheters can cause ischaemia leading to necrosis so the limbs and fingers or toes should be checked to ensure that they are warm and well perfused. Vasospasm can occur after blood sampling. This is seen when a limb or fingers or toes become white. It should pass within two minutes of sampling. If it persists, then senior medical staff should be informed and consideration should be given to removing the cannula. If the catheter becomes blocked, then it must be removed. Flushing the catheter to dislodge the clot may lead to **emboli** formation.

Transcutaneous oxygen (TC*P*O$_2$) monitoring uses a sensor and a monitor. The sensor consists of a cathode and an anode. A small drop of electrolyte

solution is placed on the sensor and then covered with a membrane which is permeable to oxygen. Oxygen diffuses through the membrane and reacts with the electrolyte solution. The electrical current used to reduce the oxygen is proportional to the amount of oxygen at the membrane surface so the TCPO_2 monitor converts the electrical current into a PO_2 measurement which is then displayed (Rithalia 1989).

The main factor affecting the TCPO_2 reading is the temperature of the skin. The TCPO_2 reading is lowered by skin consumption of oxygen but this can be countered by the application of heat. Applying a heated sensor to the area causes the local blood flow to increase. The area becomes 'arteriolised'. Another important factor to bear in mind is that skin oxygen consumption varies with gestational age, so the more preterm an infant is, the lower the level of skin oxygen consumption.

The recommended sensor temperature is between 43 and 44 °C (Rithalia 1989). Preterm infants will tolerate temperatures of 43 °C while term infants require a temperature of 44 °C. The TCPO_2 monitor should be calibrated at the temperature at which it is to be used on the baby. If the temperature is changed without recalibrating the monitor, readings may vary by as much as 19 per cent. The sensor should be resited every 3–4 hours. Partial-thickness burns may occur if the sensor is left on too long or at too high a temperature.

Normally TCPO_2 readings correlate well with arterial oxygen measurements. However, they may underestimate arterial oxygen measurements if the baby is shocked or severely acidotic. Hypothermia, severe cyanotic heart disease and the use of drugs such as tolazoline may also adversely affect the accuracy of the readings.

The position of the monitor may also affect the reading. The best sites are where there is a good blood flow and the skin surface is level. Bony surfaces should be avoided as the blood flow is not particularly good in these areas. In preterm infants, intercostal recession may cause air to leak under the membrane and give a false reading. Normally there are no site differences in readings except in the case of right-to-left shunting of blood through a patent ductus arteriosus. A sensor placed on the right upper chest will show a higher preductal reading. Sensors placed on the abdomen will show a lower post-ductal reading.

When the TCPO_2 monitor is first applied, the reading falls rapidly until vasodilatation begins, then it rises gradually. The true reading may not be reached until 20 minutes after application. Calibration of TCPO_2 monitors should be undertaken regularly, according to the manufacturer's instructions. It is important to avoid calibrating the sensor near sources of supplemental oxygen as this can affect the accuracy of the monitor. *In vivo* calibration can be undertaken to correlate with readings of arterial blood gases.

Transcutaneous monitoring of carbon dioxide (TCPCO_2) is based on the same principle as the TCPO_2 monitor. In this case the gas-permeable membrane allows carbon dioxide to diffuse though. Carbon dioxide diffuses through the skin and through the sensor membrane. It reacts with the water in the electrolyte solution to form hydrogen ions and bicarbonate ions. This alters the electrical

potential of the solution and this is detected by the sensor and converted into a TC*P*CO_2 reading.

Where the skin is not heated the TC*P*CO_2 reading equals the venous carbon dioxide level. When the skin is heated there is an increase in the amount of carbon dioxide diffusing through the skin. In addition to this, the cells of the capillaries and skin give off carbon dioxide as part of normal metabolism. At a temperature of 44 °C, the TC*P*CO_2 reading is about 35 per cent higher than the arterial carbon dioxide level (Rithalia 1989).

When the sensor is applied, the TC*P*CO_2 reading rises rapidly and then falls as the skin carbon dioxide stores are washed out. True readings are obtained after 20 minutes with a temperature of 44 °C but this may take longer with a lower temperature setting.

Pulse oximetry is based on the way that oxyhaemoglobin and deoxyhaemoglobin absorb light at the red end of the spectrum. Oxyhaemoglobin absorbs more infrared than red light and deoxyhaemoglobin absorbs more red than infrared light. The oximeter probe consists of a light emitter and a light sensor aligned on opposite sides of a narrow part of the body such as the hand or the foot. The emitter sends out equal intensities of red and infrared light into the tissues and the sensor detects the ratio of red to infrared light that emerges. From this the proportion of oxyhaemoglobin to deoxyhaemoglobin can be measured and the percentage saturation of haemoglobin with oxygen can be calculated. In order to detect arterial rather than capillary or venous blood, the machine is programmed to look only at pulsatile increases (Stoddart *et al*. 1997).

This method of oxygen monitoring is light sensitive and light from external sources such as sunlight and phototherapy lamps can interfere with readings. Movement may also cause artefacts which can cause the machine to alarm. Pulse oximeters do not require a warm-up period and the response time is fast, unlike TC*P*O_2 monitors. Little skin damage occurs but the probe site should be checked regularly to ensure that pressure sores do not occur.

Pulse oximetry and transcutaneous oxygen monitoring are designed to estimate arterial oxygen levels in different ways. In arterial blood, 98 per cent of the blood is bound to the haemoglobin and 2 per cent is dissolved in the plasma. Indwelling transducers and transcutaneous monitors measure the 2 per cent dissolved in the plasma and give an indirect measurement of the amount of oxygen taken up by the haemoglobin. The main advantage of pulse oximetry is that it measures arterial oxygen saturation directly.

Several attempts have been made to show the relationship between oxygen saturation and partial pressure of oxygen. Wasunna and Whitelaw (1987) demonstrated that the saturation curve follows roughly the same shape as the oxygen dissociation curve. At the lower end of the curve, a wide range of saturation is equal to a narrow range of partial pressure. At the upper end of the curve, the opposite is true—a wide range of partial pressures corresponds to a narrow range of saturation. In practice this means that a baby with a saturation of 97% could have a *P*aO_2 of 18 kPa, while a baby with a *P*aO_2 of 6 kPA may have a saturation as low as 70%. In general, if the baby is at risk of hyperoxia

then a $TcPO_2$ monitor should be used. If there is little risk of hyperoxia causing retinal damage, then a saturation monitor can be used, with the alarms set between 90 and 95%.

Respiratory support

Continuous positive airway pressure (CPAP)

CPAP is a method of delivering a predetermined continuous pressure and supplemental oxygen to the airways of a spontaneously breathing infant (Jones and Deveau 1997). It improves oxygenation by recruiting collapsed alveoli, thus increasing the surface area available for gas exchange. The CPAP acts as a splint, stabilising the chest wall and inhibiting paradoxical movements during inspiration and collapse on expiration. If there is a low functional residual capacity, CPAP will improve ventilation and perfusion. It is also thought to conserve surfactant by preventing alveolar collapse (Jones and Deveau 1997).

CPAP can be delivered in a number of different ways. It can be used with an endotracheal tube in situ. This has all the disadvantages of intubation but it has the advantage of being able to switch to mechanical ventilation quickly, if required. The presence of the endotracheal tube causes increased airway resistance, thus increasing the work of breathing. This may affect the effectiveness of the CPAP and this makes it unsuitable for weaning from a ventilator (Davis and Henderson-Smart 1999).

A nasopharyngeal tube can be used to deliver CPAP. This is a long tube which is inserted into the nose and rests in the pharynx. It avoids the risks associated with endotracheal intubation but it can cause increased resistance, again increasing the work of breathing. Facial CPAP is still used in some units. It works by applying a face mask firmly to the baby's face. It is non-invasive but it can be difficult to maintain a seal. The pressure used to achieve an effective seal can lead to facial oedema and there is a large amount of dead space in the face mask to ventilate. Gastric distension is also a problem if high flow rates are used. In addition, many parents find the method distressing as the baby's face is almost completely obscured.

Nasal prongs are short, wide prongs which are inserted into the nares. They produce less resistance than the nasopharyngeal prong and thus reduce the work of breathing. However, mouth leaks can alter the level of CPAP. Nasal prongs have been associated with erosion of the nasal septum and facial disfigurement. These complications are related to poor fixation of the prongs.

Infants receiving CPAP should have the gases humidified to prevent oedema and thick secretions blocking the airway. Generally gases are warmed to about body temperature (36.5–37.2 °C). The airway should be suctioned as often as required following methodical assessment. This includes assessing blood gases, lung sounds and chest X-rays.

In some units, CPAP is used as the first choice for respiratory support in very preterm infants. Jacobsen *et al.* (1993) stated that prophylactic nasal CPAP was

associated with a decreased need for positive-pressure ventilation. However, a systematic review by Subramanian and Henderson-Smart *et al.* (1999) suggested that nasal CPAP in very small infants did not decrease the incidence of chronic lung disease and was associated with an increase in the number of intraventricular haemorrhages and increased mortality rates. However, most studies into the use of prophylactic nasal CPAP are small and not randomised. A large multicentre trial is currently being carried out in the UK to assess the effectiveness of nasal CPAP as an alternative to long-term intubation and ventilation in babies of 27–29 weeks' gestation.

Positive-pressure ventilation

Care of the intubated baby

Endotracheal tubes (ETT) can be passed orally or nasally (see p. 289). Although the tube position will be checked after intubation, the tube may become dislodged after placement. It is important to fix the ETT carefully to avoid slipping of the tube or accidental extubation. The position of the ETT should be checked each time a chest X-ray is taken. The nurse should auscultate the chest to ensure that bilateral breath sounds can be heard. Careful observation of the chest wall should reveal symmetrical chest wall movement (Turner 1997).

All gases given to the infant should be filtered, warmed and humidified. The aim is to deliver gases at near body temperature and 100% saturated with water vapour. Water can gather in the tubing. This should be removed regularly as it can interfere with gas pressures and the noise can be very irritating for the baby.

When the ETT is in situ, normal ciliary action is suppressed, tracheobronchial secretions are increased and the infant is unable to cough. If the tracheobronchial secretions are not removed, they can cause atelectasis and decrease functional residual capacity. There is great debate over how often an infant should be suctioned and very little research on which to base practice. The decision on how often to suction an infant will depend on the nurse's knowledge of the baby, together with the clinical findings.

Some infants respond to suctioning by dropping their oxygen levels and becoming bradycardic. Suctioning can also cause trauma to the trachea, so it should be done with great care. The suction tube should be soft and pliable. A large-bore tube will help remove secretions but it should not occlude more than two-thirds of the internal diameter of the ETT. If the ETT is totally obstructed the infant is likely to become hypoxaemic and bradycardic.

Physiotherapy is used in some neonatal units prior to suctioning. There is considerable debate over its usefulness. It has been shown to be helpful in removing tracheal secretions and preventing atelectasis but it is also associated with decreasing oxygenation, bradycardias and an increased incidence of intraventricular haemorrhages (Turner 1997). Most babies who are intubated are very unstable and minimal handling is usually advocated. Chest physiotherapy

should be used with caution in the neonatal unit and should only be carried out by an appropriately trained member of staff.

The use of normal saline lavage as an adjunct to suctioning is controversial. Ackerman (1985) demonstrated that saline inserted into the ETT remains in the main bronchus and is therefore of limited value. Hodge (1991) suggests that adequate humidification reduces the need for additional saline.

Mechanical ventilation is a method of ensuring that the infant is adequately oxygenated and that carbon dioxide is removed. Effective mechanical ventilation can be achieved by managing the different components of mechanical ventilation. This enables the carer to alter the mean airway pressure (MAP).

The MAP is an approximate measure of the average airway pressure throughout a respiratory cycle. It is written as

$$Paw\ (\text{MAP}) = k(\text{PIP-PEEP})\ [\text{IT}/(\text{IT}+\text{ET})]+\text{PEEP}$$

where k is a constant that depends on the rate of respiratory pressure increase and ET is the expiratory time. The MAP is sometimes given as a numerical value on ventilators or it may be seen as a waveform pattern. From the equation it can be seen that manipulation of any one of the variables can alter the MAP.

There is very little research on the effect of flow rates on the mean airway pressure and ventilation outcome. Usually flow rates of 6–10 l/min are used.

Ventilation rates have been shown to influence respiratory efforts. However, there is conflicting evidence as to what constitutes the optimal ventilator rate. Slow rates (<40 breaths per minute) are associated with increasing arterial oxygenation in some studies (Field *et al.* 1984). To maintain ventilation at slower rates the peak inspiratory pressure must be increased and this may lead to an increased incidence of barotrauma. If breathing is asynchronous, the infant may need to be sedated (see p. 203) or paralysed. Slow rates can also be used to wean infants from the ventilator and when reverse inspiratory: expiratory (I:E) ratio ventilation is in use.

Rapid rates (>60 breaths per minute) have also been shown to increase arterial oxygenation in very small infants (OCTAVE Study Group 1991). The rapid rate allows for a lower PIP, thus decreasing the incidence of barotrauma. However, caution must be exercised at very high rates as inadvertent PEEP may result at rates of more than 80 breaths per minutes. Rapid rates can lead to over-ventilation and respiratory alkalosis and in some infants they may result in an inadequate tidal volume or minute volume. In the past, high ventilator rates have been used to induce hypocarbia in infants with cerebral insults. It was believed that this reduced cerebral oedema. However, the resulting reduction in cerebral blood flow leads to increased periventricular leucomalacia and so this practice is no longer recommended.

The inspiratory: expiratory ratio is the relationship between the time taken to achieve lung inflation and the time taken to achieve expiration. Spontaneous respiration has an I:E ratio of between 1:1.2 and 1:1.5 and this is the ratio commonly used initially in small infants with RDS. Increasing the inspiratory time may increase the mean airway pressure and improve oxygenation but it

may also cause active expiration, thus increasing the risk of pneumothorax. After the first week of life a rate of 60 bpm may be effective with an I:E of 1:1 (Greenough *et al.* 1989).

Reverse I:E ratios can be used in infants with very severe RDS who are not responding to conventional ventilatory methods (Manginello *et al.* 1978). This works by providing a longer inspiratory time which allows a longer time for lung inflation and a short expiratory period. In particular, it seems to be effective in babies with very stiff lungs. This means using an I:E ratio of 1.5:1 to 2:1. Low rates (30–40 bpm) should be used to avoid gas trapping which is caused by failure to complete the respiratory cycle. This can be detected by rising *P*aCO_2 values. Reverse I: E ratios can impede venous return and are contraindicated in diseases associated with increased pulmonary vascular resistance. They are also associated with an increased risk of developing pneumothoraces.

A prolonged expiratory time (1:2, 1:3) can be used for weaning where there is increased pulmonary resistance, provided oxygenation is not a problem. It is also used in obstructive lung disease, e.g. MAS or where there is poor elastic recoil. Caution must be exercised, as the short inspiratory time can cause lowered tidal volumes and lead to inadequate ventilation.

The peak inspiratory pressure is the peak pressure used to achieve lung expansion. There is continuing debate over the most effective PIP in infants (Foote *et al.* 1990). Increasing the PIP will increase the MAP and generally improves oxygenation. However, high levels of PIP are associated with barotrauma. Initially a high PIP may be used to inflate the infant's lungs and then dropped as compliance improves.

A low PIP (<20 cmH_2O) is associated with a lower incidence of bronchopulmonary dysplasia (BPD) and pulmonary air leaks. However, it may lead to insufficient ventilation and the *P*aO_2 may fall if PIP is too low. A very low PIP can also cause generalised atelectasis. A high PIP (>20 cmH_2O) can be used to re-expand atelectasis and increase the arterial *P*aO_2 while decreasing the arterial *P*aCO_2. Unfortunately, a high PIP is associated with an increased incidence of pulmonary air leaks and BPD and it can impede venous return.

The positive end-expiratory pressure (PEEP) holds open the airways in between inflations caused by PIP. PEEP also appears to conserve surfactant by reducing the shearing forces when ventilation starts in partially collapsed alveoli (Ahumada and Goldsmith 1996).

Low PEEP (<3 cmH_2O) has been found to be useful to maintain lung volume during weaning. However, it may be too low to maintain adequate lung volume and it can result in CO_2 retention. Medium PEEP (4–6 cmH_2O) can stabilise areas of atelectasis and increase lung volume in surfactant deficiency. Care must be exercised because this level of PEEP can overdistend the lungs if compliance is normal.

High PEEP (>6 cmH_2O) can prevent alveolar collapse in surfactant deficiency where there is reduced lung compliance and can improve the distribution of ventilation. However, a high PEEP increases the risk of pulmonary air leaks and

can cause over-distension as well as impeding pulmonary vascular resistance. It may also lead to CO_2 retention.

Intermittent positive-pressure ventilation (IPPV)

The most common method of delivering IPPV to a neonate is via a pressure-limited time-controlled device, e.g. Sechrist and Draeger ventilators. A constant flow of gas is delivered to the neonate. During expiration, this flow delivers the PEEP. During inspiration, the gas distends the lung to a predetermined peak inflating pressure (PIP) for a preset inspiratory time. The amount of gas entering the lungs is determined by the peak pressure set on the ventilator blow off valve and the gas flow rate.

Volume-set time-limited ventilation is used in neonates but is less common than pressure-limited, time-controlled ventilation. Volume ventilators deliver the same tidal volume with each breath regardless of the pressure needed to deliver it. This means that areas of the lung that are collapsed or obstructed can be ventilated. However, there is a risk that volume ventilators may overdistend healthy areas of the lung and this may cause pulmonary air leaks (Spitzer and Fox 1996).

Pressure-supported ventilation is being introduced into some neonatal units. It is a form of ventilation that allows the ventilator or the baby to regulate the number of breaths but the ventilator takes over the work of breathing by providing pressure support during inspiration (Hakanson 1996). This method of respiratory support is still being evaluated.

Synchronous intermittent mandatory ventilation (SIMV)

Conventional ventilators deliver a predetermined number of breaths and asynchrony may occur between spontaneous breaths and ventilator breaths. This can be detected by observing the infant's respiratory movements and monitoring the arterial oxygen levels. The chest should be auscultated to ensure that bilateral breath sounds can be heard. Chest wall movement should be symmetrical. Asynchronous breathing can cause problems such as pneumothorax. In an attempt to overcome the problems associated with asynchronous breathing, it is possible to ventilate the infant so that the ventilator breath is delivered to coincide with the infant's own breathing pattern.

The total number of ventilator breaths will be decided by the operator—any further breaths that the infant makes will be unsupported by the ventilator. For example, if the ventilator rate is set at 60 bpm and the infant has a respiratory rate of 70, then the ventilator will deliver 60 breaths in synchrony with the baby's own respirations but the baby will breathe a further 10 breaths unsupported by the ventilator. If the infant becomes apnoeic then the ventilator will deliver breaths at the predetermined ventilator rate. Synchronised ventilation appears to facilitate the use of lower inspiratory pressures in infants, thus reducing the risk of air leaks (Donn and Hicks 1996).

Patient-triggered ventilation (PTV)

Patient-triggered ventilation is a method of positive-pressure ventilation where the ventilator rate is controlled by the neonate. A critical pressure level is preset and every time the infant's breath exceeds this level, the machine will deliver a breath. This should ensure that the infant and ventilator are in synchrony. This means that if the infant does not breathe within a predetermined period, then the ventilator will deliver a breath. Every breath initiated by the baby will be ventilator-assisted, unlike SIMV where the rate of the ventilator is predetermined by the clinician.

It is essential to set up a background ventilator rate. This is usually similar to the baby's own respiratory rate. The ventilator will then deliver this rate if the baby does not initiate a spontaneous breath. The inspiratory time should be shorter than on conventional ventilation to allow time for the ventilator to sense that a breath is being taken by the infant. If a normal inspiratory time is used, the short delay between the machine detecting the breath and delivering a ventilator breath will coincide with the expiratory phase and cause gas trapping.

PTV is unsuitable for very immature infants or those who have frequent apnoeas or poor respiratory effort as the background ventilation rate will be insufficient to achieve adequate ventilation.

Weaning

Weaning the infant from dependence on positive-pressure ventilation and achieving extubation is a complex process. Some units reduce the PIP and PEEP gradually and then wean the infant into a headbox when the pressures are very low. Other approaches include turning down the rate gradually and weaning the infant on to endotracheal tube CPAP. This method has been shown to be ineffective, as the presence of the ETT in the trachea increases the work of breathing (Davis and Henderson-Smart 1999). Some researchers have suggested that trigger ventilation can be helpful in weaning the baby off positive-pressure ventilation (Greenough and Pool 1989). This requires further investigation. A further approach is in the use of nasal CPAP to support respiratory efforts when the baby no longer requires intubation. Again, the effectiveness of this method is not particularly well evaluated.

Dexamethasone is being used in many units to shorten the weaning period. It does appear to shorten the time taken to wean the infant from the ventilator and reduces the number of infants who go back on ventilation but does not reduce the amount of time on oxygen (Halliday and Ehrekranz 1999a, b, c). There is still a great deal of debate about the correct dose of dexamethasone and when it should be commenced. Dexamethasone is associated with a number hypertension and hypoglycaemia, infection and gastric irritation and perforation of serious side-effects—it has been shown to adversely influence bone density and growth and it has been associated with an increased incidence of (Halliday and Ehrekranz 1999a, b, c).

There is still debate about the actual process of extubation. Some practitioners suction the infant prior to extubation and then remove the tube while other practitioners apply suction during the actual extubation procedure. Opponents of suctioning during the extubation procedure believe that this method leads to collapse of the alveoli.

Extubation is a significant event for the baby and the parents. It is important to emphasise that it is common for babies to require reintubation and recommencement of positive-pressure ventilation. For this reason it is essential that there is always someone available who can reintubate the baby and restart ventilatory support, if necessary.

High frequency ventilation (HFV)

High frequency ventilation is one of the newer technologies being used to provide respiratory support in sick infants. Conventional ventilation involves the bulk flow of gases through the airways. Molecular diffusion occurs in the terminal airways and alveoli. The usual respiratory rate in conventional ventilation is less than 100 breaths per minute. High frequency ventilation provides breaths or respiratory cycles of 240–3000 per minute (Karp 1997). The tidal volumes used in HFV may be less than or equal to the amounts used in conventional ventilation. The high frequencies used facilitate the diffusion of gases and may improve ventilation and perfusion matching. Generally HFV is used in cases where there is poor gas exchange and the infant is not responding to conventional ventilation. This includes severe lung disease such as RDS and meconium aspiration syndrome, pulmonary air leaks, persistent pulmonary hypertension and as an intermediate step in lung conditions such as diaphragmatic hernia.

High frequency jet ventilation (HFJV)

High velocity bullets of gas are fired down the airways from a cannula placed in the endotracheal tube. The bullets travel at rates of between 200 and 600 per minute and carry humidified gas in their wake. HFJV is associated with a high incidence (44–85 per cent) of necrotising tracheobronchitis (NTB). This condition occurs if the jet stream is not exactly in line with the trachea. For this reason, HFJV is rarely used in neonatal units in the UK as the benefits are outweighed by the adverse effects (Ophoven *et al*. 1984).

High frequency flow interruption (HFFI)

A high pressure flow of gas is interrupted by a valve to deliver heated, humidified oxygen. This delivers frequencies of up to 1200/minute. Sometimes this is classified as a form of oscillation.

High frequency oscillation (HFO)

A high pressure flow of gas is oscillated by a piston or diaphragm to deliver heated, humidified oxygen. This delivers frequencies of up to 1200/minute. The waveform refers to the shape of the breath delivered. This is demonstrated graphically on many oscillators. In HFO, the waveform is sine-shaped. The expiratory phase in oscillation is active in some ventilators such as the Sensormedics. This means that gas is assisted out of the chest by a negative force, rather than relying on passive exhalation. This will influence the I: E ratio as more time is required for exhalation if active expiration is used. The MAP is used to influence oxygenation, while the amplitude of the wave controls the *P*aCO_2. The amplitude is the size of the pressure wave produced by the oscillator.[2]

There are important differences in the nursing care given to infants receiving HFO as the ventilator systems make positioning more difficult. It is important to ensure that the air flows in straight lines so the ventilator tubing needs to be held in a fixed position otherwise necrotising tracheobronchitis may result. Suctioning can be problematical as it is important not to lose mean airway pressure. To avoid this risk, the pressure can be increased during the procedure or immediately afterwards to recruit collapsed alveoli. Some systems have indwelling catheters which avoid the need for this.

It is impossible to count the respiratory rate in infants receiving HFV because of the high rates and small tidal volumes delivered. The rapid chest movement can also affect ECG readings. The breath sounds in babies receiving HFV are different to those of a baby receiving conventional ventilation. High-pitched breath sounds are associated with secretions and decreased sounds are associated with poor ventilation or a pneumothorax (Karp 1997).

Infants receiving HFO need repeated X-rays to check for over-expansion and under-expansion. Over-expansion is shown by the presence of more than nine posterior ribs. There may also be a flattened diaphragm. Under-expansion of the lungs is demonstrated by fewer than nine posterior ribs and poor aeration of the lungs. Over-expansion can cause a decrease in CO_2 which leads to a drop in blood pressure and oxygenation.

When setting up HFO as a rescue therapy, the mean airway pressure should be set above that which is currently being used. The amplitude (or delta P) can then be slowly increased until the chest wall vibrates. Weaning from HFO is still poorly understood. The usual approach is to lower the amplitude and reduce the mean airway pressure and then use conventional ventilation or nasal CPAP until the baby is sufficiently recovered to breathe unaided by a ventilator.

A major strength of HFO is the improvement it produces in the condition of infants with severe lung disease who are not responding to conventional ventilation. There is also a putative advantage in lowering the pressures of gas delivered into the infant's lungs—this is thought to reduce the incidence of chronic lung disease, although this has not been demonstrated in any of the clinical trials so far. Some studies have suggested that babies who received HFO were more likely to be at risk of serious adverse effects such as intraventricular haemorrhage (IVH) (Henderson-Smart *et al.* 1999). These studies were carried

out without the use of surfactant and when the technique was relatively new. Further studies are being carried out into the use of HFO both as a rescue treatment and as a first line treatment in babies with severe lung disease.

Negative extrathoracic pressure

Negative-pressure ventilation mimics normal physiological respiration and is reported to decrease barotrauma and chronic lung disease (Samuels *et al.* 1996). Sub-atmospheric pressure is applied to the outside of an infant's chest by nursing the baby in a specially designed chamber. The baby can be nursed in a headbox or with nasal prongs if additional oxygen is required. The negative pressure can be applied continuously (CNEP), which is similar to positive end-expiratory pressure, or intermittently (INEP), which is similar to positive-inspiratory pressure ventilation.

Currently negative-pressure ventilation is used to wean infants with chronic lung disease. However, Samuels *et al.* (1996) used negative-pressure ventilation as a first line treatment for preterm infants with respiratory distress syndrome. Although the treatment appeared successful, there were concerns about the mortality and morbidity associated with use of negative pressure in preterm infants and it appears unlikely that negative pressure will be a first line treatment for infants with RDS.

Extracorporeal membrane oxygenation (ECMO)

Some infants with severe pulmonary dysfunction do not respond to either conventional ventilation or high frequency ventilation. ECMO is a form of cardiopulmonary bypass which permits gas exchange outside the body and has been shown to be effective in babies with reversible lung disease (UK Collaborative Trial Group 1996).

ECMO is a highly invasive form of treatment and it is associated with a number of adverse effects. Because of the need for significant heparinisation, infants of less than 34 weeks' gestation are not suitable. ECMO cannot be carried on indefinitely so it is only suitable for infants with respiratory disorders that are potentially reversible. The usual method of determining whether a baby requires ECMO is by calculating the oxygenation index (OI). It can be calculated using the following equation:

$$\mathrm{OI} = \frac{\text{mean airway pressure} \times (F\mathrm{iO}_2 \times 100)}{P\mathrm{O}_2 \text{ (mmHg) post ductal}}$$

An oxygenation index value of more than 40 has been associated with an 80 per cent mortality rate (UK Collaborative Trial Group 1996). Therefore babies with a rapidly rising oxygenation index should be referred to a specialist centre as soon as possible before the baby becomes too sick to transport.

A large randomised controlled trial of ECMO carried out in the UK has shown that it is an effective method of treatment for infants with conditions such as meconium aspiration syndrome who do not respond to conventional ventilation (UK Collaborative Trial Group 1996). However, because it is such a complex treatment, it is offered only in specialist centres.

Nitric oxide (NO)

NO is produced in the lungs and influences pulmonary vasodilatation because of its molecular properties. It is thought that its release may contribute to the normal transition of fetal circulation to adult circulation. It is a potent vasodilator with a very short half-life (1–4 seconds) and exogenous NO has been used to treat pulmonary hypertension in children (Finer and Barrington 1999). More recently, it has been used in the treatment of persistent pulmonary hypertension in neonates—particularly that associated with meconium aspiration syndrome. It has also been used in very preterm infants with severe respiratory distress syndrome.

Some centres are now using NO as a treatment in persistent pulmonary hypertension because it avoids the systemic effects associated with drugs such as tolazoline, which causes systemic hypotension. Inhaled NO produces localised vasodilatation in the pulmonary circulation without the systemic effects. The main disadvantage to using NO is its ability to combine with oxygen to form nitrogen dioxide which is toxic to lung tissues as it causes pulmonary oedema. It is also a relatively new treatment and its use in neonates, as yet, has not been extensively evaluated.

Generally NO is introduced when the infant is not responding to conventional ventilation or high frequency ventilation. It may be used to support the infant while a decision is made about the appropriateness of ECMO therapy. NO therapy has been used in very preterm infants with severe RDS. However, there are concerns about the use of the gas on developing lungs, and in the absence of any substantive data to support its use it is not recommended for preterm infants (Barrington and Finer 1999).

Because NO is unstable, it readily reacts with oxygen to form nitrogen dioxide. This is a toxic substance which causes acute lung injury and fulminating pulmonary oedema. It is therefore important to try to minimise damage while maximising the therapeutic effects. This can be achieved using a gas blender to introduce NO into the ventilatory circuit and a scavenging system to remove gas from the system as it is expired.

The amount of NO required for a therapeutic effect is debatable. The needs of the individual infant need to be taken into account. The recommended doses vary between 0.1–18ppm and 2–40 ppm (Finer 1997). Further research is required to establish the appropriate dose range in neonates.

The infant receiving NO is critically ill and so will require intensive monitoring. Haemoglobin is oxidised by NO, converting it into methaemoglobin, this compound will not carry oxygen and cyanosis will result. Screening facilities for

methaemoglobinaemia should be available and the infant should be screened regularly for the condition. The aim of treatment is to keep the methaemoglobin level below 2%. Methylene Blue can be used to treat methaemoglobinaemia and should always be available in the unit when NO therapy is being used. Spitzer *et al.* (1996) suggest that pulse oximeters can interpret methaemoglobin as a falsely high saturation level so pulse oximetry is best avoided in babies receiving NO therapy if the methaemoglobin levels are greater than 2%.

NO administration can produce very rapid changes in the infant's condition so changes in the amount of NO being delivered should be made gradually. Any interruption in the gas flow, including suctioning, can cause deterioration in the infant's condition. Neonatal units which use NO should have facilities available to provide NO when hand ventilating. Similarly the NO should be weaned gradually before discontinuation. Some babies can become dependent on NO and may require prolonged weaning.

The levels of NO in the environment should be monitored carefully. They should not exceed safe levels if a scavenging system is used. However, local health and safety policies should be available to ensure that the well-being of staff is not compromised.

Liquid ventilation

Liquid ventilation is achieved by replacing the gas in the baby's lungs with a liquid (Spitzer *et al.* 1996). Perfluorcarbon (PFC) liquids are clear, colourless, odourless and inert. They have a low surface tension and are non-biotransformable. Carbon dioxide and oxygen are highly soluble in PFC fluids. The fluids are minimally absorbed and do not appear to cause long-term biochemical or toxic changes. Research is currently being undertaken to assess the usefulness of liquid ventilation in conditions such as respiratory distress syndrome, meconium aspiration syndrome and PPHN.

Conclusion

Respiratory disorders are one of the commonest problems likely to be encountered in neonatal units. There are a range of technologies currently available to the neonatal multidisciplinary team to assist in the management of infants who have respiratory problems. However, it is apparent that many technologies are inadequately evaluated for today's NICU population and require further trials in order to establish the most appropriate methods of application in neonatal care.

Case study: ventilatory management of a preterm infant

Amanda was born at 27 weeks' gestation, weighing 924 g, and is now 3 hours old. She was born by emergency cesarean section for maternal antepartum haemorrhage so no antenatal steroids were given. The baby made minimal respiratory efforts at delivery and was intubated by 1 minute of age and ventilated. The initial blood gases were: pH 7.14; $PaCO_2$ 6.8; PaO_2 3.0; $StHCO_3$ 16; BE -8.

The baby was ventilated on continuous mandatory ventilation using a rate of 60 bpm, PEEP of 3 cmH$_2$O and a PIP of 22 cmH$_2$O. The FiO_2 is 100% and the indwelling arterial transducer shows PaO_2 of 4 kPa. Her mean arterial blood pressure is 26 mmHg. The initial chest X-ray shows a correctly positioned ETT and features suggestive of RDS. She has received one dose of surfactant at one hour of age.

Q.1. What does the initial blood gas result show?

Q.2. How may Amanda be further managed in order to gain an improved physiological state?

Q.3. What are the potential complications for Amanda, and how may they be prevented?

Q.4. What is Amanda's potential outcome?

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Chapter 6

Cardiovascular Management

chapter 6

Contents

Introduction

Nursing an infant with congenital heart disease requires considerable clinical skill and knowledge of the particular defect. Parents will often ask the neonatal nurse to explain how the defect happened. Nurses familiar with embryonic development, fetal and neonatal blood flow, and normal cardiac function will be able to help parents better understand the nature of the defect. Recognising the signs and symptoms of heart disease and undertaking an accurate clinical assessment of the infant allows for early intervention and treatment. Nursing staff caring for these infants must be alert to any changes in respiratory effort, oxygenation, blood pressure and renal output. Monitoring these infants' respiratory, cardiac and renal status enables the neonatal nurse to recognise any deviations and initiate appropriate management. The neonatal nurse is a vital link in the recognition and management of infants with congenital heart disease.

This chapter presents an overview of the embryological development of the heart and indicates some of the major structural defects that can occur during this period. The fetal circulation, the changes that occur during adaptation to extrauterine life, normal cardiac function and the nursing management of infants with congenital heart disease are discussed.

Cardiac embryology

The embryonic period is considered to be from two weeks post fertilisation to the end of the eighth week (Larson 1993). Between the third and fourth weeks, the heart starts to beat and blood begins to circulate. The fourth to eighth weeks constitute a very important period of embryonic development because the beginnings of all major external and internal structures appear during these five weeks. By the end of the eighth week, all the main organs have begun to develop, but the function of most is limited, the exception being the heart which is fully formed and functioning (Moore and Persaud 1998). The majority of cardiac abnormalities are due to errors occurring between the third and eighth week of embryonic development. In order to understand how cardiac defects occur, it is necessary to understand how the heart develops. An overview of the development of the heart is presented here; for a more detailed description and explanation the reader should refer to Moore and Persaud (1998), *The Developing Human: Clinically Oriented Embryology.*

Development of the heart

During the third week of the embryonic period, paired heart tubes are formed from the splanchnopleuric mesoderm, a layer of the lateral mesoderm (Figure 6.1). By the end of the third week, these two tubes have fused together to form a primitive single heart tube. The blood supply to and from the embryo is via the vitelline, umbilical and cardinal veins. The

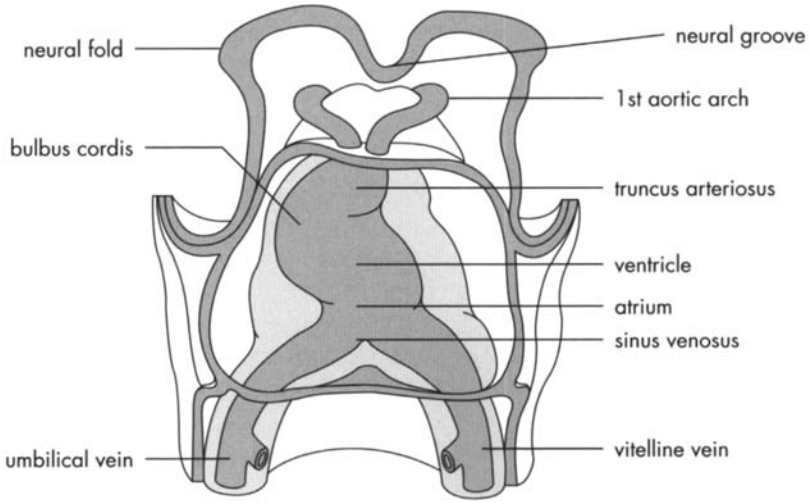

Figure 6.1 **The heart tubes fuse to form a single heart tube**
Source: after Moore, K. and Persaud, T.V.N. (1998) *The Developing Human—Clinically Oriented Embryology,* 6th edn, Philadelphia: W.B.Saunders, p. 356, with permission

developing heart wall has two layers—an inner layer, which will eventually become the endocardium, and an outer layer, which will later form the myocardium.

As the heart tube begins to grow, a series of dilatations and constrictions appear which mark five distinct areas. Starting from the caudal (tail) and moving to the cephalic (head), the areas are the sinus venosus, the primitive atrium, the primitive ventricle, the bulbus cordis and the truncus arteriosus. This elongating tube then begins to fold and bend back upon itself. The primitive atrium is carried behind and above the primitive ventricle. The primitive atrium will then expand to form the two atria, the primitive ventricle will give rise to most of the left ventricle and the bulbus cordis will form the right ventricle. The sinus venosus is eventually incorporated into the right side of the heart and will become the inferior and superior vena cava. The truncus arteriosus dilates to form the aortic sac from which the six aortic arches arise. From the aortic arches develop the carotid arteries, the arch of the aorta, the subclavian arteries, the pulmonary artery and the ductus arteriosus. Because of the many changes involved in the transformation of the embryonic aortic arches to the adult arterial system, anomalies in the aortic arch and pulmonary arteries can occur. One such malformation is coarctation of the aorta. Although there are several theories, the embryological reason why this occurs is unclear (Tikkanen and Heinonen 1993). Another malformation which may occur during folding is dextrocardia, when the heart tube bends to the left instead of to the right (Moore and Persaud 1998).

Formation of the four chambers of the heart

The endocardial cushions

The result of folding is to bring the four chambers of the future heart into their correct position. The right atrium and ventricle are separated from those on the left by the endocardial cushions. The atrioventricular canals develop from the endocardial cushions. The mitral valve and the tricuspid valve are formed by the thinning and hollowing of the tissue surrounding these orifices. Failure of the endocardial cushions to develop can lead to atrio-ventricular canal defects or mitral or tricuspid valve abnormalities (Witt 1997).

The atria

At the same time as the endocardial cushions are forming, separation of the atrium begins. The atrium is divided by two adjacent septa—the septum primum and the septum secundum (Figure 6.2). An opening between the two atria—the foramen ovale—is created. Throughout the rest of fetal development, blood that has entered the right atrium from the inferior vena cava passes from the right atrium to the left atrium via the foramen ovale. Atrial septal defects can occur due to patency of the foramen ovale or failure of the endocardial cushions to form properly. If the pulmonary veins fail to connect with the left atrium, total anomalous pulmonary venous return can occur. In this defect the pulmonary veins return blood into the right atrium or into a systemic vein, bypassing the left atrium (Moore and Persaud 1998).

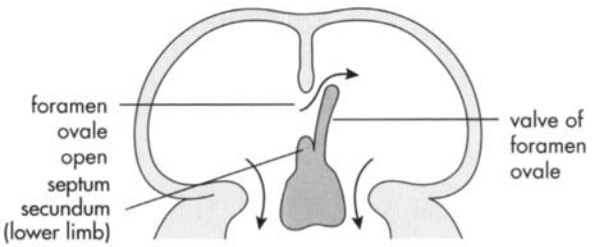

Figure 6.2 **When pressure in the right atrium exceeds that in the left atrium, blood passes from the right to the left side of the heart. When the pressures are equal or higher in the left atrium, the valve formed by the septum primum closes the foramen ovale**

Source: after Moore, K. and Persaud, T.V.N. (1998) *The Developing Human—Clinically Oriented Embryology,* 6th edn, Philadelphia: W.B.Saunders, p. 364, with permission

The ventricles

Separation of the ventricles is completed about a week after atrial separation. The primitive ventricle is divided into two chambers by a thick muscular septum

—the intraventricular septum. A small foramen exists between the intraventricular septum and the endocardial cushions, allowing the two ventricles to communicate. This foramen closes about the end of the seventh week with the formation of the septum separating the aorta and pulmonary arteries (Figure 6.3). Ventricular septal defects occur when it fails to close (Moore and Persaud 1998).

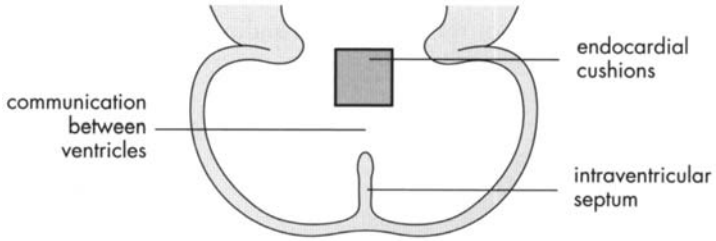

Figure 6.3 **Septation of the ventricles by a thick muscular ridge called the intraventricular septum. The small aperture between the endocardial cushion and the intraventricular septum allows the two ventricles to communicate**

Formation of the aortic and pulmonary trunks

Further septation of the ventricles and outflow tract must occur if the heart is to function properly. The truncus arteriosus and the bulbus cordis are divided longitudinally by a spiral septum called the aorticopulmonary septum (Figure 6.4*a*). This septum is formed by two intertwining ridges which spiral downwards. This spiral effect allows the vessel to twist upon itself as it divides to form the aorta and pulmonary artery (Figure 6.4*b*).

Persistent truncus arteriosus occurs when the aorticopulmonary septum fails to develop, leaving one single arterial trunk arising from the ventricles. Because this septum also forms part of the intraventricular septum a ventricular septal defect is always present in persistent truncus arteriosus (Moore and Persaud 1998). Transposition of the great arteries occurs when the aorticopulmonary septum fails to spiral. This results in the aorta arising from the right ventricle and the pulmonary artery from the left ventricle. The semilunar valves are formed from endocardial tissue surrounding the orifices of the aorta and pulmonary artery.

The fetal circulation

Knowledge of fetal blood flow is essential to understand the changes that occur during the transition from fetal to neonatal circulation.

The fetal circulation involves four unique anatomical structures: the placenta, which is the exchange organ for oxygen, carbon dioxide, nutrients and waste; the ductus venosus, which allows most of the blood from the

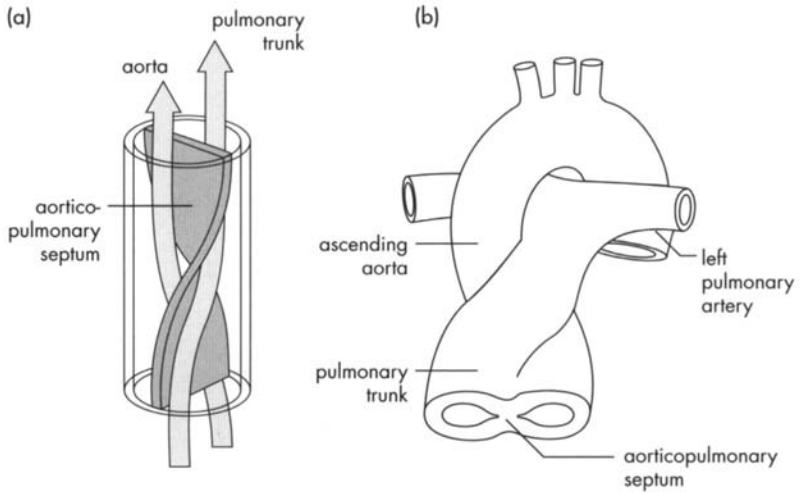

Figure 6.4 **(a) Diagram illustrating the spiral form of the aorticopulmonary septum, (b) The spiral effect allows the vessel to twist upon itself as it divides to form the aorta and pulmonary artery**

Source: after Moore, K. and Persaud, T.V.N. (1998) *The Developing Human—Clinically Oriented Embryology,* 6th edn, Philadelphia: W.B.Saunders, p. 373, with permission

placenta to bypass the liver and enter the inferior vena cava; the foramen ovale, which permits blood to flow directly from the right to the left atrium; and the ductus arteriosus, which connects the pulmonary artery with the descending aorta, through which blood flows in order to bypass the fetal lungs. The ductus arteriosus protects the lungs and allows the right ventricle to strengthen in preparation for functioning at birth (Carlson 1994). In utero, the fetal lungs do not provide gas exchange, therefore the pulmonary blood vessels are vasoconstricted. The fetal circulation is designed in such a way that the most highly oxygenated blood is delivered to the brain and heart whilst being diverted from the lungs.

After oxygenation in the placenta, blood flows via the umbilical vein to the fetus (Figure 6.5). At the inferior surface of the liver, the umbilical vein divides: one branch joins the portal vein and the other branch, the ductus venosus, bypasses the liver and enters the inferior vena cava. Here, oxygen-rich blood mixes with venous blood. The mixed blood then enters the right atrium via the inferior vena cava. Most of the blood passes through the foramen ovale and enters the left atrium. From the left atrium the blood flows through to the left ventricle and then to the head and neck via the ascending aorta. Blood returns from the head and neck via the superior vena cava to the right atrium, then to the right ventricle and the pulmonary artery. However, as the fetal lungs are not functioning, most of the blood in the pulmonary trunk, instead of flowing through the pulmonary artery to the lungs, passes through the ductus arteriosus into the aortic arch, where it mixes with blood coming from the left ventricle. Blood flows via the descending aorta to the trunk and lower

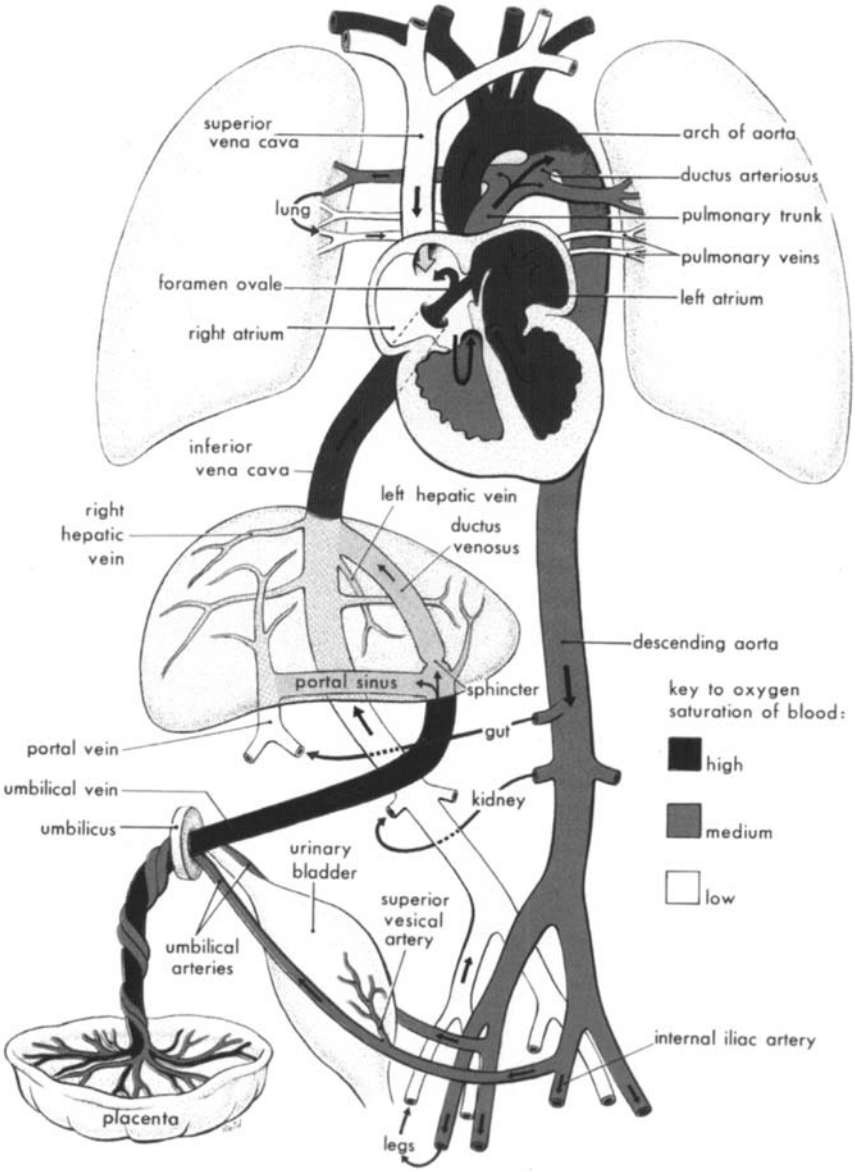

Figure 6.5 **Schematic illustration of the fetal circulation. The shading indicates the oxygen saturation of the blood, and the arrows show the course of the blood from the placenta to the heart. The organs are not drawn to scale. Observe that three shunts permit most of the blood to bypass the liver and lungs: the ductus venosus, the foramen ovale and the ductus arteriosus. The poorly oxygenated blood returns to the placenta for oxygen and nutrients through the umbilical arteries**

Source: after Moore, K. and Persaud, T.V.N. (1998) *The Developing Human—Clinically Oriented Embryology,* 6th edn, Philadelphia: W.B.Saunders, p. 392, with permission

limbs and is returned to the placenta via the two umbilical arteries for re-oxygenation and re-circulation.

Transition from fetal to neonatal circulation

The changes that occur to the cardiopulmonary system at birth are accompanied by a series of complex events which switch gas exchange from the placenta to the lungs. As mentioned previously, the fetal lungs are not required for gas exchange, the placenta supplies the fetus with oxygen and removes carbon dioxide. The alveoli of the fetal lungs are filled with fluid. At birth, with the onset of respiration, the lungs expand and replace the fetal lung fluid with air. The fetal lung fluid is absorbed into the blood and lymphatic systems. At the same time as the lungs are expanding and the fetal lung fluid clearing, the arterioles in the lungs begin to open allowing a considerable increase in the amount of blood perfusing the lungs. With the increase of blood flow to the lungs, pulmonary vascular resistance falls. The pressure in the left atrium is then greater than in the right atrium, with this pressure change closing the foramen ovale. The ductus arteriosus begins to contract as arterial oxygen levels rise (Bernstein 1996). Blood previously diverted through the ductus arteriosus now flows directly to the lungs via the pulmonary arteries.

Although oxygen is the main factor in the closure of the ductus arteriosus, other substances also regulate pulmonary blood flow at this time, such as bradykinin, a substance released from the lungs during inflation, and prostaglandin which, affects the smooth muscle in the wall of the ductus arteriosus (Cavaliere and Sansoucie 1997). Closure of the ductus arteriosus in healthy term infants occurs immediately after birth, with 20 per cent of the ducts closing functionally by 24 hours, 82 per cent by 48 hours and 100 per cent by 96 hours (Hammerman 1995). Anatomical obliteration usually occurs by constriction at about three to four weeks of life. The ductus arteriosus then becomes the ligamentum arteriosum.

When the umbilical cord is clamped and cut, there is a loss of placental circulation with a resultant increase in peripheral vascular resistance. Blood flow through the ductus venosus ceases. The ductus venosus is obliterated by about one to two weeks of life, and then becomes the ligamentum venosum. The umbilical arteries and vein are no longer needed to transport blood. The umbilical vein becomes the ligamentum teres (round ligament) and the umbilical arteries atrophy, becoming the lateral umbilical ligaments.

In certain circumstances, a return to a fetal type of circulation (without the placenta) is possible in an otherwise normal term infant. Persistent pulmonary hypertension of the newborn, also known as persistent fetal circulation, is the term applied to the combination of pulmonary hypertension, subsequent right-to-left shunt through the foramen ovale and ductus arteriosus and a structurally normal heart (Heywood *et al.* 1993). Asphyxia with hypoxia, hypercarbia and acidosis causes the pulmonary arterioles to vasoconstrict, resulting in

increased pulmonary vascular resistance, leading to pulmonary hypertension and reopening of the fetal channels. As a consequence, the infant becomes severely hypoxaemic. The main goal of treatment is to correct the hypoxia and acidosis and promote pulmonary vascular dilatation whilst supporting other systems (see p. 104).

Because of the nature of the fetal circulation, conditions such as transposition of the great vessels, hypoplastic left heart, coarctation of the aorta, and tetralogy of Fallot may not present until after the fetal channels close.

Cardiac function

The function of the heart is to maintain a constant circulation of blood throughout the body.

The cardiac cycle

This is the term used for the contraction and relaxation phases of the heart. The contraction phase is termed systole, the relaxation phase diastole. A neonate's cardiac cycle is approximately 0.4 seconds, with 0.2 seconds for diastole and 0.2 seconds for systole, based on a heart rate of about 150 beats per minute (Conover 1988). Contraction of the heart muscle is controlled by a bundle of neuromuscular cells. When the sinoatrial node stimulates the atria, the atria contract, forcing blood into the ventricles. Atrial relaxation allows the right atrium to fill with blood from the inferior and superior vena cava and the left atrium to fill from the pulmonary veins. Ventricular contraction occurs at the same time as atrial relaxation. The atrioventricular node, stimulated by atrial contraction, sends impulses to the ventricles to contract. Pressure in the ventricles increases rapidly, thus exceeding atrial pressure. This high pressure causes the mitral and tricuspid valves to close and the pulmonary and aortic valves to open, forcing blood into the pulmonary artery and aorta.

Blood pressure

Blood pressure is an important measurement of cardiovascular status. There are three main factors that affect arterial blood pressure: cardiac output, blood volume and peripheral resistance.

Cardiac output is the amount of blood pumped out of each ventricle per minute (Tortora 1991). It is determined by the amount of blood pumped from each ventricle per heart beat and the number of heart beats per minute. Stroke volume is the term used for the amount of blood pumped out of each ventricle per beat. Stroke volume is influenced by preload, afterload and myocardial contractility. Preload is the volume of blood in the ventricles at the end of relaxation (diastole). Afterload is the force against which the ventricle must contract in order to eject blood during systole (McCance and Richardson 1990).

Normally the more cardiac fibres are stretched by the filling of a chamber with blood, the stronger the walls will contract to eject it. This is referred to as Frank Starling's Law. However, the neonate's heart has fewer fibres to accommodate increased volume. The only effective mechanism by which the neonate can respond to increased volume is by increasing heart rate (Lott 1993). If the volume exceeds the heart's ability to pump, cardiac failure will result.

The average blood volume of the term infant is approximately 85 ml/kg and of the preterm infant 100 ml/kg (Brown 1988). Any decrease in circulating blood volume, for example from haemorrhage, will cause the blood pressure to fall. A further factor which influences blood pressure is resistance. Arterioles control peripheral resistance and therefore blood pressure and flow by changing their size. The greater the size (diameter) of a vessel, the lower the resistance; the smaller the diameter, the higher the resistance.

Regulation of blood pressure

Blood pressure is regulated by the autonomic nervous system. Baroreceptors located in the aorta and carotid arteries are sensitive to changes in blood pressure. Feedback from these receptors stimulates the sympathetic and parasympathetic nervous system. Sympathetic impulses to the adrenal medulla release adrenaline, which increases heart rate and myocardial contractility. It also affects venous tone by bringing about peripheral resistance. Under the influence of the parasympathetic nervous system acetylcholine is released. This affects the sinoatrial and atrioventricular nodes of the conduction system causing decreased heart rate and contractility.

When blood pressure falls, the kidneys secrete renin, which indirectly stimulates the secretion of aldosterone which in turn acts on the tubules of the kidneys causing the re-absorbtion of sodium and water, resulting in increased circulating blood volume. As blood volume increases, blood pressure increases. These mechanisms maintain blood pressure homeostasis.

The management of blood pressure

The neonatal nurse must be able to measure and assess blood pressure accurately as this allows for early identification of hypotension, enabling appropriate treatment to be initiated. Blood pressure in the neonate can be measured using either oscillometry or direct intra-arterial methods. Oscillation methods have proved to have a certain degree of accuracy when compared with direct intraarterial methods (Versmold 1991). However, in hypotensive very low birth weight infants, direct intra-arterial methods are more reliable (Cunningham and McIntosh 1992).

Blood pressure usually increases with gestational age. The normal range (systolic pressure) for infants at 28–32 weeks is 52 mmHg, at 33–36 weeks it is

56 mmHg and at term is 63 mmHg (Cantu *et al.* 1991). Preterm very low birth weight infants have a range of mean pressure from 22 to 42 mmHg (Gunderson 1990).

Hypotension in the neonate may be due to hypovolaemia, septicaemia or secondary to heart disease.

An infant with hypovolaemia presents with pallor, poor peripheral perfusion, weak pulses with a good heart rate, usually a tachycardia and associated hypotension. The nursing management of this infant includes early recognition of the clinical signs, the administration of prescribed volume expanders and inotropic agents, continuous monitoring of the infant's heart rate, blood pressure, toe/core temperature and urinary output for desired or adverse effects of the administered therapy, followed by further referral to medical staff if necessary. Volume expanders are used to counteract the effects of hypovolaemia by increasing vascular volume and subsequently tissue perfusion. Volume expanders are whole blood ('O' negative), albumin, plasma, sodium chloride 0.9% and Ringer's lactate, used in 10–20 ml/kg increments given over 5–10 minutes (Shaw 1993). Inotropic support such as dopamine and dobutamine can be used in conjunction with volume expansion infusions to improve cardiac contractility. Dopamine is a natural catecholamine precursor of noradrenaline and its effect is dose-dependent. Low doses stimulate renal dopaminergic receptors, resulting in increased renal blood flow, urine output and sodium excretion. Higher doses increase cardiac contractility and heart rate, thus raising blood pressure (Givens-Bell 1998). It is administered by continuous infusion at 5–10 µg/kg per minute, with the dose titrated to the clinical response. Dobutamine hydrochloride is a synthetic catecholamine whose overall effect is to increase cardiac output and stroke volume without increasing heart rate. The dose is 5–20 µg/kg per minute and is also given by continuous infusion. Hypovolaemia should be corrected before using dopamine and dobutamine as they are ineffective in the presence of inadequate blood volume (Givens-Bell 1998).

Congenital heart defects

Congenital heart defects are fairly common, occurring in about 0.5 per cent of live births and 2.7 per cent of stillbirths (Thomas 1992). The factors that influence the development of congenital heart defects are classified as chromosomal, genetic, maternal or environmental. For example, maternal viral infections such as rubella, cytomegalovirus or toxoplasmosis are known to cause congenital heart defects (Lott 1993). Trisomy 21 (Down's syndrome) is associated with endocardial cushion or ventricular septal defects.

The common congenital heart defects seen in the first week of life are atrial septal defects, ventricular septal defects, endocardial cushion defects, patent ductus arteriosus, transposition of the great arteries, tetralogy of Fallot, pulmonary atresia, hypoplastic left heart syndrome, coarctation of the aorta and pulmonary and aortic stenosis (Monett and Moynihan 1991).

Congenital heart malformations are usually described as either acyanotic or cyanotic. Acyanotic heart defects produce a left-to-right shunt and do not cause cyanosis because blood flows from the systemic to the pulmonary system resulting in increased blood flow to the lungs. In right-to-left shunts, blood flows from the pulmonary system to the systemic circulation, deoxygenated blood mixes with oxygenated blood producing cyanosis (Wood 1997).

Patent ductus arteriosus and coarctation of the aorta are examples of acyanotic heart defects; transposition of the great arteries and hypoplastic left heart syndrome are classified as cyanotic heart defects. These defects will now be discussed in detail.

Patent ductus arteriosus

Patent ductus arteriosus in preterm infants has long been recognised as a common heart lesion (Musewe and Olley 1992). As the infant with respiratory disease begins to recover, oxygenation improves and pulmonary vascular resistance falls. The ductus arteriosus in the preterm infant is not as responsive to increased oxygen and therefore remains open. This allows left-to-right shunting which causes an increase in pulmonary blood flow.

Nursing staff caring for the preterm infant need to be alert that an increased oxygen requirement and the need for ventilatory support may indicate the presence of a patent ductus arteriosus. However, worsening lung disease and sepsis need to be considered and ruled out. Other clinical signs and symptoms include: tachycardia, due to the heart increasing its stroke volume to maintain adequate systemic perfusion; tachypnoea, reflecting increased pulmonary blood flow; a widened pulse pressure and bounding pulses, which occur in response to an increase in cardiac output; the presence of a ductal murmur (Wood 1997). As heart failure develops, additional signs are mottling of the skin, a prolonged capillary refill time, hypotension, a decrease in renal output and a respiratory and metabolic acidosis. Continuous monitoring of these infants' respiratory, cardiac and renal status enables the neonatal nurse to recognise any deviation from normal and initiate appropriate management. Diagnosis is usually made following clinical examination. Treatment is closure of the ductus.

The initial management for an infant with a patent ductus arteriosus involves supportive care, which includes fluid restriction, correction of anaemia and treatment of hypoxia and acidosis. Increased fluid and sodium intake in the first few days of life have been associated with an increased risk of patent ductus arteriosus. However, fluid restriction by itself is of limited value once the duct is symptomatic. Fluid restriction and the administration of a diuretic may be used to reduce pulmonary oedema if the infant is in acute heart failure (Cotton 1987). Anaemia may need to be corrected when a patent ductus arteriosus is present as a low haemaglobin increases the demand on left ventricular output to ensure an adequate oxygen supply to the tissues (Cotton 1987). By maintaining the infant's neutral thermal environment and oxygenation the neonatal nurse can help reduce the demands on left ventricular output.

Indomethacin, a potent prostaglandin inhibitor, may be used to close the duct. A suggested dose is 200 µg/kg every 12 hours for 3 doses (Laing 1998). More than one course may have to be given.

When administering indomethacin it is important that the neonatal nurse observes for signs of its side-effects (Kirsten 1996):

- Bleeding: oozing from venepuncture and heelstick sites, presence of petechiae, presence of blood in gastric aspirate or stools.
- Decreased renal function: urine output of <0.6 ml/kg per day, due to a decrease in renal blood flow and therefore a decrease in glomerular filtration rate, increased urea and serum creatinine >1.8 mg/dl.
- Electrolyte imbalance: hyponatraemia and hypokalaemia as a result of water retention, hypoglycaemia because of a transient decrease in plasma glucose.
- Hyperbilirubinaemia: due to displacement of bilirubin from binding sites.

Because indomethacin administration has adverse effects on gastrointestinal and renal blood flow, the urinary output, platelet count, blood coagulation, serum electrolytes and creatinine require to be monitored. Improvement in the infant's condition is indicated by improved oxygenation, the need for less ventilatory support, a decrease in pulse pressure, normal peripheral pulses and no evidence of a murmur.

Some neonatologists and cardiologists advocate prophylactic indomethacin for all preterm infants. Research has shown that in treated infants it has reduced the incidence of symptomatic patent ductus arteriosus and grade 3 and 4 intraventricular haemorrhage. However, with prolonged treatment there is no apparent improvement in morbidity and mortality; therefore further research is required before the routine use of this treatment can be recommended (Fowlie 1996). Surgical ligation may also be indicated for infants with major contraindications, or where medical treatment has failed.

Transposition of the great arteries

Transposition of the great arteries occurs when the aorticopulmonary septum fails to spiral during partitioning of the bulbus cordis and truncus arteriosus. This results in the aorta arising from the right ventricle and the pulmonary artery from the left ventricle and two independent circulations exist.

Oxygenated blood from the lungs is returned to the left atrium, enters the left ventricle and goes through the pulmonary artery to the lungs again. Deoxygenated blood from the systemic circulation enters the right atrium, flows to the right ventricle, then to the aorta and is directed back into the systemic circulation. This defect is incompatible with life unless there is a communication between the two circulations to allow mixing of the oxygenated and deoxygenated blood. This communication can be a patent ductus arteriosus, or via the atrial or

ventricular septum. However, a large ventricular septal defect offers the best mixing of blood.

The infant will present with marked central cyanosis, usually within the first 24 hours of life. The severity of the cyanosis is variable and will depend on the size of the aperture between the two circulations. On chest X-ray, an egg-shaped heart may be seen. This is caused by the aorta rising anterior to the pulmonary artery and right ventricular enlargement (Paul 1995). Diagnosis is usually made on echocardiography and colour flow Doppler. Despite the existing communication, an additional opening is usually made to promote mixing of blood: a cardiac catheterisation is performed and a balloon atrial septostomy carried out. One of the main responsibilities of the neonatal nurse is to transfer the infant to and from the cardiac catheterisation laboratory with minimum distress to the infant and family and to monitor the infant's vital signs after the procedure. Observations include: the infant's colour, oxygen requirements, temperature and colour of the affected limb, heart rate in the case of arrhythmias or bradycardia, and blood pressure as hypotension may indicate haemorrhage. Usually dinoprostone (PGE_2) is commenced prior to the procedure to maintain patency of the duct. It is vital for the neonatal nurse to be aware of the sideeffects of this therapy. Transient pyrexia, respiratory depression and apnoea are common, therefore intubation and ventilation equipment must be on hand. Other side-effects—hypotension, bradycardia, tachycardia—are related to 'flushing' the intravenous cannula. For this reason dinoprostone should not be infused with other drugs (Givens-Bell 1998). Side-effects may be reduced by administration via an umbilical catheter positioned so that its tip is at the origin of the ductus arteriosus (Givens-Bell 1998). If the septostomy and dinoprostone (PGE_2) do not sufficiently improve oxygenation, surgical intervention will be required. Surgical correction involves switching the arteries back to normal and closing the communication, or redirection of blood within the atrium and closing the communication. These procedures require open heart surgery. The type and timing of surgery depends on the clinical condition of the infant and whether the anatomical defect is simple or complex (Kirklin *et al.* 1990).

Hypoplastic left heart syndrome

Hypoplastic left heart syndrome is the underdevelopment of the left side of the heart and involves the aorta, the mitral valve and the left ventricle. There are usually no other accompanying cardiac defects. Infants with hypoplastic left heart syndrome are dependent on an interatrial opening and patency of the ductus arteriosus. Blood must flow from the right atrium, through the tricuspid valve to the right ventricle, which is often hypertrophied to a dilated pulmonary artery, then through the patent ductus arteriosus to the systemic circulation. Blood from the pulmonary artery also supplies the lungs, then returns to the left atrium via the pulmonary veins, where there must be a left-to-right shunt in order for the systemic circulation to receive any oxygenated blood. The left

ventricle is hypoplastic and therefore non-functioning. Patency of the ductus arteriosus must be maintained by the use of prostaglandin to ensure blood flow to the aorta.

The infant appears normal at birth then presents with mild cyanosis, tachypnoea with nasal flaring and grunting and a history of poor feeding. Gradually, as the ductus arteriosus closes, progressive cyanosis, poor peripheral perfusion, weak pulses, acidosis and circulatory collapse develop. Diagnosis is by echocardiography.

At present there are three treatment options open to families of infants with hypoplastic left heart syndrome: no treatment, surgical correction performed in stages, or heart transplantation. If no treatment is provided, the infant will experience cardiovascular collapse and die, usually within the first few days of life. Surgical correction involves two stages (Johnson and Davis 1991). The first stage—the Norwood procedure—is performed in the neonatal period to maintain pulmonary blood flow and create an atrial septal defect to allow mixing of blood. The second stage is a modified Fontan procedure, usually performed about 6 months of age, which involves bypassing the right ventricle by anastomosis of the right atrium to the pulmonary artery and closure of the atrial shunt. Heart transplantation raises many ethical questions. If transplantation is the chosen option then issues such as donor availability, organ rejection, immunosuppression and the psycho-social implications must be considered (Francis 1994).

For parents, the decision to treat or not to treat is extremely difficult. There are no guarantees that the treated infant will survive. Staff need to present the parents with all of the facts, the risks, the benefits, the possible outcome, the effects of drug therapy, without bias, in order for parents to make an informed decision. Whatever treatment option is chosen, the parents require all the support available. Staff cannot be judgemental. A multidisciplinary team approach can offer continuity and consistency of care. This allows the parents to express their feelings, discuss their fears and anxieties as well as explore coping strategies.

Coarctation of the aorta

Coarctation of the aorta is constriction or narrowing of the aorta near the ductus arteriosus and it may be classified as pre- or post-ductal. Pre-ductal narrowing of the aorta causes an obstruction to blood flow which leads to elevated pressure in the ascending aorta and left ventricle. The increase in left ventricular pressure causes left ventricular hypertrophy and dilatation. Preductal coarctation is associated with ventricular septal defects, transposition of the great vessels and patent ductus arteriosus in 40 per cent of cases. Post-ductal coarctation is not usually associated with other heart defects (Lott 1993). The symptoms of coarctation include tachypnoea, difficulty with feeding, higher blood pressure in the upper limbs than lower limbs and weak pulses in the legs. A murmur may or may not be present. The signs of congestive heart failure may also be present.

Diagnosis is made following clinical examination, chest X-ray, electrocardiogram and echocardiography. Initial medical management involves stabilisation of the infant, the administration of a prostaglandin infusion to maintain ductal patency and prevention or treatment of congestive heart failure. The nursing care would be directed towards the management of an infant receiving oxygen therapy, the care of a prostaglandin infusion, monitoring and assessing the infant's vital signs, comfort measures and support of the family.

Surgical correction is usually performed at 3–5 years of age if the signs and symptoms can be medically managed until that time, otherwise surgical intervention is required. This involves the excision of the constricted segment of the aorta with end-to-end anastamosis and patch graft or subclavian flap aortaplasty. Long-term antibiotic therapy is required to prevent subacute infective endocarditis.

Congestive heart failure

A complication of congenital heart disease is congestive cardiac failure. This is the inability of the heart to pump an adequate amount of blood to the systemic circulation (Lott 1993). Obstruction to flow, ineffective myocardial function or any condition in which the circulatory demand is greater than the cardiac capacity because of increased volume can cause congestive cardiac failure.

The clinical manifestations of congestive cardiac failure are: decreased cardiac output, pulmonary venous congestion, systemic congestion and electrolyte imbalances. Decreased cardiac output stimulates the sympathetic nervous system causing tachycardia, increased contractility and peripheral vasoconstriction. It also causes reduced blood flow to the kidneys, which stimulates the angiotension-aldosterone mechanism causing re-absorption of sodium and water. Pulmonary venous congestion results in tachypnoea, dyspnoea, intercostal recession, flaring nostrils, grunting and rales. Pulmonary oedema and decreased lung compliance can also occur. Systemic congestion results in increased pressure and pooling of blood in the venous circulation. The liver may be enlarged and oedema in the soft tissues present. Because of decreased systemic blood flow, arterial blood gases show a metabolic acidosis. A concurrent respiratory acidosis due to pulmonary oedema caused by left-sided heart failure may also occur. Electrolyte imbalances are also usually present. Hypoglycaemia may occur because the myocardium is dependent on glucose. Decreased glucose levels lessen the heart's ability to compensate in congestive cardiac failure. Diagnosis is based on clinical signs and symptoms, chest X-ray and laboratory data.

The aim of treatment is to improve cardiac function whilst identifying the underlying cause. Oxygen therapy and ventilatory support will improve alveolar perfusion. Fluid restriction will reduce circulating blood volume. Dopamine used at a low dose will improve renal blood flow. Dobutamine improves cardiac output (Givens-Bell 1998). Digoxin may also be used in the management of

congestive heart failure. It slows conduction through the atrioventricular node and heart rate through vagal effects on the sinoatrial node. Digoxin levels need to be monitored because there is a narrow range between therapeutic and toxic drug levels (Lott 1993). Diuretics are useful in the treatment of congestive heart failure as they increase the renal excretion of sodium by inhibiting tubular re-absorption of sodium. An accurate measurement of fluid intake, output and serum electrolytes must be maintained in infants with congestive heart failure.

The prognosis for infants with congestive heart failure depends on the severity of the underlying disease and the degree of failure.

Nursing management

Nursing staff caring for the newborn infant must be able to recognise the signs and symptoms of heart disease, undertake an accurate clinical assessment, initiate referral to allow for early intervention and treatment, implement a nursing care plan and offer family support.

Clinical assessment

A clinical assessment enables the neonatal nurse to identify an infant with a cardiac problem. The assessment involves a physical examination of the infant, which includes: observation of the infant's general appearance, an evaluation of peripheral perfusion, pulses and blood pressure, palpation of the chest wall and liver and auscultation of the heart.

Physical examination

Observation of the infant's general appearance includes colour, respiratory pattern and posture. Examination should ideally be carried out when the infant is resting quietly or asleep.

Colour

Note the colour of the skin, mucous membranes and nailbeds. The skin colour should be observed for pallor, plethora or cyanosis. Pallor may be due to vasoconstriction resulting from severe anaemia, sepsis, pulmonary disease or congestive heart failure. The polycythaemic infant may appear cyanosed. Evaluating the haemoglobin, haematocrit and PaO_2 can help exclude the possibility of cardiac disease. Generally, when an infant's colour improves with crying or supplemental oxygen, the cyanosis is considered to be pulmonary in origin. If, however, the infant's colour worsens with crying and there is little improvement with oxygen or the infant remains cyanosed, the origin is likely to be cardiac. A hyperoxic test, measuring

the infant's PaO_2, may help differentiate between pulmonary and cardiac disease. If the PaO_2 rises to above 150 mmHg or 20 kPa then the cyanosis is probably caused by pulmonary disease (Turrell *et al.* 1994). An infant with an interruption of the aortic arch may present with cyanosis of the lower limbs and left arm, with a pink upper body and right arm. This is due to pulmonary blood flowing through the ductus arteriosus to supply the lower body while the upper body receives well-oxygenated blood from the ascending aorta.

Respiratory effort

Observation of the infant's respiratory pattern should include rate, regularity and effort of breathing. In the infant with heart disease the respiratory pattern may be either regular rapid shallow breathing or irregular with slow deep breathing accompanied by sternal and intercostal recession. Tachypnoea and tachycardia are early signs of left ventricular failure. An increased oxygen requirement may suggest a patent ductus arteriosus.

Evaluation of the pulses, peripheral perfusion and blood pressure

The pulses in all four limbs should be evaluated, blood pressures obtained and peripheral perfusion assessed. Pulses easily felt in the newborn are the brachial, radial, femoral, posterior tibial and dorsalis pedis. Strong bounding pulses suggest a patent ductus arteriosus. Weak pulses indicate low systemic output, as is found in left heart obstructive lesions. A widened pulse pressure, the difference between systolic and diastolic pressures, may be present in patent ductus arteriosus and truncus arteriosus or narrowed, as in heart failure. If the systolic blood pressure in the upper extremity is more than 20 mmHg greater than that in the lower extremity, this may indicate a post-ductal coarctation of the aorta (Johnson 1990). If a pre-ductal coarctation of the aorta exists, then the blood pressure is usually higher in the right arm than the left (Monett and Moynihan 1991).

Peripheral perfusion

Peripheral perfusion can be assessed by determining capillary refill time. One method of testing refill time is to press the great toe, then release it. The refill time is counted in seconds, the normal being 2 seconds. However, due to peripheral vascular adaptation following birth, refill may take 3–5 seconds. Greater than 5 seconds is abnormal in the newborn and reflects diminished perfusion of the tissues.

Palpation of the chest wall and the liver

The chest should be palpated to determine the quality and strength of cardiac activity and the location of thrills. A thrill is a palpable vibration that is produced as the blood flows through a defective valve or stenotic vessel (Monett and Moynihan 1991). Thrills are best felt in the upper left, upper right and lower left sternal border, in the suprasternal notch and over the carotid arteries. The liver's edge should be palpated next. This is normally no more than 3 centimetres below the right costal margin. Hepatomegaly is usually a late sign and is due to venous congestion in the liver. It is important if hepatomegaly presents early that haemolytic disease and sepsis are ruled out.

Auscultation

Auscultation involves the assessment of heart rate and rhythm, heart sounds and the presence of murmurs. There are four distinct heart sounds: S_1, S_2, S_3 and S_4 (Lott 1993); however S_3 and S_4 are rarely heard in the newborn period. The first heart sound S_1 ('lub') is produced during closure of the mitral and tricuspid valves, and is best heard at the lower sternal border or at the apex of the heart. S_2 ('dub') is created by the closure of the pulmonary and aortic valves and is best heard at the second intercostal space. Murmurs are caused by turbulent blood flow through the heart and are classified according to loudness (grade 1–6), timing (systolic or diastolic), location on the chest and quality. Tachycardia is more commonly associated with cardiac abnormalities than bradycardia (Verklan 1997). Although palpation and auscultation are normally performed by medical staff or advanced neonatal nurse practitioners, these elements of the nursing assessment with practice and experience are not beyond the capabilities of neonatal nurses.

Once the initial assessment has been completed, diagnosis of congenital heart disease can be confirmed by chest X-ray, electrocardiogram, echocardiography and colour flow Doppler, cardiac catheterisation and angiography.

Care plan

The nursing care of infants with congenital heart disease would aim to:

- minimise environmental stimulation by correct positioning and nesting which can reduce energy consumption and by offering pacifiers or sedation for irritability;
- maintain an optimal neutral environmental temperature to minimise oxidative metabolism;
- administer oxygen therapy as ordered to maintain oxygenation parameters for specific defect;
- time nursing activities to disturb the infant as little as possible;

- offer small frequent feeds to prevent fatigue and further cardiac compromise (see p. 242 and p. 248);
- provide continuous monitoring of the heart rate, respiratory rate, blood pressure, temperature, oxygen saturation, fluid input and output;
- administer medications safely and observe the infant for signs of improvement or adverse reactions;
- encourage the family to participate in the care of their infant.

Family support

Most parents look forward to the birth of their baby with excitement, elation and hopes for the future. For the parents of an infant diagnosed with heart disease these emotions are replaced with feelings of anger, guilt and fear (Rees *et al.* 1992). They are confused as to why this has happened when their baby appeared healthy at birth. The uncertainty of what the future holds causes a great deal of anxiety and stress. Parents require open, honest, truthful explanations about the severity of the defect, availability of treatment, the prognosis and future implications. Some infants may require to be transferred to a specialist unit for further investigation and treatment, often resulting in the mother and infant being separated from the rest of the family. There may be the added burden of financial costs of travel, accommodation and child care for siblings. In the midst of all this emotional turmoil parents also have to cope with making decisions about the short- and long-term treatment options for their child.

The neonatal nurse therefore plays an integral part in the support of parents and their families by providing continuity of care and further information in the form of diagrams and written material on their infant's particular defect, a resource list for financial assistance and referral to either a local or national support group. Many units offer support by referring parents to other families who have experienced a similar situation. The neonatal nurse acts as a vital link between parents, their other children and members of the family by recognising these persons' individual needs and facilitating their understanding of the situation.

Another important role for the neonatal nurse is the education of parents of infants who do not require immediate surgery and who are being discharged home. Parents often feel very inadequate to provide care for their child; they are suddenly taking on fully a responsibility that up till now has been shared with hospital staff. Careful discharge planning is essential for these infants and includes teaching parents to recognise the signs and symptoms of deterioration, what to do in an emergency, the administration of medicines, how to overcome feeding problems and how to limit activity. Close follow-up by the cardiologist and home care nurse must be arranged to help prevent complications arising and to provide ongoing support for the family.

Case study: term infant with congenital heart defect

Andrew was delivered by spontaneous vertex delivery at term following an uncomplicated pregnancy. Apgars were 8 at 1 minute, 9 at 5 minutes. At birth he appeared a normal healthy baby and was transferred to the postnatal ward with his mother. His weight was 3540 g. At 48 hours of age Andrew was noted to be tachypnoeic, pale with mottling of the skin and not interested in breast feeding. The paediatrician was called and Andrew was transferred to the neonatal unit. On admission a nursing assessment was carried out, the findings being:

- General: lying awake, not irritable or crying.
- Colour: pale/grey with mottling of the skin.
- Respirations: tachypnoeic, no evidence of grunting, nasal flaring, sternal or intercostal recession.
- Cardiovascular: heart rate 180 beats per minute and regular, a ductal murmur was heard, pulses were diminished especially in the legs, discrepancy noted between upper and lower limb blood pressures, capillary refill time 4 seconds; the liver was not enlarged.

Andrew was placed in 40% oxygen initially, saturation was 92%. A full infection screen was carried out to exclude sepsis. Chest X-ray showed an enlarged heart with a slight streaky appearance over both lung fields. Antibiotics and intravenous fluids were commenced. Andrew's colour deteriorated, his tachypnoea worsened and his oxygen requirements increased. Arterial blood gases showed a normal pH, PCO_2, and base deficit with a low PO_2. He was referred to the cardiologist who performed an echocardiogram and discussed treatment options with the parents.

Q.1. Which congenital heart defect could Andrew have?
Q.2. What could be the differential diagnosis?
Q.3. What initial treatment would be commenced?
Q.4. Describe Andrew's nursing care.

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Chapter 7

Brain Injury in the Premature Infant

Contents

Introduction: the vulnerability of the premature infant brain

Periventricular-intraventricular haemorrhage (PIVH), parenchymal haemorrhagic infarction (PHI) and periventricular leukomalacia (PVL) are the major causes of brain injury in the premature infant. Before an understanding of the pathogenesis and risk factors for these problems can be gained, it is essential to appreciate the factors that cause the premature infant's brain to be vulnerable to injury (Volpe 1997).

Subependymal germinal matrix

The subependymal germinal matrix is an embryonic structure which is the site of neuronal and glial precursors that eventually migrate to form the cerebral cortex. This area of cellular proliferation is found immediately to the front and side of the lateral ventricles. The matrix is at its largest and most active between 24 and 32 weeks of gestation. As the brain matures, however, the matrix progressively decreases in size, so that by 36 weeks it has almost disappeared. Prior to this involution, a number of factors make the matrix fragile and prone to bleeding.

The matrix is highly cellular, gelatinous in texture and richly vascularised. Until 32 weeks this region receives a large proportion of the cerebral blood flow. The blood vessels, however, are large, thin-walled and have been described as simple 'channels' (Volpe 1995). The gelatinous structure of the matrix gives little support to the vessels running through it. As the cells of the matrix migrate towards the cortex, there is a constant process of remodelling. This remodelling is associated with an excessive amount of fibrinolytic activity, thought to prevent early clotting and thus enabling a small haemorrhage to enlarge rapidly.

Should the periventricular area become over-perfused, these factors make the vessels of the matrix susceptible to rupture and haemorrhage. In addition, injury can occur when under-perfusion is followed by a return to normal blood pressure. Being an area of cellular proliferation and growth, the matrix has a high oxygen requirement for metabolism. When blood supply is interrupted hypoxic-ischaemic damage results, leaving the vessels prone to rupture when reperfusion occurs.

Periventricular vascular anatomic factors

A well-developed venous system drains blood from the matrix, cerebral white matter, choroid plexus and thalamus. The course of this venous system takes an unusual U-turn before it drains into the vein of Galen. This U-turn occurs at the most common site of germinal matrix haemorrhage. This is thought to have implications in the pathogenesis of PIVH and PHI (Volpe 1997) when associated with an increase in cerebral venous pressure.

The arterial circulation to the developing premature brain forms short and long penetrators (arteries) which eventually grow deep into the cerebral white matter. At 24–28 weeks, however, these penetrating arteries are few in number and have few interconnections. The areas of the brain where the penetrators end, in the deep periventricular region, are known as end zones or watershed areas. These watershed areas have a tenuous blood supply and are most susceptible to under-perfusion and ischaemia should a fall in cerebral blood flow occur. From 32 weeks to term the number of penetrators and their connections increases, and thus the vulnerability of the periventricular region to ischaemia decreases.

Alterations to cerebral blood flow

Autoregulation is the ability to maintain a constant cerebral blood flow, by arterial vasodilatation or constriction, in the event of systemic hypotension or hypertension. Premature infants have a limited capacity for autoregulation. Hypoxia, hypocarbia and hypercarbia blunt this capacity further. Thus, when a premature baby is exposed to fluctuations in systemic blood pressure, this will be directly relayed to the brain creating a pressure-passive cerebral circulation. Raised cerebral blood pressure may cause rupture of the fragile blood vessels of the matrix. Lowered cerebral blood pressure may damage those vessels, leaving them prone to rupture when the blood pressure returns to normal. In addition, low cerebral blood pressure may leave watershed areas ischaemic and hypoxic.

Hypoxic-ischaemic reperfusion injury

A well-established consequence of ischaemia and reperfusion is the release of oxygen-free radicals. The cerebral white matter of the premature infant, specifically the developing oligodendrocytes, are vulnerable to free radical attack. This can lead to apoptosis (cell self-destruction), loss of oligodendrocytes and impairment in myelinisation.

Risk factors for brain injury

As previously described, the premature brain is vulnerable to damage due to the immature nature of its anatomy and the possible presence of a passive-pressure circulation. The pathogenesis of PIVH, therefore, is strongly linked to events that cause hypoxia and/or alterations to the infant's systemic blood pressure and cerebral blood flow.

Perinatal factors linked to hypoxia include maternal bleeding, fetal distress, perinatal asphyxia, prolonged labour and abnormal presentations. Following birth, hypoxic events such as apnoea, respiratory distress or hypotension are associated with PIVH. In addition, studies have correlated infants with a patent

ductus arteriosus and the occurrence of fluctuations in cerebral blood flow (Volpe 1997).

Ventilated premature infants are at particular risk due to acid-base imbalances and the potential complication of pneumothaorax. An identified subset of infants with respiratory distress syndrome (RDS) have been shown to be at extreme risk of subsequent PFVH. These infants demonstrated a fluctuating systemic arterial blood pressure, with variable peaks and troughs in systolic and diastolic flow. These fluctuations were shown, by Doppler ultrasound, to be related to cerebral blood flow. Babies who breathe out of synchrony with the ventilator can experience these fluctuations. The data from a randomised controlled trial conducted by the HIFI Study Group (1989) showed a greater incidence of PIVH in infants assigned to the high-frequency ventilator with active expiration. The design and ventilator strategy used in the study are thought to have contributed to this outcome. However, further research is required to refute or confirm this link.

Increases in cerebral blood flow have been linked to episodes of systemic hypertension. These events may be associated with care-taking procedures such as the noxious stimulation that occurs with tracheal suctioning, inappropriate prolonged handling, crying and environmental factors such as excessive light and noise (Dietch 1993). Rapid volume expansion, marked hypercarbia causing vasodilatation of the cerebral arteries, decreased haematocrit and blood sugar levels can also result in increased cerebral blood flow (Volpe 1995).

Decreases in cerebral blood flow prenatally or following birth may play an important role in the pathogenesis of PIVH and PVL. Although the mechanisms that link perinatal asphyxia to these conditions are complex, this is clearly a significant event. Postnatal hypotensive events that lead to decreased cerebral blood flow include severe apnoea, myocardial failure and sepsis.

Impeded venous return which leads to elevation of cerebral venous pressure (CVP) may contribute to the occurrence of PIVH. A significant cause for increased CVP is compression of the compliant premature skull during labour and vaginal delivery. Compression of the skull can also occur with the use of constricting head bands or hats (Cowen and Thorensen 1985). Examples of equipment that could produce such compression, if applied too tightly, are bands to apply eye protection for phototherapy or hats used to secure continuous positive airway devices. Impeded venous return has been associated with aspects of mechanical ventilation such as excessive positive end-expiratory pressure, prolonged inspiratory pressure and high positive-inspiratory pressure (Volpe 1995).

Infusions of sodium bicarbonate have been implicated in the causation of PIVH. There are, however, conflicting reports on the mechanism. Rapid administration and its osmotic properties may play a part, but it is thought that an abrupt rise in arterial carbon dioxide (due to buffering effects) and the subsequent increase in cerebral perfusion is also important.

Periventricular-intraventricular haemorrhage (PIVH)

Periventricular-intraventricular haemorrhage occurs in approximately 25–40 per cent of infants who weigh less than 1500 g or who are born at less than 32 weeks' gestation (Volpe 1995). The essential feature of periventricular haemorrhage is bleeding into the subependymal germinal matrix. If bleeding continues, blood enters the lateral ventricles (intraventricular haemorrhage) and spreads through the ventricular system. Ultrasound scanning has made it possible to describe the distribution and severity of PIVH according to grades I–III, as illustrated in Table 7.1.

***Table 7.1* Grading scale for periventricular haemorrhage**

Grade I	Germinal matrix haemorrhage with little or no blood in the ventricles
Grade II	Intraventricular haemorrhage filling up to 50 per cent of the ventricles
Grade III	Intraventricular haemorrhage filling greater than 50 per cent of the ventricles and causing dilatation

Based on Volpe 1995

Clinical features

The timing of the initial clinical features is consistent with the occurrence of haemorrhage. That is, within the first day of life for 50 per cent of infants, with 90 per cent of haemorrhages occurring by 72 hours. (Three clinical syndromes have been described in association with PIVH and PHI: catastrophic, saltatory and silent.)

The catastrophic syndrome is the most dramatic but least common presentation, in which neurological deterioration occurs rapidly over minutes to hours. The neurological features of this syndrome include deep stupor or coma, hypoventilation, apnoea, seizures, decerebrate posturing, unreactive pupils, flaccid quadriparesis. Other features include a falling haematocrit, a bulging anterior fontanelle, hypotension, bradycardia, temperature instability, metabolic acidosis and water and glucose imbalances.

The evolution of the saltatory syndrome occurs over hours to days, during which the bleeding may stop and start. The most common features are an alteration in the level of consciousness, a decrease in spontaneous movements, hypotonia, abnormal eye position and movement, an abnormally tight popliteal angle and respiratory disturbance. Other non-neurological signs could include a low packed cell volume that does not respond to blood transfusion and persisting jaundice. There may be a lack of improvement in the infant's general condition despite an improving chest X-ray. The subtlety of this syndrome may cause it to be missed in some infants or confused with infection.

Most common is the silent syndrome. In 25–50 per cent of infants with PIVH careful serial neurological examination will not show any evidence of a bleed. Diagnosis is made by cranial ultrasound.

Cranial ultrasound is an excellent method for the identification of PIVH and PHI and has become the mainstay in diagnosis of these lesions since the 1980s. Advantages include its portable nature, which enables rapid diagnosis at the bedside without the need for transportation of an unstable infant. In addition, it does not expose the infant to ionising radiation (Ramey 1998).

Parenchymal haemorrhagic infarction

Rather than a fourth grade in the severity of PIVH, PHI is now considered to be a complication of this condition. Eighty per cent of cases are associated with a severe PIVH. PHI is a relatively large, asymmetric region of haemorrhagic necrosis in the periventricular white matter, located just dorsal and lateral to the external angle of the lateral ventricle. The necrotic area may eventually form a porencephalic cyst. It appears that PHI is a venous infarction and is distinguishable from PVL, although the two processes may occur at the same time. The PIVH creates congestion in the periventricular venous system. This build-up of pressure disrupts the blood supply, resulting in ischaemia and an area of infarction into which bleeding occurs as the perfusion returns (Volpe 1997).

Periventricular leukomalacia (PVL)

PVL is predominantly a lesion found in premature infants, although it has been diagnosed in term babies. The proven incidence in premature infants has varied from 25 to 75 per cent (Volpe 1997). PVL consists of necrosis of white matter in a characteristic distribution. Focal lesions occur in the end zones or 'watershed' areas, where the long penetrating arteries terminate. These are situated dorsal and lateral to the external angles of the lateral ventricles.

Initially evident as areas of increased echodensity or 'flaring' on ultrasound, this is a transient finding in some infants. While in others, after one to three weeks, multiple cavities form giving a 'Swiss cheese' appearance. Ventricular dilatation and myelin loss follow. More diffuse regions of cerebral white matter injury have also been observed, although these regions are less likely to undergo cystic change and may be undetected by ultrasound during life.

Electroencephalographic (EEG) studies have identified a unique neonatal pattern quite specific for white matter injury. This pattern, associated with positive rolandic sharp waves (PRSW), may appear when white matter lesions are still echodense but not yet cystic on ultrasound. While PRSW are specific, being found in 80–100 per cent of neonatal EEGs with documented white matter injury, they appear to lack sensitivity in detection in extremely low birth weight infants (Connell *et al.* 1987). Magnetic resonance imaging (MRI) is an increasingly used investigation and is currently the most sensitive tool

for the detection of white matter injury in the premature infant. Advanced MRI techniques with quantitative volumetric analysis are an area of current research which may provide a more sensitive analysis of the impact of prematurity and brain injury on subsequent cerebral development (Inder *et al.* 1999).

Management

Prevention of brain injury in the premature infant

The ultimate, but elusive, aim in the prevention of brain injury in this group of infants is the prevention of premature birth. Despite this, the incidence of PIVH and PHI has fallen in recent years due to the success of preventative strategies. This trend has not been evident in the incidence of PVL, which continues to be a major cause of death and neurological impairment in the premature infant (Volpe 1997). Prevention can be divided into prenatal and postnatal strategies. A number of pharmacological interventions have also been evaluated.

Prenatal strategies focus on the delivery of high-risk pregnancies in a perinatal centre that can provide optimal management of labour, delivery, resuscitation and neonatal care. This may necessitate in utero transfer. Infants transported in utero are reported to have a lower incidence of PIVH (Volpe 1995). A course of antenatal steroids administered to mothers prior to premature delivery results in a lower incidence of respiratory distress syndrome. Since infants with respiratory distress syndrome are known to be at high risk of PIVH, reducing the incidence of the former also reduces the incidence of the latter (Leviton *et al.* 1993).

Postnatal strategies focus on preventing those events that cause hypoxia or fluctuations in systemic or cerebral blood flow, outlined in the previous section on risk factors. In addition, muscle relaxation with neuromuscular blocking agents such as pancuronium bromide have been shown to be beneficial in reducing the sudden changes in arterial blood pressure associated with endotracheal tube suction (Franconi and Duc 1987), pneumothorax and breathing out of synchrony with the ventilator (Greenough *et al.* 1984). Muscle relaxation does have disadvantages and has been associated with increased oxygen requirements and failure of skeletal muscle growth in low birth weight infants. Therefore other methods for reducing the risks in ventilated infants should be explored. This might include the use of patient-initiated (triggered) ventilation (Heldt and Bernstein 1994). Endotracheal suction protocols that advocate performing the procedure on clinical indications only, preoxygenation, avoidance of deep uncontrolled suction and limiting suction time to 10–15 seconds will minimise adverse effects (Wallace 1998).

A randomised controlled trial to investigate the effectiveness of individualised developmental care in reducing neurodevelopmental sequelae for very low birth weight infants showed a reduced incidence of PIVH in the group of infants receiving this type of care (Als *et al.* 1994). The authors attribute this reduction

to developmentally individualised care (see p. 34), yielding calmer infants with reduced cerebral blood flow velocity changes and thus fewer PIVHs. It is beyond the scope of this chapter to describe fully the concepts behind the model of care, but the essential elements to the delivery of nursing care are contained in Dietch (1993), which is recommended reading.

Pharmacological interventions

Phenobarbarbitone has been evaluated as a prenatal and postnatal pharmacological intervention. The proposed mechanism of action relates to a reduction in abrupt arterial blood pressure and thus cerebral blood flow associated with spontaneous activity such as crying, and care-giving activities such as tracheal suction. Indomethacin given postnatally is thought to decrease cerebral blood flow by inhibiting cerebral prostaglandins that cause vasodilatation. Ethamsylate is thought to stabilise the capillaries of the germinal matrix and promote platelet adhesiveness, both of which would inhibit bleeding. The beneficial effects of vitamin E relate to the protection of capillary cells from hypoxic injury by scavenging free radicals. However, none of these drugs has sufficient proved beneficial effect to warrant its routine use in premature infants (Volpe 1995).

Acute management

In 20–40 per cent of infants the initial haemorrhage will enlarge (Volpe 1995). Therefore infants with a demonstrated haemorrhage must be evaluated using repeat cranial ultrasounds because ethical issues about the continuation of treatment arise for infants with a grade III PIVH or PHI. Changing the focus of care, to one of palliation rather than cure, is a complex and highly sensitive decision. When parents are provided with honest and accurate information, regarding short- and long-term outcomes, they can participate in discussion and decision-making for their infant (Van Putte 1988) (see p. 424).

Acute management focuses on supportive measures such as ensuring adequate ventilation and oxygenation, maintaining normal temperature, acid base and glucose balance. Maintenance of a normal arterial blood pressure is essential. This may necessitate the use of volume expanders or inotropic drugs. However, these must be used cautiously in view of the possibility of a passive-pressure circulation, where sudden or excessive increases in arterial blood pressure could exacerbate bleeding. Attention to the preventive measures discussed previously is vital. In rare cases lowering intracerebral pressure by lumbar puncture is beneficial (Volpe 1995).

Post-haemorrhagic ventricular dilatation

Thirty-five per cent of infants develop progressive ventricular dilatation following PIVH or PHI (Volpe 1995). The disruption to flow of the cerebrospinal fluid

(CSF) is due to blockage within the ventricular system or an obilerative archnoiditis preventing reabsorption. Close surveillance is essential for any infant who has dilated ventricles. Surveillance includes serial cranial ultrasound, measurement of head circumference and observation of level of consciousness and activity.

The ventricular dilatation resolves spontaneously in less than four weeks in the majority of these infants. If the dilatation continues beyond four weeks serial lumber punctures or drugs to reduce CSF production, such as acetazolamide, are used. The dilatation will fail to arrest or progress rapidly in 15 per cent of infants. These infants require ventricular drainage either via an external ventricular drain in the short term, or the insertion of a ventriculoperitoneal shunt for long-term management of hydrocephalus (Volpe 1995).

Nursing management

Awareness of which infants are at risk and what factors may precipitate a haemorrhage are primary to the nurse's role in the prevention and management of brain injury in the premature infant. In addition, the nurse must be vigilant to detect subtle signs of haemorrhage such as changes in activity, level of consciousness or abnormal eye movements (Rozmus 1992).

Two important aspects to the nursing role are monitoring and minimising swings in haemodynamic stability and the reduction of noxious environmental stimuli. Dietch (1993) has devised a comprehensive table covering major nursing interventions to reduce the risk of PIVH. Prevention of hypoxia and haemodynamic instability are also major nursing aims in the prevention of PVL (Doran 1992). Therefore, the interventions outlined in Dietch's table are applicable nursing strategies for prevention of all types of brain injury in the premature infant and this table is recommended to the reader.

The outcome of brain injury in the premature infant

Periventricular-intraventricular haemorrhage

The outcome of PIVH relates to the severity of the haemorrhage. Outcomes include death or progressive ventricular dilatation in the short term, and in the long term, neurological sequelae such as spastic motor and cognitive deficits.

Infants with grade I PIVH have mortality rates similar to infants without haemorrhage (5 per cent). The mortality rates are 10 per cent for infants with grade II haemorrhage. With grade III haemorrhages the mortality rate increases to 20 per cent. Ventricular dilatation in survivors is rare for grade I, but rises to 25 per cent for grade II and 55 per cent for grade III. Neurological sequelae occur in 5, 15 and 35 per cent of infants respectively (Volpe 1995).

Parenchymal haemorrhagic infarction

Adverse outcomes for this group of infants is markedly higher. Severity is related to the degree of injury to the cerebral white matter rather than the actual amount of blood. Fifty per cent of these infants die, while 80 per cent of survivors have ventricular dilatation and 90 per cent have neurological sequelae (Volpe 1995).

Periventricular leukomalacia

Approximately 60–90 per cent of infants with cystic changes associated with PVL will have neurological deficits on follow-up. The major long-term sequela is spastic diplegia, with spastic paresis of the extremities affecting the lower, more frequently than the upper limbs. This is explained by the passage of descending fibres from the motor cortex that supplies the lower extremities, through the area of the cerebral white matter affected by PVL. Larger lesions will also affect upper extremities and intellectual function (Volpe 1997).

Case study: infant at risk of PIVH

When her mother was diagnosed with pregnancy-induced hypertension, Jody was transferred in utero to a perinatal centre at 28 weeks' gestation due to the possibility that a premature delivery would be necessary. Following deteriorating Doppler studies, Jody was delivered by cesarean section. She weighed 960 g. Jody required resuscitation at birth and was transferred to the neonatal intensive care unit, intubated and ventilated.

Q.1. What antenatal factors put Jody at risk of PIVH?

On arrival she was placed in a humidified incubator and attached to a ventilator on intermittent positive-pressure ventilation. Intravenous fluids via a peripheral intravenous cannula were commenced. An umbilical arterial line was inserted for blood pressure and arterial blood gas monitoring. A chest X-ray revealed findings consistent with respiratory distress syndrome and Jody was given the first of two doses of surfactant. During her first 24 hours of life Jody had a significant problem with hypotension which required volume expansion and an infusion of dopamine. Her blood pressure became unstable again on day 3, when a patent ductus arteriosus was diagnosed. A periventricular haemorrhage with blood present in normal sized ventricles was diagnosed by a routine cranial ultrasound on day 7.

Q.2. From Jody's clinical history, how could the presentation of her PIVH be described?

During these first days the nurse assigned to Jody's care paid vigilant attention to avoiding fluctuations in blood pressure from care-taking procedures such as endotracheal suction by rationalising the need for such procedures and responding to Jody's cues rather than delivering care via a set routine. Environmental stressors were minimised. An incubator cover was used to reduce the effects of bright lighting, and noise around the incubator was reduced as much as possible.

Q.3. Identify two major nursing roles in the prevention and management of PIVH.

A follow-up ultrasound on day 14 revealed that further bleeding had occurred and the ventricles were now slightly dilated with blood. Jody's condition had by now improved so that she could be extubated to nasal continuous positive airways pressure. Although further bleeding at this stage was unlikely, regular ultrasounds were planned, due to the risk of post-haemorrhagic ventricular dilatation. The scans over the following 4 weeks showed slow progressive dilatation of the ventricles. Jody's head circumference over this time remained within normal parameters and she appeared well.

Q.4. What is the likely outcome for Jody?

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Chapter 8

Haematological Problems

Contents

Introduction

Caring for infants with any haematological disorder provides the neonatal nurse with many challenges, from correctly interpreting clinical signs of the condition to monitoring changes and, of course, to providing information and support to the parents. In order to meet these challenges knowledge of pathophysiology and treatment modalities is necessary. This chapter will review four of the disorders: jaundice, haemorrhagic disease (vitamin K deficient bleeding), anaemia and disseminated intravascular coagulopathy. A background knowledge of basic anatomy and physiology in relation to haematology and intrinsic and extrinsic clotting pathways is needed.

Jaundice in the newborn

Caring for infants with jaundice seems to be a universal aspect of the work of neonatal units. In fact, there are estimates that some 50–70 per cent of newborns develop hyperbilirubinaemia (high levels of bilirubin in the blood) leading to jaundice (Schwoebel and Sakraida 1997). It is therefore important to understand how jaundice occurs, what treatment modalities are available, how they work and finally what the complications of jaundice and its treatments are.

Physiology of bilirubin production

Bilirubin is primarily a product of the breakdown of the protein haemoglobin (Hb), which is a major constituent of red blood cells. These soft pliable biconcave non-nucleated cells have a lifespan of between 40 and 70 days in the neonate (compared with 120 days in an adult). Up to 85 per cent of the bilirubin produced by the neonate is from this process with the remaining 15–20 per cent coming from non-haem proteins, such as cytochrome P450 and myoglobin, in the liver and bone marrow (Merenstein and Gardner 1998). The red cells are removed from the circulation by the reticuloendothelial system, principally the liver and spleen. The haemoglobin is then broken into globin—a reusable protein —and haem. The haem is degraded further by the substrate inducible enzyme microsomal haemoglobin oxygenase that is present in the liver, spleen and macrophages. Biliverdin is produced, which itself is broken down further by biliverdin reductase into bilirubin (Figure 8.1). One gram of haemoglobin produces approximately 600 µmol of bilirubin that is unconjugated and fat-soluble. This means that it cannot be excreted via the gut or kidney but instead has an affinity for fatty tissue and the brain.

Bilirubin metabolism and excretion

Conjugation occurs in the liver but free bilirubin cannot enter the hepatic cells unaided, so bilirubin first binds itself to albumin: 16 mg of bilirubin

binds to 1 g albumin, although this ratio might be lower in the very small/sick infant. Once bound, the bilirubin enters the smooth endoplastic reticulum of the hepatocyte in a carrier-mediated process, with the help of carrier proteins Y (ligandin) and Z. Here a series of reactions occur, catalysed by the enzyme uridine diphosphate (UDP) glucuronyl transferase. The bilirubin unites with glucuronic acid to produce bilirubin diglucuronide or conjugated bilirubin (Figure 8.1). This is water-soluble and thus can be secreted from the gut as stercobilin and the kidney as urobilinogen.

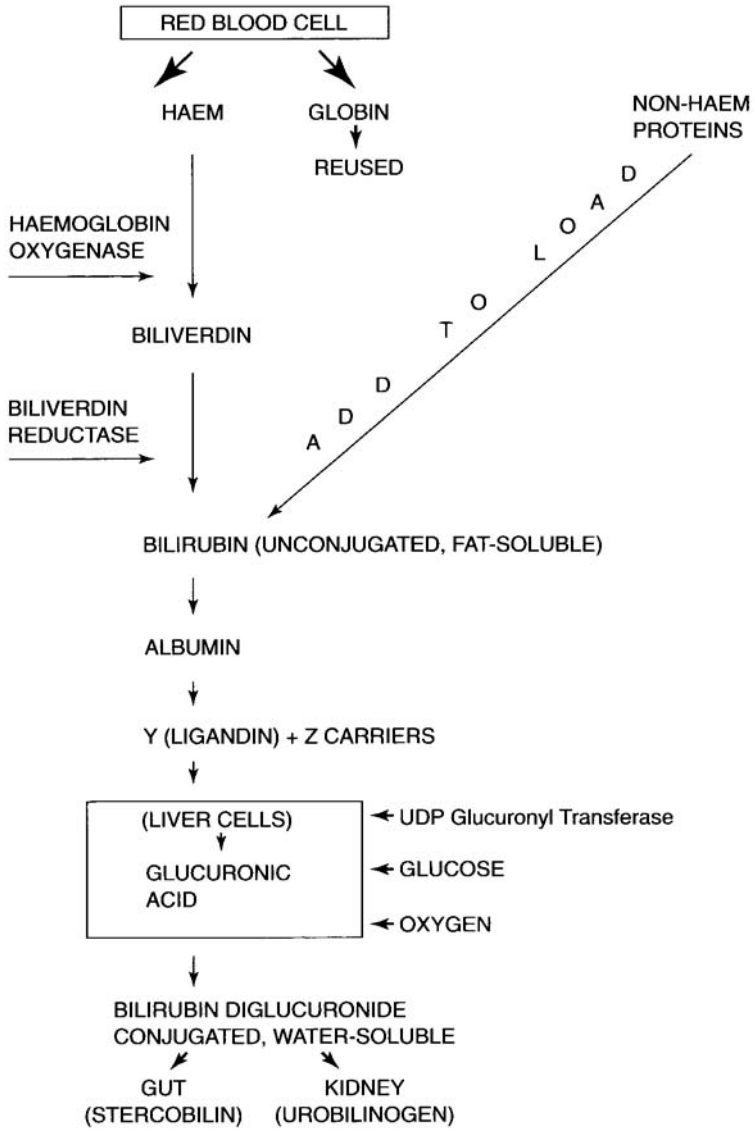

Figure 8.1 **Physiology of bilirubin production**

Predisposing factors to physiological jaundice in the newborn

Physiological changes that should occur during transition from intra- to extrauterine life can be overwhelmed and the infant develops a condition known as physiological jaundice:

- Newborn babies have a relatively high haemoglobin level (18–19 g/dl), which was needed by the fetus to maintain oxyhaemoglobin levels. This coupled with the short red cell life means that there is a high rate of haemolysis. Compared to adults on the basis of body mass bilirubin production in the neonate is more than double, at 135–170 µmol bilirubin/kg body weight per day.
- Newborn infants are thought to be deficient in the Y and Z carrier proteins, although this is not proven, and also in the enzyme UDP glucuronyl transferase. After birth an exponential increase occurs and adult levels are attained by about 14 weeks. This rapid rise may be triggered by the disappearance of intrauterine suppressants (Roberton 1992).
- Newborn infants have the substance beta glucuronidase present in the gut from fetal life. This acts to deconjugate the conjugated bilirubin, which is unstable and readily changed, allowing it to be reabsorbed into the enterohepatic circulation and excreted via the placenta and maternal liver. Unfortunately, it continues to act in the neonate!

Physiological jaundice appears during the second day of life and reaches a peak by about 3–7 days. The infant will have visible jaundice when the bilirubin levels reach 80 µmol (Kelnar *et al.* 1995).

Other causes of jaundice

Whilst physiological jaundice is a naturally occurring phenomenon, jaundice does occur for other reasons, often the result of various disease processes. Table 8.1 summarises these, and some of the more common ones are discussed in more detail.

Rhesus incompatibility

Jaundice due to rhesus incompatibility usually manifests within 24 hours of birth, since the underlying cause was present in utero. If a woman has rhesus negative blood and her partner has rhesus positive they may have a baby who is rhesus positive (Figure 8.2). During the first pregnancy this does not usually cause problems. At delivery approximately 5 ml of fetal blood leaks into the maternal circulation, triggering the production of an antibody response. If no action is taken to prevent this, in subsequent pregnancies the normal small amount of fetal cells that leak during the

***Table 8.1* Causes of jaundice**

Example	*Mechanism*
G6PD deficiency ABO/Rhesus incompatibility Haemoglobinopathies	Increased red cell breakdown
Trauma	Sequestered blood
Physiological	Temporary reduction in efficiency of normal process
Breast milk	? Pregnanediol ? Free fatty acids Low fluid intake
Hypothyroidism Galactosaemia	Interfere with metabolism and excretion
Drugs	Need albumin binding sites
Infection	May affect liver +/– increase haemolysis
Biliary atresia Cystic fibrosis Tumours	Impair ability to excrete the conjugated bilirubin

pregnancy (0.1–0.2 ml) results in the secondary antibody response being evoked. With the fetus still in utero the antibody sets to work destroying all rhesus positive cells. In some instances this can lead to significant anaemia and heart failure, characteristics of a condition known as hydrops fetalis which can ultimately lead to death. Because of this it is essential to prevent the initial antibody production. This is achieved by administering anti-D immunoglobulin to the mother within 72 hours of delivery, or any event that may cause sensitisation to occur, for example amniocentesis, miscarriage or termination of pregnancy. The anti-D acts to 'hide' the rhesus positive cells allowing natural cell death to occur before the woman has the opportunity to initiate an antibody response. In 1969 a policy of administration of anti-D immunoglobulin to rhesus negative women following the birth of a rhesus positive baby and after any sensitising event in pregnancy was introduced. In the intervening 30 years perinatal deaths due to rhesus alloimmunisation have fallen a hundred-fold (RCGOP 1997). However, maternal sensitisation is still reported to be 1–2 per cent (Letsky 1994). Some writers have suggested that a programme of prophylactic administration of anti-D immunoglobulin to both primigravidae and multigravidae could reduce this even further and offer a cost-effective treatment in the future (Raafat and Urbaniak 1996; Mayne *et al.* 1997). This may not happen fully until a synthetic monoclonal anti-D preparation is available as opposed to the less predictable supply of polyclonal anti-D from immunised donors (RCGOP 1997).

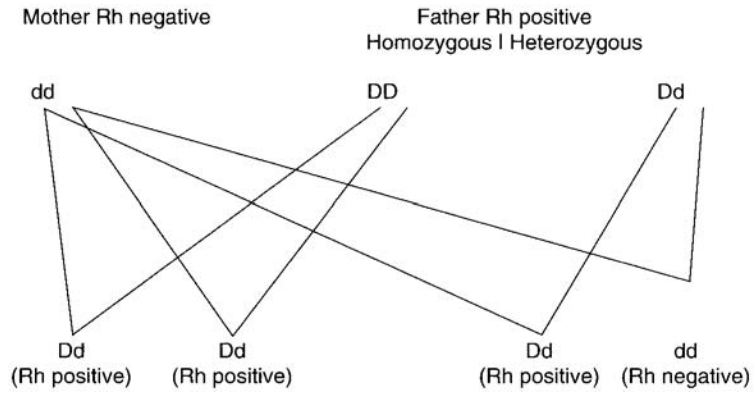

Figure 8.2 **Rhesus incompatibility: potential inheritance**

ABO incompatibility

Like rhesus incompatibility, jaundice due to blood group ABO incompatibility usually presents within 24 hours of birth. The jaundice is likely to be less severe than rhesus incompatibility. The classic picture is a mother with blood group O and a baby who is group A or B. The mother has naturally occurring anti-A or anti-B antibodies or lysins in her blood which destroy the red cells in the fetus leading to jaundice. The chance of ABO incompatibility is 1:5 with severe ABO haemolytic disease reported as being 1:3000 births (Letsky and de Silva 1994). One benefit of ABO incompatibility is that it actually confers some protection against rhesus alloimmunisation since the mother's naturally occurring anti-bodies induce haemolysis before the 'foreign' rhesus positive cell is detected.

G6PD deficiency

Glucose-6 phosphate dehydrogenase is an enzyme which is responsible for the integrity of the red cell membrane. A deficiency renders the cell liable to haemolyse. This happens especially when certain drugs, for example sulphonamides, have been taken, specific foods ingested, for example Fava beans (broad beans), or during periods of infection. The baby is usually symptom-free until oxidant stresses from an infection or drug triggers haemolysis. However, spontaneous haemolysis with no recognised exogenous trigger has been noted. This condition has a sex-linked recessive inheritance pattern which means that females are usually carriers whilst males are usually affected. Once diagnosis is established the family will need advice on appropriate diet and drugs for the infant. They may also need to be referred for genetic counselling. Obviously all of this will involve a multidisciplinary team approach with dietitians and specialist nurses being involved. The neonatal nurse plays a vital role in ensuring effective communication between the family and all health care personnel.

Breast feeding

There appear to be two different processes working to cause jaundice in the breast feeding baby.

- Breast feeding jaundice—the exact mechanism for this is uncertain but it is postulated that low fluid intake during the period of colostrum production and decreased caloric intake play a part. Also the infant initially has a slower intestinal transit time which increases exposure to beta-glucuronidase and thus increases the unconjugation process. Some studies have suggested a direct relationship between the amount of weight lost by the early breast feeding infant and the degree of jaundice (Merenstein and Gardner 1998).
- Breast milk jaundice—this is a later onset jaundice and again the exact onset is unknown. A number of factors are still being considered, including the role played by lipoprotein lipase, beta-glucuronidase and free fatty acids (Schwoebel and Sakraida 1997). An inhibitory substance was also once postulated, resulting from pregnanediol in the breast milk but this has not been consistently proved. Whatever the cause, breast milk jaundice is considered to be generally harmless, due in part to its later onset, usually peaking during the second/third week of life.

Due to its unknown aetiology the management of breast milk jaundice can be controversial. Withholding breast milk for 2–3 days usually causes the bilirubin levels to decline and resumption does not usually lead to high rebound levels occurring. However, this may suggest that the mother has 'caused' the jaundice to occur and because of the delicate balance of neurohormonal mechanisms in breast feeding this would need to be very sensitively handled by the neonatal nurse/midwife. The mother must not be made to feel that her breast milk is 'bad for the baby'.

Management of jaundice

Visible jaundice occurs once bilirubin levels reach 80 µmol, although visual observation is a subjective assessment. Hey (1995) believes that an experienced practitioner can accurately assess the level of serum bilirubin by observing the infant's colour. The use of devices such as a transcutaneous jaundice meter described by Ruchala *et al.* (1996) could provide for a non-invasive method of assessing the need for treatment. These techniques do, however, have limitations with non-Caucasian babies or inexperienced staff. It is important to remember that the classically described yellow discoloration of the skin may not be visible so easily in black or Asian babies and extra vigilance is needed to ensure that those skin tone changes that do occur are detected along with any yellow conjunctivae. Laboratory testing generally remains the method of choice.

Once the level of jaundice is ascertained the issue of when to treat it has been and remains a controversial question. Some writers (Dodd 1993) suggest commencing phototherapy when the bilirubin levels reach 340–350 µmol in a term infant with proportionately lower levels in the preterm infant. All seem to agree that treatment levels depend upon the infant's gestation, postnatal age and general clinical condition. The move to initiate treatment at higher serum bilirubin levels has been welcomed by some (Dodd 1993), but others (Seidman *et al.* 1997) sound a warning that it is still unclear at what threshold bilirubin toxicity occurs and that the risk factors related to the occurrence of kernicterus are not fully identified. Meisels and Newman (1995) report on an apparent increase in the incidence of kernicterus in the USA. Although the reasons for this are unclear, early discharge with less than optimal monitoring and undiagnosed G6PD deficiency are among the 'hidden risks' being considered (MacDonald 1995). Whatever the reason for this increase, it certainly highlights the point that jaundice is not to be under-estimated in the newborn—it is not yet a problem that we have fully solved.

Investigation of jaundice

It is likely that any investigations will include:

Jaundice in the first 24 hours
Serum bilirubin levels
Full blood count
Blood group and Coombs' test
Maternal blood group
G6PD
Galactose-1-PO$_4$ uridyl transferase (to detect galactosaemia)
TORCH screen (to detect *T*oxoplasmosis, *R*ubella, *C*ytomegalovirus and *H*erpes)

Jaundice appearing at 2–5 days
As above plus:
Blood cultures
Urine metabolic screening
Split bilirubin (to differentiate between conjugated and unconjugated bilirubin levels which may help to diagnose obstruction in the biliary tree)

Prolonged jaundice (longer than 14 days)
As above plus:
Liver function tests
Thyroid function tests

As with many conditions in the neonate, the management of jaundice depends upon the underlying pathophysiology and the severity of the hyperbilirubinaemia. The aim of any treatment is to prevent serum bilirubin levels from rising to such a degree that they will cause kernicterus (bilirubin encephalopathy) to develop. For less severe jaundice a number of therapies are available.

Phenobarbitone

Administering phenobarbitone to the mother from 32 weeks of pregnancy can be effective in reducing jaundice in the baby. The drug acts by inducing microsomal enzymes, including glucuronyl transferase, and also increases the level of ligandin. However, because many of the causes of jaundice are not predictable its use in this way is limited. When given postnatally to the baby, serum bilirubin levels will be reduced but it also results in a very sleepy, poorly feeding baby. Because of these factors other, more appropriate techniques are used.

Intravenous albumin

One of the first steps in the metabolism of bilirubin is that it binds with albumin. There is great competition for these albumin-binding sites from such things as drugs and free fatty acids. It seems logical therefore to administer albumin to increase the available number of sites. Initially conventional measurement of the serum bilirubin levels will show a rise, but because it is bound to albumin it is not so toxic and the infant is safer.

Metalloporphyrins

Recently published clinical trials are suggesting the administration of metalloporphyrins to the infant soon after delivery. These are compounds that inhibit haemoglobin oxygenase. As this is the first enzyme in the catabolism of haemoglobin, limiting its availability reduces the production of bilirubin. Reports on the use and value of the compounds vary, but some suggest that tin mesoporphyrin (SnMP) administered intramuscularly soon after birth is as effective as phototherapy in controlling non-specific hyperbilirubinaemia (Steffensrud 1998). Minimal side-effects have been reported, particularly an erythematous rash which is transient and appears in those infants who go on to need phototherapy after administration of SnMP. Ongoing studies are needed in order fully to evaluate the potential of these compounds, and since their use in infants of less than 30 weeks' gestation is uncertain the therapy may have little impact on the NICU population. However, it remains an exciting development for the treatment of otherwise well infants which could reduce the need for hospitalisation and treatment with potentially harmful interventions.

Phototherapy

The influence of light on jaundice has been recognised for over 30 years (Cremer *et al.* 1958). The discovery of phototherapy is attributed variously to either a chance nursing observation that infants exposed to sunlight had less jaundice, or that samples left on a laboratory shelf over the lunch break were noted to be affected by the light. Whichever is true—and it is probable that both are—it is remarkable that from such a serendipitous observation (Merenstein and Gardner 1998) should evolve a powerful and universally used therapy. Indeed, over the past three decades phototherapy has become the most commonly used treatment for jaundice.

Bilirubin absorbs light at 450 nanometers (nm), which is light at the blue end of the spectrum. When it is exposed to the light the molecule becomes excited and a number of processes occur to change it:

- Photo-oxidation—in which the light energy is transferred to oxygen and water-soluble products are formed. Only a small fraction of the bilirubin load is excreted in this way.
- Photoisomerisation—in which more water-soluble isomers are produced and also a substance called lumirubin (also known as photobilirubin) (Robertson 1993) which is a stable structural isomer and is excreted in bile and urine. The isomerisation process is possibly the most important in terms of bilirubin elimination.

The result of these processes is that bilirubin is transformed into less lipophilic pigments which can be excreted without the need for conjugation by the liver.

Traditionally phototherapy is administered via overhead strip lights. Because the blue light can mask cyanosis white fluorescent tubes are commonly used. The number of bulbs and the distance from the infant dictate the effectiveness of the light. Sixteen to eighteen inches is recommended as an optimal distance (Schwoebel and Sakraida 1997) and it has also been demonstrated that halving the distance from the infant doubles the amount of light being received (Hey 1995). Therefore lights should be located as close as possible to the infant without risking overheating. It should be remembered that lamps lose their effectiveness over time and so a record of their use should be maintained and manufacturer recommendations for changing the bulbs adhered to.

Phototherapy is often described as having a few not serious side-effects (Kelnar *et al.* 1995). However, there are some important nursing considerations which arise because of them.

EYE CARE Early animal studies suggested that retinal damage may occur as a result of exposure to the light (Noell *et al.* 1966; Messner *et al.* 1975). Although this was originally studied over 40 years ago it has not been conclusively established that this might also occur in humans and thus it obviously remains an important issue for carers. The infant's eyes are therefore covered with some form of protective shield. It is important that

the shields are removed at regular intervals to ensure that abrasions and infections are not being caused. It is also important that the shields are tight enough so that they cannot slip down to obstruct the airway whilst not being so tight that they increase the risk of intraventricular haemorrhage. Commercially available coloured Plexiglas shields are available which stand across the infant's head. They do not actually touch the face and thus help to prevent the complications associated with eye shields. Whichever shields are used, parents should be fully informed about the need for and use of the device.

SKIN CARE One of the effects of lumirubin excretion is that the infant has loose dark green stools. Because of this some form of nappy is desirable whilst maximising skin exposure to the lights. The use of a surgeon's mask tied on as a 'bikini bottom' is useful as a mini nappy and also ensures that the gonads are protected from chromatic radiance damage (Schwoebel and Sakraida 1997), which has been linked with sterility and genetic damage (Edwards 1995). The use of lotions, creams and oils is not recommended since the action of the light upon them may cause burns.

FLUID BALANCE There is no real consensus about the use of increased fluids during phototherapy. Increases in insensible water loss through the skin and the loose stools are reported as being 40 per cent in term infants and 80 per cent in preterm infants (Cloherty 1991). To counteract these losses some writers advise extra fluids at 30 ml/kg per 24 hours (Kelnar *et al*. 1995). Others suggest an increase of 25 per cent of daily fluid requirements (Blackburn 1995) while others recommend 1 ml/kg per hour (Fleming *et al*. 1991).

Equally, some writers recommend not routinely increasing fluids but instead measuring urine specific gravity and using that as an indicator of the need for extra intake (Merenstein and Gardner 1998). It would seem wise to consider the general status of the infant in relation to urine output, weight gain and electrolyte balance before considering administering extra fluids since the hazards of fluid overload are well documented.

THERMOREGULATION Because the infant receiving phototherapy should be nursed naked close monitoring of the temperature should be undertaken. The action of the light on the Perspex incubator or cot lid may increase environmental temperature, whilst small babies may need a heat shield to prevent heat loss.

RASHES Rashes have been reported which have been attributed in part to local histamine release (Modi 1991). Tanning also may occur as a result of increased melanin production, especially in black infants (Merenstein and Gardner 1998).

PARENTS Because so many infants receive phototherapy it may be easy for nurses to become blasé about its effect upon parents. It can be distressing for parents to see their baby nursed naked under the bright lights with eyes covered by shields.

Equally, because physiological jaundice does not appear in the first few days of life the infant may seem to be on the road to recovery when the jaundice manifests, compounding the belief that it is a deterioration or setback. The notion that the baby is sick can remain for some time and there is evidence to suggest that even after discharge home the parents still perceive the baby to be more 'vulnerable' (Kemper *et al*. 1989). Neonatal nurses must provide accurate information about the specific cause of the jaundice, the action of phototherapy and the long-term care needs.

Alternative methods of administration of phototherapy

Apart from the conventional bank of fluorescent bulbs, there are other methods available to deliver phototherapy.

PHOTOTHERAPY BLANKET Fibreoptic filaments, which carry a high intensity halogen light source, are woven into a pad which is either wrapped around the infant or which the infant lies on. The systems that use this method appear to provide comparable irradiance and effectiveness with standard phototherapy (Rosenfield 1990). Perhaps the biggest advantage of this form of phototherapy is that it eliminates the need for eye coverings and, since the infant does not need to be naked, it is easier for the parents to care for the baby.

HALOGEN SPOTLIGHT These produce an intense beam which can be used in isolation or as part of a conventional system. However, some writers (Hey 1995) suggest that the narrow beam which generates heat may be no more effective than the more conventional bank of lights.

Continuous vs intermittent?

The debate about whether to use continuous light or intermittent exposure is controversial. Intermittent exposure is reported to be as effective as continuous, since the action of the light is in the outer 2 mm of the skin. Recommendations about how 'intermittent' the exposure should be vary from 15 minutes on and 60 minutes off, to 1 hour in 4 (Dodd 1993). Reducing exposure to the lights does, of course, limit the majority of the side-effects. However, some cell damage may occur when the baby is exposed to phototherapy and in particular to intermittent exposure. This is because DNA strands break in the light and repair during periods of darkness.

Exchange transfusion

In cases where serum bilirubin levels are considered to be too high for phototherapy to be safely used exchange transfusion may be required. It should

be remembered that the technique may, of course, be performed for other conditions, such as severe infection.

Since the advent of phototherapy as a treatment for jaundice, and the introduction of anti-D immunoglobulin to prevent rhesus isoimmunisation, exchange transfusion has become an unusual procedure. This is perhaps fortunate since the technique is not without risk. A mortality rate of 0.1–0.5 per cent is suggested (Robertson 1993), although of course it could be argued that the more uncommon a procedure becomes the less skilled the staff become and the more likely it is that mortality and morbidity will increase.

The procedure entails removing blood from the infant's circulation in 5–20 ml aliquots depending upon the size of the infant, and transfusing the same volume of donor blood. Removing an amount of blood prior to transfusing the same amount of donor blood avoids causing fluid overload. This will gradually remove any bilirubin or toxins. A double volume exchange in which twice the infant's circulating volume is exchanged will give an 87 per cent replacement of the neonate's erythrocytes compared with a 45 per cent decrease in pre-exchange bilirubin levels (Schwoebel and Sakraida 1997). Because of this, multiple exchanges may be necessary.

The actual serum bilirubin level at which an exchange transfusion should be performed may vary from unit to unit but all should consider not only the actual SBR level but also the rate of rise of the concentration and the infant's age and condition.

Technique

Prior to undertaking an exchange transfusion parental consent is needed and should be obtained by the doctor. Since blood is to be transfused some parents may have religious objections, which they should have the opportunity to discuss fully with the nursing and medical staff. The nurse should be fully aware of his or her role as advocate for the baby and parents in this situation.

The baby should be kept warm and quiet and the use of gentle restraints may be necessary. Equipment for inserting an umbilical catheter should be available if one is not in situ. Other equipment includes an exchange transfusion pack, blood warmer and equipment to monitor blood pressure, temperature and ECG. The infant's stomach contents are aspirated and the nasogastric tube left in place. The umbilical vessels are cannulated using one of two routes:

- The umbilical vein only—the required amount of blood is firstly removed from the baby in 5–20 ml aliquots (depending upon age and size), discarded into the waste bag and the same amount of warm fresh donor blood is then administered via a four-way tap.
- The umbilical artery and vein are cannulated (or peripheral vessels may be used or a combination of both) and the blood is removed in aliquots via the artery and replaced via continuous infusion into the venous circulation.

This method is reported to carry less risk of fluctuations in blood pressure and less disruption to blood flow to the organs, so theoretically it should be less likely to cause intraventricular haemorrhage or necrotising enterocolitis (Todd 1995).

The whole procedure should take between 1 and 3 hours depending upon which method is used. Pain relief or sedation may be necessary to maintain infant quiescence and comfort. At some point during the exchange blood samples will be taken for monitoring electrolytes, calcium, glucose and SBR levels. In addition to checking the infant's temperature the nurse should observe the cardiac monitor for signs of arrhythmias which are associated with hypocalcaemia and hyperkalaemia. Problems can occur rapidly and skilled nursing observation along with thorough documentation are essential to ensure that small changes in the infant's condition are noted and reported accurately. Phototherapy will be recommenced after the exchange is completed

Kernicterus (bilirubin encephalopathy)

The reason to treat high levels of unconjugated bilirubin is its ability to cross the so-called blood-brain barrier and cause kernicterus. In this condition the bilirubin enters the basal ganglia and cerebellum and stains the nuclei of the cell yellow, which disrupts cellular metabolism. The unconjugated bilirubin uncouples oxidative phosphorylation and reduces protein synthesis in the mitochondria. It is said to be fatal in 75 per cent of cases (Swanwick 1989). The infant with kernicterus becomes lethargic and then goes on to become hypertonic with a high pitched cry. Opisthotonus and 'sun setting' eyes might be evident as may convulsions and respiratory disorders. A period of apparent recovery is followed by developmental delay, sensorineural deafness and extrapyramidal cerebral palsy (Roberton 1992).

Haemorrhagic disease of the newborn/vitamin K deficient bleeding

Haemorrhagic disease of the newborn (HDN) has been recognised as an important bleeding disorder for over 100 years, but it was not until the 1940s that the role played by vitamin K in the prevention of the disease was fully recognised (Passmore and McNinch 1995). During the 1990s the title HDN has been used less and less and the new term vitamin K deficient bleeding (VKDB) has been adopted. This new name clearly identifies the condition with vitamin K clotting disorders but also establishes the concept that it is not solely a disease of newborns

To understand the processes involved in VKDB it is important to understand the physiology of blood clotting.

Coagulation

When blood vessel injury occurs platelets adhere to the damaged area and release adenosine diphosphate (ADP) which activates the process of clotting. The platelets are pulled together because they have an affinity for the blood protein fibrinogen and together they form a mesh over the tear. In order to prevent the mesh from being torn away the platelet prostaglandin pathway releases thromboxane, which encourages vasoconstriction and decreased blood flow in the damaged area. A process known as the clotting cascade is then initiated (see Table 8.2) in which a series of proteins and enzymes are activated sequentially and, through a variety of complex biochemical reactions, will produce a blood clot. Other proteins work to regulate the process and ensure that clotting does not occur in the systemic circulation with potentially disastrous results. A number of the proteins/enzymes—'clotting factors'—are vitamin K dependent and need the vitamin to become functional. The dependent factors are Factor II, VII, IX and X. The vitamin K promotes carboxylation and after this process has occurred the calcium binding proteins in the clotting process can be activated.

***Table 8.2* Blood clotting factors**

Factor I	Fibrinogen
Factor II	Prothrombin*
Factor III	Thromboplastin
Factor IV	Calcium ions
Factor V	Pro-accelerin
Factor VI	Is no longer recognised in the clotting pathway
Factor VII	Pro-convertin* (Serum prothrombin conversion accelerator)
Factor VIII	Antihaemophilic factor
Factor IX	Plasma thromboplastin component* (Christmas factor)
Factor X	Thrombokinase* (Stuart–Power factor)
Factor XI	Plasma thromboplastin antecedent
Factor XII	Hageman factor
Factor XIII	Fibrin stabilising factor

* Vitamin K dependent

Sources of vitamin K

In the newborn, levels of the vitamin K dependent clotting factors are low compared to older infants and they develop slowly. Vitamin K is obtained naturally from plants (vitamin K1—phylloquinone) and bacteria (vitamin K2 —menaquinone). The main source of vitamin K1 is the most important in newborns. The absorption of bacterial vitamin K2 is reported by some writers (Kelnar *et al.* 1995) but disputed by others (Passmore and McNinch 1995). Unfortunately, the placental transfer of K1 is low, with a maternal: fetal ratio of 4:1 so the infant is dependent on dietary sources. Formulae milks contain on average 50 µg/l of vitamin K. Breast milk on the other hand has about 2 µg (Passmore and McNinch 1995). Equally, if the bacterially produced K2 actually is absorbed as some suggest, the breast fed baby is also at a further disadvantage due to the different types of colonisation as this process takes longer than in the formulae fed infant. This difficulty in acquiring vitamin K coupled with a normal postnatal drop in the levels of Factor II, VII, IX and X means that the newborn, especially the breastfeeding newborn, is at risk from haemorrhagic disease of the newborn/vitamin K deficient bleeding.

Characteristics of vitamin K deficient bleeding

There are three forms of this disease:

- Early onset—presents within the first 24 hours and is strongly linked with the maternal intake of anticonvulsant therapy. It may manifest as cord stump haemorrhage and puncture site bleeding.
- Classical onset—presents between 2–7 days and is possibly linked with delayed onset of feeding. It may manifest as GI bleeding and cephalhaematoma.
- Late onset—occurs between week 1 and week 8. It may manifest as prolonged jaundice, bleeding or spontaneous bruising.

Although it is a rare condition it carries a high mortality/morbidity rate: 30–50 per cent of babies with late onset VKDB will have significant brain damage (Kelnar *et al.* 1995).

Vitamin K prophylaxis

The use of artificial vitamin K to prevent VKDB has been common practice since the 1950s. Initially Konakion (or its predecessor Synkavit) was given intramuscularly but no consensus was reached about to whom it should be administered. Some maternity units gave it to all babies, others gave it only to those considered to be 'at risk'. Intramuscular administration continued until the 1980s when the oral route became more fashionable. This was even

though there was no oral preparation available and employing this route necessitated using the intramuscular preparation. In 1992 Golding *et al.* published a study purporting to link childhood cancer with the use of vitamin K, especially with parenteral rather than oral administration. This widely publicised work led to a great furore and obviously gave impetus to the search for a licensed oral preparation to be widely available. In 1992 national guidelines were published by the Department of Health, which recommended the use of IM or oral vitamin K. These were updated in 1998 and affirmed that all newborn babies should receive vitamin K. Later studies from the UK, America and Europe (Klebanoff *et al.* 1993; Ansell *et al.* 1996) have failed to identify a significant increased risk of childhood cancer and paediatricians continue to advocate the need for vitamin K to be given. Many units ask parents to give written consent for the drug to be given, although this may be more complex if the infant is sick or preterm. Since licensed oral preparations are now available parents can be more fully informed about the advantages/disadvantages and medical staff can make choices of therapy based on both routes being available.

Disseminated intravascular coagulation (DIC)

An understanding of the mechanisms of blood clotting is also important in underpinning knowledge when considering the infant with another serious bleeding disorder—Disseminated intravascular coagulation (DIC). This is a disease which affects infants who are already ill and as such is a secondary coagulation disorder which is characterised by a cycle of bleeding and clotting (Kuehl 1997). The underlying pathophysiology of the primary condition leads to the triggering of the clotting mechanism, which results in the inappropriate systemic production of diffuse intravascular thrombus formation. This in turn leads to increased consumption of platelets and plasma clotting factors causing haemorrhages to occur (Emery 1992). The increased clot formation also results in fibrin degradation products being released by the activation of thrombin and fibrinolysis occurring. This triggers an increase in the overall amounts of circulating anticoagulant, leading to further haemorrhages and prolonging the cycle of DIC (Kuehl 1997) (Figure 8.3).

The incidence of DIC is difficult to establish since it is a secondary disease process, but mortality rates are reported as 60–80 per cent (Kenner *et al.* 1993).

Signs of DIC

The clinical manifestation of DIC may be very similar to that of vitamin K deficient bleeding. The major difference is that the infant with VKDB will generally appear well whilst the infant with DIC is already ill when the signs of haemorrhage occur. The haemorrhages can appear at any site, including oozing from puncture sites, ecchymoses, cord stump haemorrhage and

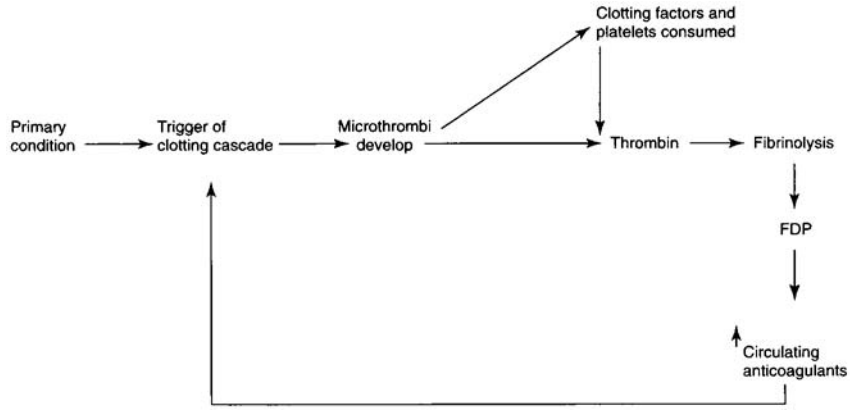

***Figure 8.3* Disseminated intravascular coagulation**

petechiae. Because of this random location cerebral haemorrhage may also manifest. The other process in DIC—the disordered clotting mechanisms—will also produce signs which include skin necrosis, oilguria and renal failure. A diagnosis is made when the platelet count is low, partial thromboplastin (PTT) and prothrombin (PT) times are prolonged and fibrin degradation products are elevated.

Management

One of the primary aims of DIC management must be to correct the underlying pathophysiology of the original triggering condition. Concurrent treatment to control the haemorrhage/clotting cycle will be given. Exchange transfusions, platelet transfusions, cryoprecipitate and fresh frozen plasma (FFP) all have a role to play. Exchange transfusions may be used to 'wash out' any toxins in the infant's blood and to replace clotting factors. Cryoprecipitate increases Factor VIII and fibrinogen levels. FFP can increase coagulation factors by 15–20 per cent and a platelet infusion of 10 ml/kg can increase the platelet count by up to 100000/mm^3 (Emery 1992; Kenner *et al*. 1993; Kuehl 1997). Skilled nursing care is essential to support the infant and to ensure close observation of any changes in condition. If these are recognised early enough it provides the baby with the best chance of survival. Obviously parental support is also a priority. Because the baby will already be sick, this profound and potentially life-threatening complication may well be difficult to deal with and it is likely that the parents will look to the neonatal nurse for information and reassurance.

Anaemia

Anaemia is a significant reduction in the concentration of haemoglobin in the blood. It can be defined as a haemoglobin value greater than two standard

deviations below the mean for postnatal age (Johnson *et al.* 1998). Apart from pathological causes, preterm infants can also suffer from anaemia of prematurity. This is a more pronounced form of the normal physiological process where a normocytic, normochromic anaemia occurs at around 2–4 months. Preterm infants experience a faster drop in Hb at around 2–8 weeks and the drop in Hb level may also be more significant at 7–10 g/dl. It occurs due to a transient developmental abnormality of erythropoietin. This glycoprotein, which is produced in the kidney, serves to regulate red cell production. In the fetus it is produced in the liver from 5 weeks' gestation. The move to renal production occurs during the last trimester and so in preterm infants the liver remains the major site for several weeks. Because the erythropoietin production is affected by tissue oxygen needs having the production site in the liver results in a less than optimal trigger since the hepatic response to hypoxia is not so efficient as the renal response (Downey 1997).

Anaemia in the newborn infant may also occur as a result of physiological processes or due to more serious blood loss. The blood loss may occur antenatally as a result of placental abruption, placenta praevia and twin-to-twin transfusion. Feto-maternal transfusion may also occur and Kelnar *et al.* (1995) suggest that this happens in up to 50 per cent of pregnancies with the fetus losing up to 100 ml of blood. A Kleihauer test will detect fetal cells in the maternal circulation. A sample of blood is taken from the mother and because fetal cells do not denature with alkali, after fixing, the fetal cells retain the red colour of haemoglobin. The adult haemoglobin becomes eluted and the adult cells become pale. These pale cells are referred to as ghost cells against which the fetal red cells can be easily seen and counted. If 10 fetal cells are seen in 30 fields it is equivalent to 1 ml of fetal blood (Johnson *et al.* 1998). This means that the size of the feto-maternal transfusion can be affected. In a twin-to-twin transfusion the affected twin is on the arterial side of a placental vascular malformation which allows blood from one twin to move across to the other twin. The degree of anaemia will depend upon the duration and extent of the haemorrhage.

Perinatally bleeding may occur as a result of trauma, for example, during a cesarean section when the cord or placenta may be incised. This is a rare event and a more common birth trauma is cephalhaematoma, which is bleeding under the periosteum on the skull as a result of contact with the hard structures of the maternal pelvis.

Postnatally the causes of neonatal bleeding are many and various. Apart from vitamin K deficient bleeding (discussed elsewhere), they include intracranial and subaponeurotic haemorrhage, pulmonary haemorrhage, failure of the cord clamp and gastrointestinal bleeds. With the latter it is important to differentiate between maternal blood which the infant may swallow during the delivery and the infant's own blood. Performing an APTS test can do this. The same alkali resistance that is the basis of the Kleihauer test is used here. A sample of the blood is diluted with water and 1 per cent sodium hydroxide (NaOH) is added. The alkaline solution denatures any adult haemoglobin that will become yellow but the fetal haemoglobin will remain pink.

Acute anaemia may present with tachycardia, hypovolaemia, hypotension, tachypnoea and shock. A more chronic onset may present as lethargy, pallor, tachypnoea and tachycardia. The neonatal nurse, who is often the primary caregiver during this time, may be the first person to notice the insidious onset. The nurse also plays a key role in monitoring blood sampling in order to prevent iatrogenic anaemia due to repeated investigations.

In order to treat anaemia the underlying cause is first established and treated. More generic treatment then includes exchange transfusion, repeated 'top-up' transfusion and iron supplementation.

Top-up transfusions

Because top-up transfusions are a common aspect of neonatal care it may be the case that they are under-estimated in terms of potential side-effects. These include:

- Circulatory overload—which ensues from either administering blood to an infant with normal intravascular volume, or administering a transfusion too rapidly.
- Haemolysis—following transfusion mismatch with donor/recipient ABO and rhesus group.
- Graft versus Host Disease (GVHD)—which is a potentially fatal incompatibility between recipient and donor cells. This rare condition is said to be more likely when the recipient receives blood from multiple donors (McCormack 1998) and the use of smaller packs of blood produced from one donor may help to prevent this.
- Febrile reactions—which usually occur within the first 30 minutes of the transfusion commencing. An immune response to the donor white cells may be responsible for this.
- Metabolic complications—which include hypokalaemia, acidosis and hypoglycaemia, are most likely to be associated with the more significant procedure of exchange transfusion.
- Bacterial/viral contamination—should be a rare event since blood is screened for most significant organisms (for example, hepatitis B and C, HIV, cytomegalovirus). However, 'new' diseases will continue to occur and the Department of Health are currently paying close attention to new variant Creutzfeldt-Jakob disease and its possible transmission in blood and blood products. Although screening is comprehensive it may be unwise to be too complacent about the safety of blood.

In order to prevent or detect any possible side-effects the nurse needs to:

- Ensure that blood is administered with regard to strict infection control procedures.
- Ensure that all details are diligently checked and recorded to avoid incompatibilites.

- Ensure that the infant's baseline observations (pulse, blood pressure, temperature and respiratory rate) are recorded at the beginning of the transfusion and then at 15 minutes, 30 minutes and, if no reaction occurs, hourly until the procedure is completed.

Iron supplementation

The need for supplementary iron should be assessed according to the infant's gestation, postnatal age, weight and feeding regimen. Data suggest that preterm, especially very low birthweight, babies should receive iron from about 2 months of age (Ehrenkranz 1994) but there is some debate about how much should be given and for how long. Local policies and guidelines should be considered.

Recombinant erythropoietin

Erythropoietin is the glycoprotein that regulates the production rate of red blood cells. Rather than transfusing whole blood or packed cells for the treatment of anaemia of prematurity more recent studies are researching the efficacy and safety of recombinant human erythropoietin. This substance stimulates erythroid progenitor cells to produce red blood cells and is administered via the intravenous or subcutaneous route (Downey 1997). Although the treatment is not without possible side-effects and limitations, studies are suggesting that it is a potentially valuable new therapy (Shannon *et al.* 1995).

Conclusion

Many questions remain unanswered about the optimum treatment for haematological disorders. As primary caregivers, neonatal nurses have a duty to keep abreast of clinical developments which lead to the introduction of new therapies and to be ready to incorporate these therapies when planning appropriate nursing care.

Case study 1: infant with jaundice

Steven is a 33-week gestation baby who is now 54 hours old. He is being breast fed and is fixing and sucking well. During a feed his mother asks you if he 'looks yellow'.

Q.1. What is the most likely cause of Steven's jaundice?

Q.2. Give your rationale for excluding the other causes.

Q.3. What are the physiological processes happening to cause it?

Q.4. Outline the investigations that will need to be undertaken.

Case study 2: infant with vitamin K deficient bleeding

Rose has just been admitted to your neonatal unit. She is 29 weeks' gestation and is 20 minutes old. Her condition is stable. Her mother intends to breast feed.

Q.1. Prior to administering vitamin K, what information should you give the parents?

Q.2. What is the underlying pathophysiology in vitamin K deficient bleeding?

Q.3. What are the implications of the mother's choice to breast feed?

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chapter 9

Chapter 9

Pain and Comfort in Neonatal Intensive Care

Contents

Introduction

> *If the awareness of the problem of neonatal pain is heightened then it is hoped that more episodes of pain would be recognised, reported and treated.*
>
> *(Carter 1990:10)*

The aim of this chapter is to increase knowledge and understanding about neonatal pain in order for neonatal nurses to utilise this information and research evidence to ensure that the infants they are caring for receive a uniformly high standard of care. The background knowledge required by neonatal nurses and midwives in order to understand this chapter is experience in caring for sick newborn babies.

Pain in the newborn is a vast topic, and whilst encompassing infants receiving intensive, high dependency and special care, this chapter will focus on neonatal intensive care. It is hoped, however, that neonatal nurses will be able to utilise this information and knowledge and apply it to any category of infant that they are responsible for.

In 1986, Mersky described pain as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (Sparshott 1997:xviii). Pain in neonatal intensive care can be a major problem, not only for the neonate, in which it can have both short- and long-term consequences, but also for the neonatal nurse, in effectively assessing the infant's pain and acting appropriately to resolve it. The experiences that sick newborn and preterm infants suffer can be traumatic and there is concern about whether enough is done to relieve this suffering and if more can be done to provide comfort for these infants. Porter (1989) found that acutely ill neonates are subjected to between 50 and 132 bedside procedures every 24 hours. Many of these procedures involve pain. Nurses are required to 'act always in such a manner as to promote and safeguard the interests and well-being of patients [and to] ensure that no action or omission…is detrimental to the interests, condition or safety of patients' (UKCC 1996:9). Therefore they have a duty to minimise pain and discomfort.

In the past ten years there has been a considerable amount of research into neonatal pain, encompassing many different aspects, some of which will be discussed within this chapter. Although there is a deluge of research surrounding the issue of neonatal pain, attitudes have been slow to change, possibly due to inadequate knowledge, limited assessment tools, and failure to recognise responses to pain, often resulting in inadequate pain management of the vulnerable, dependent baby (Van Cleve *et al.* 1995).

All nurses caring for these infants require an awareness about the effects and consequences of pain, and how to assess and manage it in order to prevent any detrimental effects, to improve the well-being of both the infant and his or her parents. It is no longer possible to neglect this issue. Knowledge generated by research must be disseminated amongst professionals in order to further advance the care of the neonate and improve clinical practice.

Theory of pain

Until recently, it was generally accepted that neonates did not feel pain and therefore pain assessment and relief were not considered. In 1984 D'Apolito stated that sick newborn infants respond minimally to pain (cited in Lawrence *et al.* 1993:59). This idea has now been discredited by research which indicates that neonates perceive, experience and remember pain (Anand *et al.* 1987). Owens (1984) highlighted the factors contributing to the mismanagement of neonatal pain when he stated 'for years, researchers and clinicians have made assumptions about whether infants experience pain based on casual observations or theoretical indifference' (Owens 1984:213). Other research has shown that premature infants not only experience pain, but are hypersensitive and have a lower threshold for pain than adults (Fitzgerald *et al.* 1988).

It is now widely accepted that infants, including those in utero and those born preterm, have the neurological capacity to feel pain (Anand *et al.* 1987). In order to appreciate the issues surrounding neonatal pain it is helpful to have an understanding about the development of the central nervous system (CNS).

Bildner and Krechel (1996) suggest that pain in the neonate has been undertreated in the past due to inadequate knowledge of neonatal physiology and nociception. Nociception is the term used in relation to pain perception in neonates because 'pain is a sensation with strong emotional associations' (Anand *et al.* 1987:1321). It is the perception by the nerve centres of painful stimuli and is a process involving the peripheral nervous system, where pain signals are generated, the autonomous nervous system, which responds to these signals, and the central nervous system, where the information is processed. Nociceptors, which are modified neurones, receive and transmit information about pain. Transmission of a pain sensation is carried out by two specific types of nerve fibre: A delta fibres, which are myelinated and conduct impulses quickly, and C fibres, which are unmyelinated and conduct impulses slowly (Carter 1994; Sparshott 1997). However, there is a third type of fibre: A beta fibres, which have a large diameter, are myelinated and transmit impulses very quickly and these are thought to be the triggers for the gate control theory (see below). A summary of the development of the structures needed for nociception to occur is described in Table 9.1.

The lack of myelination is frequently used to support the argument that the neonate cannot feel pain. However, even in the adult periphery, pain impulses are carried through unmyelinated fibres. Incomplete myelination implies that impulses are transmitted more slowly, but in the neonate this is offset by the fact they have shorter distances to travel (Clancy *et al.* 1992). Many pain mechanism theories exist, but the most widely accepted explanation of the mechanism of pain is the gate control theory, proposed by Melzack and Wall in 1965 (cited in Carter 1994:33). This suggests that the CNS has the ability to control the input of pain information by use of a 'gate'. When the 'gate' is open pain impulses can flow through but when the 'gate' is closed then no impulse can. Impulses from large fibres (A beta) close the gate but impulses from smaller ones such as A delta and C fibres can reopen the gate.

***Table 9.1* Summary of the development of the structures needed for nociception to occur**

Gestational age	*Developmental stage*
3–4 weeks	Primitive but functioning cerebral circulation
6 weeks	Development of synapses between sensory fibres and neurones in the spinal cord
7 weeks	Sensory nerve endings appear
8 weeks	Differentiation of the spinal grey matter
11 weeks	Sensory nerve endings have spread to the rest of the face, palms and soles of the feet
12–13 weeks	Hypothalamus and cortex connect. It is suggested that the fetus cannot feel pain before this time (Concar 1996)
15 weeks	Sensory nerve endings have spread to the trunk and pxominal parts of arms and legs
20 weeks	Sensory nerve endings have spread to all cutaneous and mucous surfaces
20–24 weeks	Cortex is functional and a stress response can be mounted
30 weeks	Nerve tracts concerned with pain in the brain stem and spinal cord are completely myelinated. Sensory impulses for pain are conducted along the spinal cord and brain pathways. Recognition of the pain is ultimately localised in the cerebral cortex

Source: Stevens and Franck 1995; Concar 1996

Research undertaken by Giannakoulopoulos *et al.* (1994) showed that the fetus can mount a hormonal stress response to an invasive procedure in utero. Observation of the fetus revealed vigorous fetal movements and increased heart rate in response to intrauterine needling. This suggests that the systems for nociception may develop in the fetus before the spinal and CNS centres are complete. It is evident therefore that the infant has all the anatomical structures necessary for the perception of painful stimuli, not only at birth but also before birth, and therefore it is important that this information is widely disseminated on the part of those concerned with the care of the neonate.

Infant response to pain and distress

A considerable amount of research has been published about the responses of the newborn to painful stimuli. Responses can be classified chemically, behaviourally and physiologically. The chemical response to pain is described by Anand *et al.* (1985), behavioural responses include facial

expression, crying and altered body movements (Grunau *et al.* 1990), while the physiological responses include changes in heart rate, blood pressure, respiratory rate, palmar sweating and oxygen saturation (Owens and Todt 1984).

If it is accepted that neonates can feel pain, then consideration must be given to how this pain can be recognised by the caregivers. Pain assessment in neonates is not only challenging but also extremely important as the appropriateness of treatment is dependent on an accurate assessment (Porter 1989). Research has suggested that each infant must be considered as an individual, each reacting to painful stimuli in different ways (Grunau *et al.* 1990). Responses vary depending on the gestational age, the physical condition and state of consciousness of the infant (Sparshott 1989). The neonate is completely dependent, non-verbal, and may be too ill or too premature to summon a response to a painful stimulus. The interpretation of pain experienced by the neonate may be influenced by the attitudes and beliefs of the carer, but by being aware of the different responses to pain, the carer will be able to recognise the signs and will therefore be able to assess pain more effectively.

Chemical responses

Most of the research concerning the chemical response to pain has been undertaken on neonates undergoing surgery with minimal anaesthesia. Anand *et al.* (1985) demonstrated that the term infant appears to respond chemically to a painful stimulus in a similar way to adults. Catecholamines, glucagon, growth hormone, cortisol, aldosterone and other corticosteroids are released resulting in suppression of insulin secretion which then leads to increased metabolism of fat stores and carbohydrates, and hyperglycaemia (Anand *et al.* 1987; Clancy *et al.* 1992). These stress effects may last for a longer period of time in preterm infants because they have limited stores of carbohydrates, protein and fat (Sparshott 1997). This may adversely affect such infants by causing metabolic instability, which is associated with increased morbidity and mortality (Givens-Bell 1994).

The utilisation of biochemical responses to assess pain in the neonate in clinical practice is not a practical option for the neonatal nurse as it involves analysis of blood samples, which are not only expensive and time-consuming, but the neonate will probably have to be subjected to additional pain in the collection of the sample. However, the nurse can observe for signs of metabolic change by monitoring the blood glucose, and testing the urine for glucose, ketones, protein and pH (Givens-Bell 1994).

Behavioural response

There is dispute in the literature about whether facial expression is an accurate sign of pain in the preterm infant. Johnston *et al.* (1993) suggest that facial activity can reflect pain in both preterm and term neonates whereas Carlson

et al. (1996) state that facial responses to pain have been demonstrated in term infants, but more research is required as to their significance in the preterm infant. Grunau and Craig (1987) examined facial expression and cry in response to discomfort (heel rub) and pain (heel lance) in well, term infants. They found that the response was dependent on the infant's sleep/wake states, of which there were four: quiet sleep, active sleep, quiet awake and active awake. They observed that there was a different facial response between those infants in a quiet sleep state and those in a quiet awake state (Grunau and Craig 1987). Grunau *et al.* (1990) also researched facial expression and crying in term neonates and found that both occurred in response to a painful procedure. Research undertaken by Craig *et al.* (1993) evaluated facial expression in reaction to a heel lance in both preterm and term infants and demonstrated that facial activity increases with gestational age. Grunau and Craig (1987) developed the neonatal facial coding system (NFCS) in order to assist professionals in recognising infant responses to pain. They described the following facial actions within this coding system: brow bulge, eye squeeze, nasolabial furrow, open lips, mouth stretch, lip purse, taut tongue and chin quiver. Tongue protrusion was added to this list in subsequent research by Grunau *et al.* (1990). Nurses in neonatal intensive care describe facial expressions as an indicator of pain for both preterm and term neonates (Shapiro 1993). When assessing pain in the neonate using facial expression, the sleep/wake state of the baby and the gestational age (Grunau and Craig 1987; Craig *et al.* 1993) must be considered.

Cry responses to pain are a frequently noted behavioural sign in both term and preterm neonates (Grunau *et al.* 1990), and are one of the few ways that neonates can signal that they are in pain. However, caution must be exercised with the ventilated baby to whom the only form of crying available is the 'silent cry', which involves gaping around the endotracheal tube (Sparshott 1989, 1996, 1997; Stevens and Franck 1995). Variations in the pitch and intensity of the cry are associated with the intensity of the pain (Porter 1989). Different cries have been recognised, each describing a different emotion: hunger, anger and pain (Carter 1990). The pain cry has been described as high pitched, shrill, intense and intended to attract attention from the caregiver (Clancy *et al.* 1992; Stevens *et al.* 1993; Lynam 1995; Van Cleve *et al.* 1995; Carlson *et al.* 1996). The cry of the term infant in pain may resemble that of the preterm or sick infant (Porter 1989), and therefore consideration must be given to the gestational age and condition of the baby. Owens (1984) suggests that the infant cry is a factor to be considered when using a behavioural assessment tool, but that it should not be used in isolation.

Body movement is another area that has been studied in relation to a painful event. Research has shown that infants respond to painful stimuli with diffuse body movements and will attempt to withdraw from the painful stimulus (Franck 1986): they may kick, thrash their legs and become rigid (Owens and Todt 1984). Some neonates will, however, become habitualised to the experience and may not respond to a second painful insult (Sparshott 1997), so this must be considered in any pain assessment. A premature infant

may not have the capacity to respond and may become limp or flaccid when subjected to pain (Franck 1993:917). Therefore a decrease or lack of body movement should not be misconstrued as an indication of absence of pain in the preterm neonate. Preterm neonates may be physically constrained by splints and equipment within the neonatal intensive care unit and therefore may not demonstrate body movements in relation to pain. Caution should be used in interpreting pain in the neonate by assessing body movements alone (see p. 19).

Physiological response

There are several parameters that may be measured to assess physiological responses to pain, including heart rate, blood pressure, respiratory rate, blood oxygenation, palmar sweating, vagal tone and intracranial pressure. However, changes in these parameters may occur for other reasons and it is important to determine this when assessing whether the neonate is in pain (Carter 1994; Carlson *et al.* 1996).

Heart rate is usually bi-directional: it increases at first and then decreases (Owens and Todt 1984; Anand *et al.* 1987) but bradycardia alone may occur in the 'fragile' infant (Sparshott 1996, 1997). Blood pressure increases in response to pain (Williamson and Williamson 1983; Owens and Todt 1984), as does respiratory rate (Field and Goldson 1984). Blood oxygenation usually decreases, but this may be due to the neonate crying and becoming agitated (Williamson and Williamson 1983). Palmar sweating is increased in infants following a heel lance (Harpin and Rutter 1982), although this may not occur in neonates of less than 36 weeks' gestation. Stevens *et al.* (1993) demonstrated, in their preliminary study, that neonates of 32–34 weeks' gestation exhibited similar responses to term infants in their physiological response to pain, and Craig *et al.* (1993) also showed that preterm neonates, from 25 weeks' gestation, demonstrated an increased heart and respiratory rate and a decreased blood oxygenation in response to a painful procedure.

A considerable number of responses to pain have been identified, none of these can be used in isolation, but they may be utilised in conjunction to assess the degree of pain in the neonate.

Assessment of pain in a non-verbal client group

The area of pain assessment is an area that has been neglected in the past, possibly due to a number of beliefs: that neonates could not feel pain due to lack of myelination; that newborn infants do not remember pain or if they do, it has no adverse effects; that responses to pain are not understood, and that it is dangerous to administer analgesia to newborn infants. These misconceptions are questionable and many have been disproved by research.

Nurses can provide comfort measures for neonates but they currently have to rely on their medical colleagues to prescribe analgesia. The nurse may be

the most appropriate person to assess the needs of the baby but she 'must have tools for defending her judgement in requesting medication' (Lawrence *et al.* 1993: 60). Pain assessment tools are important in the management of neonatal pain as they allow the neonatal nurse to make visual and written observations to substantiate her claim for pain relief for an infant. Previously the request may have been ignored because of the lack of evidence and misconceptions by health professionals. Sparshott (1989:64) asks, 'how can we claim that pain has any effect on a baby unless we show it to be so?' That nurses require evidence of pain in order to justify requesting pain relief for the neonate was demonstrated in the results of a survey by Franck (1987), when only 34 out of 76 nurses stated that medication was used when a nurse believed clinical signs indicated pain. This appears to indicate the confusion within this area and the need for written documentation to justify the need for pain relief.

Many neonatal units do not currently use a tool to assess neonatal pain and so the classification of pain in the neonate is variable, depending on each nurse's interpretation and feelings about pain and the nurse's own experience of pain. Research has been undertaken into nurses' perceptions of pain in the newborn (Carter 1990; Shapiro 1993; Bildner and Krechel 1996) and this would seem to suggest that more education is required in order to give a standardised approach to pain assessment. Franck's survey (1987) into nurses' attitudes about pain in neonatal intensive care also revealed that there was a lack of consistency in attitudes and practice. It may be that the use of an assessment tool would help to standardise care and ensure that the neonates receive adequate and appropriate pain relief. Franck (1993:921) suggests that any assessment criteria should be standardised, and stated 'nurses' abilities to compare treatment methods and establish standards for the care of neonates in pain will be severely limited without valid and reliable methods for assessing pain' (Franck 1987: 392). It is important to consider the practicality and feasibility of assessment tools and whether they are relevant for clinical practice. Time is of the essence in neonatal intensive care and if an assessment tool is found to be time-consuming or complicated it will not be used effectively or efficiently. If the assessment tool requires the use of expensive equipment it may be considered impractical for use. Ethical considerations must also be taken into account. The aim of pain assessment is to reduce pain and if the neonate is being subjected to more pain or distress by the actual assessment, then the tool requires modification. Neonatal nurses, in their role as advocate for these infants, have to consider benefit versus harm.

Pain assessment tools

Neonatal individual developmental care and assessment programme (NIDCAP)

NIDCAP was developed by Als *et al.* in 1986 (cited in Sparshott 1997) to assess infants' behaviour so that the health professional caring for the infant could provide more support and comfort. It is a complex system-

by-system approach, looking at body and extremity tone, flaccidity of facial muscles, body posture, breathing patterns, chest expansion and other signs of infant stress. It can be used to assess pain in the neonate and can be utilised to provide an appropriate physical environment, organise and time medical and nursing procedures with minimal disturbance to the infant, and enhance the parents' understanding of their baby as an individual. Tribotti and Stein (1992) evaluated the use of NIDCAP in neonatal intensive care and their findings suggest that it may be valuable in the prevention of short- and long-term effects of pain in premature neonates. However, caution should always be exercised when using it as a tool to assess extremely premature or sick neonates because they may have difficulty in displaying these behaviours due to the instability of their condition (Stevens and Franck 1995). It is made more complex to use in clinical practice because, in order to use NIDCAP, professionals have to undertake formal training and yearly updates, which has financial implications that must be considered before implementation can take place.

Postoperative pain score

Attia *et al.* (1987) devised an assessment tool to measure postoperative pain and the effectiveness of analgesia in infants of 1–7 months of age who had undergone minor surgery. The score is based on behaviour and has ten criteria: sleep, facial expression, quality of cry, spontaneous motor activity, responses to ambient stimulation, flexion of fingers and toes, sucking, evaluation of tone, consolability and sociability. It has been used in research to assess the effectiveness of analgesia by Mayer *et al.* in 1989 (cited in Franck 1993:917) and used by neonatal nurses in assessment of postoperative pain (Givens-Bell 1994).

Neonatal infant pain scale (NIPS)

NIPS is a tool which was devised by Lawrence *et al.* (1993) to assess the behaviour of preterm and full-term neonates in response to the pain of needle puncture. Their scale was based on previous research into children's pain. The operational definitions of the NIPS describe facial expression, cry, breathing patterns, movement of arms and legs and state of arousal. A score was given to these criteria, a low score indicating minimal or no pain and a high score indicating an infant in pain. NIPS is designed to be used before, during and after a painful procedure. It appears to be quick and easy to use and the neonate does not have to be disturbed. However, as with all pain scales, care must be taken if the neonate is very immature or ill as a low score may be achieved, indicating that the neonate is not in pain when the baby is actually too ill or immature to respond.

CRIES

CRIES was developed by Bildner and Krechel (1996). CRIES is an acronym for: Crying, Requires oxygen to maintain saturation greater than 95%, Increased vital signs, Expression and Sleeplessness. This tool therefore uses a scoring system based on the five signs—crying, oxygen saturation, vital signs, expression and sleep—a score of four or more indicating pain. It has been used successfully in neonatal intensive care for assessing pain in the postoperative period (Bildner and Krechel 1996). It was tested on neonates of over 32 weeks' gestation but its effectiveness for neonates of less than 32 weeks has not been established.

Distress scale for ventilated newborn infants (DSVNI)

Sparshott (1996) developed the DSVNI to identify pain and distress in ventilated newborn infants. The DSVNI takes into consideration physiological and behavioural signs in response to any invasive procedures. The behavioural responses include facial expression, body movement and colour. The physiological responses include changes in heart rate, blood pressure, oxygenation and core/peripheral temperature differential. The behavioural signs are scored and the physiological responses are read from the monitors. The nurse has to take four recordings, one before, one during and two following the procedure. This may be complicated if multiple procedures are being performed.

Liverpool infant distress score (LIDS)

LIDS was developed by Horgan *et al.* (1996) to measure postoperative distress in neonates. They describe their choice of the word 'distress' rather than pain, stating 'in neonates one can never be absolutely sure that one is measuring pain' (Horgan *et al.* 1996:27) and so the word 'distress' was chosen. Their results suggest that distress is strongly linked to pain. This tool is based on both behavioural and physiological responses to pain. The behavioural responses are graded and the physiological responses are observed and charted. The behavioural responses include spontaneous movement, spontaneous excitability, flexion of fingers and toes, tone, cry quantity, cry quality, sleep and facial expression, and each is graded from 0 to 5, 0 being no pain and 5, extreme pain. The physiological observations include heart rate, oxygen saturation and blood pressure. LIDS requires the nurse to make assessments of all the criteria twice preoperatively, hourly for 6 hours postoperatively and then a further six assessments at 18, 19, 24, 25, 42 and 43 hours following surgery. Horgan *et al.* (1996) acknowledge that the scale requires shortening in order to make it more clinically acceptable.

Knowledge of the assessment tools available will assist the nurse in choosing one appropriate to individual needs, but in order to provide a consistently high standard of care to the neonate requiring intensive care one tool used throughout the neonatal unit would be more appropriate. It is essential to have a consistent approach to the assessment of neonatal pain.

Barriers to effective pain assessment must be overcome through education and research. In order to teach other health professionals and parents about the signs of pain in neonates requiring intensive care, the neonatal nurse must be able to readily recognise and interpret, not only the behavioural but also the physiological signs of pain. It is important that the carer recognises the significance of these signs if assessment of pain is to be successful (Givens-Bell 1994). Physiological changes are not always pain-related and the carer must be able to rule out other factors, such as agitation (Carlson *et al.* 1996). Horgan *et al.* (1996:24) suggest that 'a multidimensional assessment' is used and this should include physiological as well as behavioural criteria.

Potential consequences of untreated pain

In the neonate receiving intensive care, pain is a frequent and significant stressor on the baby's vulnerable central nervous system. Pain in the neonate that is not recognised or treated may not only have an adverse physiological effect, it may have lifelong consequences (Carlson *et al.* 1996). Franck (1993:915) suggests that pain can have physiological effects on all body systems and can be life-threatening in an acutely ill patient. Because of the immaturity of the central nervous system, the neonate may not be able to return to homeostasis following recurrent painful stimuli (Lynam 1995). Adverse physiological consequences include fluctuations in blood pressure and blood glucose and these may significantly increase the risk of developing intraventricular haemorrhages (Anand *et al.* 1987). The energy required to maintain heart rate, blood pressure and oxygenation during prolonged pain can deplete the energy required for tissue growth and repair (Stevens and Franck 1995) and may affect neurological development (Corff *et al.* 1995), resulting in increased morbidity and mortality (Givens-Bell 1994).

In 1979, Brazelton suggested that pain, which is a type of sensory input, may cause sensory overload in the neonate leading to a form of neonatal stress (cited in Carter 1990:8). He suggested that the stressed infant has to divert energy from growth and recovery to cope with the stress and thus shuts off from certain stimuli. Prolonged pain can deplete the neonate's stress hormones so that the infant will no longer respond to painful procedures by mounting a stress response (Franck 1993:915; Givens Bell 1994; Stevens and Franck 1995).

There may be significant short- and long-term consequences of repeated painful events in neonates (Anand *et al.* 1987; Stevens *et al.* 1993; Lynam 1995). The short-term effects of pain may disrupt feeding schedules, sleep/wake cycles and the ability to interact with parents positively (Stevens and Franck 1995; Carlson *et al.* 1996). There is still much debate about the long-term consequences

of pain in preterm neonates; however, it is thought that the effects may be impairments of neurodevelopment, learning and memory, and psychological problems (Anand *et al.* 1987; Shapiro 1993). Further research is needed in this area.

Anand *et al.* (1987:1326) state that neonatal responses to pain are 'suggestive of integrated emotional and behavioural responses to pain and are retained in memory long enough to modify subsequent behaviour patterns'. This would appear to suggest that the neonate has the capacity for memory of pain. Although the pain itself may not be remembered, the stress associated with it may be recalled and affect the infant later in life (Anand *et al.* 1987). Porter and Marshall (unpublished data) comment that mothers of infants who required neonatal intensive care have suggested that 'their infants seem to have a higher threshold for subsequent pain and a lower tendency to report pain than siblings not exposed to early pain' (cited in Porter 1989:551).

It is evident that untreated, recurrent pain may have devastating effects for the present and future well-being of the neonate. As with any other patient, the neonate has a right to be comfortable and free from pain. Caring for neonates in pain requires serious attention not only to the immediate effects of pain but also to the long-term developmental consequences. The neonate's developing nervous system is vulnerable to painful stimuli and if this pain is not adequately and effectively treated at the time, it may have an effect on future development. Neonatal nurses have a clinical and ethical responsibility to ensure that the neonate has appropriate and adequate pain relief and does not suffer unnecessarily. The neonatal nurse can help by disseminating information concerning the potential consequences of pain in order to increase awareness and knowledge, ensure effective pain management and improve the standard of care for the neonate in pain.

Relief strategies

The objectives of pain management in neonates are to diminish the intensity and duration of the pain, reduce the physiological effects, enhance the neonate's ability to cope and recover and provide the most effective solution with least risk to the baby (Franck 1993:918; Stevens and Franck 1995). Treatment and management of neonatal pain is complex, requiring a multidisciplinary approach. Skilled nursing care can be complemented by pharmacological therapies. Lynam (1995) suggests that as the infant's advocate, the nurse must be aware of research into non-pharmacological comfort measures and be able to evaluate the findings.

Non-pharmacological measures

Franck (1993:918) suggests that the nurse should always start with nonpharmacological measures before progressing to pharmacological agents because the nurse cannot prescribe analgesia.

Thoughtful nursing care

Cornick (1989) suggests that attentive, thoughtful nursing care may be all that is required to maintain comfort. This involves consideration of the neonate's environment. It has been suggested that the environment within the neonatal unit is too noisy and bright and this may cause stress to the sick neonate (Acolet *et al.* 1993). Reducing noise levels allows the neonate to rest and sleep. Having a 'quiet time' at set periods during the day, when radios are turned off, lights dimmed, visiting restricted and nursing and medical procedures are kept to a minimum, has been shown to prolong sleep periods (Straunch *et al.* 1993). Mann *et al.* (1986) looked at the effects of light on preterm infants. They found that those infants who were nursed in a room where lighting was reduced at night spent more time sleeping and gaining weight than those where the lighting was unchanged.

Pain can be minimised by efficient and skilled performance of invasive procedures and proper support during the procedure. Procedures should be grouped together to ensure that the baby has long periods of rest and recuperation (Carter 1994). Ill neonates often benefit from minimal handling.

Maintaining the neonate's comfort by ensuring that the baby is warm and dry is simple and effective and is standard practice within neonatal units. Sheepskins, soft bedding, such as 'Spenco' mattresses and bean bags, can also be used to provide comfort (Acolet *et al.* 1993; Carter 1994) and improve positioning to prevent skeletal and motor deformities (see p. 30).

Swaddling and facilitated tucking

Swaddling has been found to provide comfort for neonates (Lynam 1995). Stevens and Franck (1995) suggest that swaddling is the most appropriate non-pharmacological comfort measure for preterm and low birth weight infants. Swaddling involves the containment of the infant. **Facilitated tucking,** which is a variation of swaddling, has recently been suggested as a means to provide comfort to infants during minor pain (Corff *et al.* 1995). It is described as 'the gentle motoric containment of an infant's arms and legs in flexed, midline position close to the infant's trunk with the infant in a side-lying or supine position' (Corff *et al.* 1995:144). Corff *et al.* (1995) recommend that it is used in conjunction with other comforting and support measures and pharmacological methods. Van Cleve *et al.* (1995) suggest that continued support is required once the painful procedure is completed to maximise the beneficial effects. Facilitated tucking could be carried out as routine in conjunction with other forms of pain relief.

Non-nutritive sucking

Non-nutritive sucking (NNS) is the process of giving a pacifier, or dummy, to term and preterm infants to soothe them when undergoing a painful procedure

(see p. 245). Behavioural responses have been reduced by the use of NNS in both term and preterm infants undergoing a heelstab (Field and Goldson 1984). Miller and Anderson (1993) demonstrated that nasally intubated infants benefited from NNS when subjected to intravenous cannulation. In 1989 Campos found that the effects of NNS were more immediate than other non-pharmacological methods such as swaddling (cited in Lynam 1995:60). However, NNS does not appear to reduce the physiological changes caused by pain (Lynam 1995), so the sick preterm neonate may not derive much benefit from NNS, although it probably is comforting if the infant is mature enough to suck. Blass and Hoffmeyer (1991) tested the effectiveness of a sucrose-sweetened pacifier in term infants undergoing circumcision and found that it reduced the length of crying time in comparison to those infants who did not have a pacifier or those who had a water-moistened one. It is thought that sucrose causes endogenous opiates to be released (Blass and Hoffmeyer 1991). Haouari *et al.* (1995) demonstrated that a 50% sucrose solution appeared to reduce crying and heart rate when given to term infants subjected to a heel prick. They suggest that sucrose may be beneficial as a safe analgesic for minor painful procedures. However, Ramenghi *et al.* (1996a) researched the effects on term infants undergoing a heel prick of using a non-sucrose, sweet-tasting solution for NNS and found this to be as effective as a sucrose solution. They also studied the effects of sucrose in the reduction of pain in healthy premature infants subjected to a heel prick and found this effective in reducing their response to pain (Ramenghi *et al.* 1996b).

There are other non-pharmacological methods of pain relief available for use with neonates requiring intensive care such as touch, stroking and massage and music therapy.

Neonatal nurses should be familiar with the non-pharmacological methods in order to be able to comfort and soothe neonates more effectively. However, it should be remembered that whilst these methods appear to have a useful place in the treatment of mild neonatal pain they may not be appropriate for severe pain, and analgesia needs prescribing when the situation dictates, in conjunction with non-pharmacological methods. Consideration must be given to the neonate's condition, gestational age, postconceptional age, pain responses and the type of painful stimulus, and these considerations will enable the most appropriate strategies to be selected (Lynam 1995).

Pharmacological measures

Pharmacological agents may be required to alleviate pain in infants receiving intensive care who are subjected to numerous invasive procedures. There has been considerable discussion in recent years about the adequacy of pain relief given to neonates and this mostly centred around postoperative analgesia, but consideration must be given to providing more general pain relief, especially for ventilated babies. Distressed ventilated neonates are more likely to breathe asynchronously, thereby increasing the risk of pneumothorax (see p. 104) and subsequent intracranial haemorrhage (see p. 156) (Anand *et al.* 1987; Porter 1989). Andrews and Wills (1992) suggest that in every neonatal intensive care

unit, where the patients cannot communicate verbally, a protocol should exist for the treatment of pain.

There has been a general reluctance in the past to prescribe analgesia for neonates, possibly due to fear of side-effects, such as addiction, respiratory depression and decreased blood pressure associated with opiates, but this situation is changing. However, care must be taken in the choice and administration of the drug because of the pharmacodynamic implications in the neonate. O'Dell Mainous (1995) suggests that the limited ability of the neonate's immature liver and kidneys to break down and eliminate drugs puts the infant at greater risk of toxicity and complications. Opinions differ as to which pharmacological agent is best for the neonate and the choice may be dependent on the infant's condition, the site of pain, whether the infant is receiving mechanical ventilation and which route of administration is more appropriate (Carter 1994). The neonate must be able to tolerate enteral feeds if the medication is to be given orally. However, O'Dell Mainous (1995) suggests that due to the neonate's immature gastrointestinal system and altered gastric emptying times, oral absorption may be affected and so this route may not always be appropriate. Topical application of drugs, for example EMLA anaesthetic cream, can cause systemic effects due to the amount of absorption that is dependent upon the hydration of the infant and skin integrity and maturity (Clancy *et al.* 1992; O'Dell Mainous 1995). The intramuscular route of administration is also not always suitable for the premature, sick neonate who may have poor tissue perfusion and muscle mass (O'Dell Mainous 1995). The intravenous route is also not without its difficulties: the problems of absorption are bypassed but it causes a rapid response which may be hazardous (O'Dell Mainous 1995). An understanding of the pharmacokinetics, that is, absorption, distribution, metabolism and elimination of the drug, will help the professional to decide on the most appropriate pharmacological treatment for the neonate (see Chapter 19). Nurses must have a sound knowledge and understanding about the drugs they administer, their dosage, side-effects and interactions, and should also assess their effects (UKCC 1992).

Non-opioid analgesia

PARACETAMOL Paracetamol relieves mild to moderate pain (Clancy *et al.* 1992) and is probably more appropriate for the non-ventilated infant rather than the neonate requiring intensive care (see p. 326). Long-term usage may be a problem as the liver is still very immature in neonates. Paracetamol must be administered orally or rectally and so would not be appropriate for infants with, for example, necrotising enterocolitis.

Local anaesthesia

LOCAL INFILTRATION OF LIGNOCAINE OR BUPIVACAINE This can be used in the neonatal intensive care unit before painful procedures such as chest drain

insertion and lumbar puncture. Cornick (1989) suggests that bupivacaine can be used to infiltrate a wound site either during or after surgery (see p. 326). There are side-effects related to excessive absorption: these include central nervous system depression, irritability, convulsions, cardiovascular effects, allergic reactions and local inflammatory and necrotic effects (Andrews and Wills 1992). Collier (1994) believes that lignocaine or bupivacaine are the most suitable local anaesthetics for use in neonates.

TOPICAL AGENTS EMLA is a local anaesthetic cream comprised of lignocaine and prilocaine which has been successfully used in children to reduce the pain of venepuncture and venous cannulation. However, there is concern about its toxicity in infants of less than 3 months of age. It needs to be applied at least one hour before the procedure and this could cause difficulties if the first attempt at a procedure failed, as unless another site had been prepared, another hour would elapse before the procedure could be attempted again. Ametop (amethocaine) is also a local anaesthetic cream that has been used in the management of procedural pain in neonates. It provides rapid and prolonged anaesthetic effects and, unlike EMLA, does not contain prilocaine. However, further research is needed to assess the effectiveness and safety of using such topical agents in premature infants before any recommendations can be made.

Sedatives

CHLORAL HYDRATE This is a sedative, not an analgesic, which is commonly used in neonates. O'Dell Mainous (1995) suggests that sedation has a role in the comfort of the neonate but that it should not be used instead of analgesia. Clancy *et al.* (1992) maintain that sedation should complement rather than replace analgesia. Franck (1993:919) discusses the effects of sedation, in that it suppresses the behavioural signs of pain, which may result in the nurse thinking that the baby is not in pain. She suggests that sedation should only be used when the pain has been resolved. Chloral hydrate can be given orally or rectally. It causes gastric irritation so should be given with feeds (Andrews and Wills 1992; O'Dell Mainous 1995) and may not be suitable for some neonates, such as those with necrotising enterocolitis (see p. 436).

MIDAZOLAM Midazolam is a short-acting benzodiazepine which has sedative but no analgesic effects. It is given by injection as a single dose or as a continuous infusion. Experience of its use in neonates is still limited. It is associated with adverse effects; hypotension occurs particularly after a bolus dose. Paradoxical agitation has also been reported (Northern Neonatal Network 1998). It has unpredictable pharmacokinetics in neonates, making dose selection difficult. It accumulates after long-term use (more than 72 hours), which can result in an encephalopathic illness due to impaired glucuronidation ability (Royal College of Paediatrics and Child Health 1997). The dose of midazolam should be reduced after 24 hours to prevent drug accumulation.

Opioids

Opioids are the most commonly used pharmacological agents to relieve pain in neonates requiring intensive care. They provide effective analgesia against moderate to severe pain and also have sedative effects, but they are potent drugs with adverse effects and should always be used with caution (see p. 325).

MORPHINE Morphine is the most commonly used opioid in neonates. It is most effective when it is given by continuous infusion (Cornick 1989; Collier 1994). The side-effects of morphine include respiratory depression, apnoea, hypotension, decreased gastrointestinal motility, urinary retention, constipation, tolerance and withdrawal (Collier 1994; Givens-Bell 1994). It has a long half-life (6–14 hours in preterm, 2–11 hours in term babies) and provides greater sedation than other opioids (O'Dell Mainous 1995), but due to the problems of respiratory depression it is recommended that it is not used in neonates without ventilatory support (Andrews and Wills 1992) (see pp. 325 and 435).

DIAMORPHINE Diamorphine (di-acetylmorphine) is a semi-synthetic derivative of morphine which is metabolised to morphine. It, like morphine, is given as a loading dose followed by a continuous infusion (Barker *et al.* 1995). It has little to offer over morphine with the exception that it causes less histamine release and may cause less hypotension. The hypotension which occurs may be due to a reduction in stress in the baby as a result of the analgesic and sedative effect (Elias-Jones *et al.* 1991). Diamorphine has similar effects to morphine on respiration and the gut. It is more lipid-soluble than morphine, which may cause sedation more quickly than morphine (Wood *et al.* 1998) (see p. 435).

PETHIDINE Widely used in neonatal intensive care units until recently, pethidine has now generally been replaced with morphine. The problems associated with pethidine were in its administration. It was usually given as a bolus dose and therefore it was the responsibility of the nurse caring for the infant to assess the baby's need for it at regular intervals in order to avoid peaks and troughs in the pain. The baby often did not get the constant relief that is offered by continuous infusion. However, there does not appear to be any reference in the literature as to why it was given in such a manner; indeed, a lower dose of pethidine can be administered to an infant who is not receiving ventilatory support by slow infusion in order to prevent apnoea.

FENTANYL This popular opioid is used in many neonatal units because it has a rapid onset of action and brief duration (Clancy *et al.* 1992; Carter 1994). The side-effects are similar to those of morphine, although the risks of hypotension and respiratory depression are less (Andrews and Wills 1992; O'Dell Mainous 1995). Fentanyl, however, may cause chest wall rigidity if large doses are given rapidly (Clancy *et al.* 1992), which may compromise ventilation (see pp. 326 and 435).

CODEINE PHOSPHATE This is also an opioid drug that is metabolised to morphine. It is not widely used in neonatal intensive care units, but Andrews and Wills (1992) suggest that it can be administered to non-ventilated neonates provided they are strictly monitored, as respiratory depression and apnoea can occur. They stipulate that it should not be given intravenously because the risks of apnoea and hypotension are increased; it should instead be given intramuscularly or orally.

There are several safe and effective ways of providing pain relief to the neonate requiring intensive care. Consideration must be given as to which is the most appropriate for each individual infant. Pain management is a team effort, with nurses and doctors working together to assess and manage pain. Because of all the advances made and increased knowledge of both non-pharmacological methods and pharmacological agents there is now no reason why the neonate requiring intensive care should suffer unnecessarily.

Conclusion

There is now a considerable body of evidence to support the belief that the neonate has the capacity to feel pain. The survival of small and extremely preterm infants in good condition is now possible with advanced technology, but increased awareness is required about their vulnerability to pain and stress (Corff *et al.* 1995). Providing comfort and effective pain management are important areas for the nurse, as the baby's advocate, to consider. Neonatal nurses are in an ideal position in caring for infants to assess their needs, but management of pain will only be successful if there is cooperation between all the professionals involved (Givens-Bell 1994; Carlson *et al.* 1996). The professionals' own beliefs and attitudes about pain may affect their practice and should therefore be examined (Llewellyn 1994). The use of a pain assessment score will enable the carer to move away from these and will give evidence to support claims that an infant is in pain (Keeble and Twaddle 1995).

Carr (1997) suggests that education is an important tool for improving the management of pain. She states, 'it is essential that practitioners are armed with the most appropriate knowledge, can confidently use pain-relieving strategies and have the ability to consider potential barriers to effective pain management' (Carr 1997:416). Sparshott (1991:32) maintains that 'better understanding will inevitably lead to better practice' and Keeble and Twaddle (1995) demonstrated that the management of pain improved within their unit following education and the introduction of a pain assessment tool.

In order to ensure that babies receive a uniformly high standard of care in the area of pain assessment and relief, nurses must apply their knowledge, understanding and experience to practice. Research findings should also be applied to practice. Langley (1997:18) discusses how research influences

neonatal care, suggesting that 'neonatal nurses have initiated changes and innovations in practice, through research'. Hunt (1987) observes that in order to utilise research findings, the information must be disseminated to the appropriate audience in order to create awareness and interest. Nurses have a responsibility to share their knowledge with other professionals. If research is going to be utilised within current practice then a genuine interest in improving care through research must be demonstrated by the carers (Closs and Cheater 1994).

There is no reason why the neonate requiring intensive care should suffer pain. There are effective non-pharmacological and pharmacological methods available currently to provide comfort and relieve pain. Further work is required to develop an effective and clinically usable pain scale to assess neonatal pain and to document the responses of the neonate requiring intensive care to painful stimuli. However, research is ongoing and therefore the policies and protocols related to the management of pain in the neonate requiring intensive care need constantly to be reviewed and updated to ensure that practice is appropriate, up-to-date and research-based.

Case study 1: pain relief in a premature infant

James was born at 27 weeks' gestation (weighing 1.02kg) by emergency lower segment cesarean section for maternal antepartum haemorrhage and required intubation at birth in order to establish adequate ventilation. He was then transferred to the neonatal unit, ventilated. On the neonatal unit he received intermittent positive-pressure ventilation. Investigations revealed that James had respiratory distress syndrome and he received surfactant therapy. His Blood sugar was unrecordable so an intravenous cannula was sited and immediate intravenous fluids were administered. An umbilical artery catheter was inserted in order to monitor his arterial blood gases, infuse maintenance fluids and monitor blood pressure. He needed increased oxygen requirements when handled and during endotracheal/ oropharyngeal suction. A central venous line was sited on day 2 in order to administer total parenteral nutrition.

From this case study, answer the following questions:

Q.1. Give reasons why you think pain relief is necessary for James.
Q.2. What signs might be displayed by James to indicate his need for pain relief?
Q.3. How would you assess his degree of pain?
Q.4. What pain-relieving strategies would you use? Give reasons for your choice.
Q.5. What could be the consequences of inappropriate pain relief for James?

Case study 2: management of pain relief for an infant with necrotising enterocolitis

James was extubated into 30% oxygen on day 6 and by day 10 was tolerating full nasogastric tube feeds and the central venous line was removed. By day 12 he was in air. However, at 21 days of age, James developed abdominal distension, bile-stained aspirates from his nasogastric tube, pallor and lethargy. He also developed an oxygen requirement and had apnoeic episodes and bradycardias. Investigations revealed that he had developed necrotising enterocolitis. He subsequently required intubation, ventilation and surgery and was commenced on a morphine infusion for pain relief. On day 3 post surgery, James was extubated but he demonstrated signs of pain and discomfort when handled.

From this continued case study, answer the following questions:

Q.6. What pain-relieving strategies would you recommend for James? Give reasons for your answer.

Q.7. How would you monitor the success of the pain-relieving strategies?

Q.8. How would you help to disseminate knowledge and information about pain in neonatal intensive care to colleagues?

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Chapter 10

Fluid and Electrolyte Balance

Contents

Introduction

Renal function in the neonate, though differing from that of more mature subjects, is still sufficient to maintain normal homeostasis. The adult number of nephrons within the kidney is only achieved by 34–35 weeks' gestation, and even then they are shorter and less functionally mature (Blackburn and Loper 1992). Partly, but not entirely, as a result of this fluid and electrolyte disturbances are common in newborn infants. Babies born at the extremes of prematurity are particularly vulnerable, but term infants are not exempt, and renal pathology is commonly seen secondary to sepsis or ischaemia. These problems may be further compounded by the administration of nephrotoxic chemotherapy. As it is the role of the neonatal nurse to administer fluid therapy, monitoring of its adequacy and detection of adverse signs are mandatory if problems are to be avoided. To achieve this the nurse requires a robust understanding of the subject matter.

The intention of this chapter is to highlight renal physiology in order to enable the nurse to anticipate problems, assess the adequacy of renal function, and subsequently plan the management of infants in intensive care.

Embryological development

The development of the kidneys is along a continuum that begins in the first week of gestation and continues until 34–36 weeks postconceptual age. Formation of the nephrons—nephrogenesis—occurs from week 7, with glomerular filtration and urine formation occurring two weeks later. The urine produced is excreted into the amniotic cavity, contributing to a major part of the amniotic fluid volume (Moore and Persaud 1993). Initially, the kidneys develop in the pelvis and lie close together. Slowly their position changes, following a path along the dorsal aorta to the lumbar region and then rotating medially (Bissinger 1995). By 9 weeks of gestation they are separated and have taken up the adult, retroperitoneal, position in the abdomen.

At term, each kidney contains the adult complement of 800000–1000000 nephrons. In the preterm infant nephrogenesis continues at the same rate as it would in utero (Bonilla-Felix *et al.* 1998). Therefore, an infant born at 24 weeks' gestation will not complete this process for 10–12 weeks. At 24 weeks the kidney measures 2.5 cm and has grown to 4.5 cm at term. The adult size of 6 cm is achieved postnatally by the elongation of the proximal renal tubules and the loops of Henle (Moore and Persaud 1993).

Physiology of urine production

Urine production begins with the process of glomerular filtration. As the blood flows through the afferent arteriole into the glomerulus, non-selective filtration

occurs in which fluid and solutes pass through the capillary membrane into Bowman's capsule. The rate of filtration is affected by several factors, but the most important is the hydrostatic pressure. Glomerular filtration rate (GFR) is low in newborns, 20 ml/min/1.73 m^2 at term compared with the adult rate of 80 ml/min. This decreases to 10 ml/min/1.73 m^2 at 28 weeks and further to 2 ml/min/1.73 m^2 at 25 weeks (Blackburn and Loper 1992; N.B. 1.73 m^2 is a correction factor for difference in surface area between persons of different sizes). The GFR rises after birth due to a progressive rise in systemic blood pressure, fall in renal vascular resistance and an increase in renal blood flow from 4 per cent of the cardiac output to 10 per cent. This will further rise to the adult level of 25 per cent in the first few days of life. The GFR appears to increase in a programmed way at least from 26 weeks' gestation and from the second postnatal day unaffected by the gestation at birth (Wilkins 1992a). The preterm infant should produce a minimum of 25–60 ml of urine per kilogram per 24 hours, which can rise maximally to 300 ml/kg per 24 hours with acute increases in fluid intake. The minimum value (1 ml/kg/hr) represents the lowest acceptable volume, as below this level solute accumulation will occur. Infants producing such a low volume warrant further investigation as a matter of urgency.

Sodium balance

Sodium is the major cation in extracellular fluid, and is vital for the regulation of circulating blood volume as well as weight gain and tissue growth.

The newborn term kidney can filter and reabsorb sodium reasonably efficiently. Utilising approximately 9 per cent of the infant's oxygen consumption for energy, sodium and water is reabsorbed from the nephron back into the circulation. Due to the high concentration gradient of sodium, it is the first ion to move from the filtrate in the tubular lumen to tubular epithelial cell. A normal physiological negative balance of sodium and water has been found to occur in healthy newborn infants during the first few days of life.

The preterm infant is less efficient at both filtering a delivered sodium load and reabsorbing filtered sodium, and is thus at risk of both hypernatraemia and hyponatraemia. Studies have demonstrated a high sodium excretion rate of 5–15 per cent, with infants becoming hyponatraemic before or after the first postnatal week (Wilkins 1992b). The very preterm infant is also at risk of high transepidermal water losses (see p. 72), increasing the propensity of hypernatraemia.

Modi (1993) suggests that in sick preterm infants an early intake of sodium is unnecessary and possibly harmful and should be avoided until the physiological postnatal diuresis, or a weight loss of 7 per cent of the birthweight has occurred. Thereafter normal maintenance requirements are 2–3 mmol/kg per day.

Sick infants' requirements, however, will vary according to a number of factors, and this requires careful clinical assessment prior to supplementation. Reasons for sodium imbalances are shown in Table 10.1.

Table 10.1 Reasons for sodium imbalances

Hyponatraemia	*Hypernatraemia*
Excess renal loss (particularly very preterm)	Excess transepidermal losses (particularly very preterm)
Insufficient replacement: Sodium poor intravenous therapy Expressed breast milk with low sodium fortifiers	Insufficient fluid replacements: Whilst under phototherapy or radiant heaters
Diuretics and xanthines (i.e. caffeine)	Glycosuria causing osmotic diuresis
Inappropriate ADH secretion	Drugs or parenteral nutrition fluids containing excessive sodium supplementation
Adrenocortical failure	
Barrter's syndrome (associated with hypokalaemia)	
Salt-losing congenital adrenal hyperplasia	

Hyponatraemia

Hyponatraemia is often evident at birth, reflecting maternal hyponatraemia (Wilkins 1992b). Hyponatraemia occurring during the first week of life may be due to water retention or sodium wastage, although opinion is mixed as to which is the most likely (Haycock and Aperia 1991). Inappropriate antidiuretic hormone (IADH) syndrome is frequently implicated as the factor responsible for the hyponatraemia (Rees *et al.* 1984). Elevated ADH levels are often seen along with hyponatraemia; this may be due to infant conservation of water to maintain circulating volume, at the expense of the plasma osmolality. It is likely that in these situations secretion of ADH syndrome is *physiologically appropriate*. This differentiation is important, as a true case of IADH requires restriction of fluids, whereas in physiological adaptation due to low circulating volume an increase of fluid volume and sodium may be what is really necessary (Wilkins 1992b).

Irrespective of the cause of the hyponatraemia, an appropriate positive sodium balance is vital, since a chronic deficiency is associated with poor skeletal and tissue growth with adverse neurodevelopmental outcome (Haycock 1993).

To calculate an infant's sodium deficit the following formula is used:

Sodium deficit=0.7×body weight (kg)×(140-actual sodium)

To correct the deficit, aim to replace two-thirds of the deficit within 24 hours, with monitoring of plasma and urine electrolytes at least daily to determine subsequent replacement.

Where the sodium deficit is due to inappropriate ADH secretion, fluids are restricted along with sodium replacement.

Hypernatraemia

If moderate hypernatraemia presents (serum level of up to 155 ml/l), sodium supplements are best avoided and appropriate fluid replacement should correct the imbalance.

At higher levels (serum level >155 mmol/l) care must be taken not to lower the plasma concentration faster than 10 mmol/day. Rapid falls in sodium concentration can precipitate cerebral oedema and convulsions. Judicious additional sodium may be required, whilst the fall in plasma sodium is carefully monitored.

Potassium balance

Potassium is the principal intracellular cation and required for maintenance of the intracellular fluid volume.

Potassium reabsorption, which occurs mainly in the distal tubule, is mediated by aldosterone which regulates the sodium potassium pump to maintain cellular electroneutrality. Renal regulation of potassium is linked to the arterial pH. In a metabolic alkalotic state the kidney will excrete potassium in exchange for sequestering bicarbonate within the cells, resulting in hypokalaemia. In a metabolic acidotic state, potassium is exchanged for hydrogen ion in the proximal tubule, increasing the plasma concentration.

In infants requiring parenteral fluids, potassium supplementation should commence when urine output is adequate, providing that the plasma level is not elevated. The daily requirement is 2 mmol/kg per 24 hours.

Reasons for potassium imbalances are shown in Table 10.2.

***Table 10.2* Reasons for potassium imbalances**

Hypokalaemia	*Hyperkalaemia*
Insufficient replacement	Tissue damage Excessive bruising Nephrotoxic drugs
Excess losses: Renal – polyuric states – Diuretics and other drugs – Hyperaldosteronism – Bartter's syndrome (with hyponatraemia) – Renal tubular acidosis	Renal Failure: Acute tubular necrosis Urethral valves Renal vein thrombosis Congenital chronic renal failure
Gastrointestinal – Gastric aspiration – Vomiting – Ileostomy losses – Diarrhoea	

Hypokalaemia

It has been found that extreme variations in the plasma concentration of potassium can occur in neonates, both above and below the normal range of 3.8–5.0 mmol/l, indicating rapid changes between the intracellular and extracellular compartments (Rogan 1998). A deficiency invariably involves an excessive loss via either the intestinal or renal routes, or both, or a lack of appropriate supplementation.

Potassium supplements should be commenced on the third postnatal day at a rate of 1–2 mmol/kg per day, provided an adequate urine output (over 1 ml/kg/hr) has been observed and the plasma level is not elevated. To correct hypokalaemia, it is recommended to administer appropriate replacements as indicated by normal maintenance requirements and to estimate ongoing losses with careful monitoring of serum levels.

Hyperkalaemia

Hyperkalemia (level >6.5 mmol/l) is always associated with a failure of the renal excretory mechanisms or secondary to an overwhelming situation from an acidosis or severe infection. Elevated levels can also occur if there has been extensive bruising, ischaemia or renal failure. Hyperkalaemia can also be reported following haemolysis of blood cells during sampling. True hyperkalaemia is extremely serious and potentially a lethal condition; the management must be prompt. All potassium supplementation or drugs containing potassium must be stopped and increased ECG surveillance commenced, closely observing for arrhythmias. Calcium gluconate may be infused slowly to counteract the toxic effects of potassium on the myocardium. As calcium gluconate extravasation injury causes severe and permanent tissue damage, the infusion site should be clearly visible and carefully observed. Other management strategies involve removing potassium from the body by the use of ion exchange agents such as rectal calcium resonium, or reducing the serum potassium level by altering the cell membrane threshold to 'push' potassium back into the cell. This can be achieved by the intravenous infusion of sodium bicarbonate (4 mmol/kg 4.2% solution over 5–10 minutes), glucose and insulin solution (0.3 units of insulin per kilogram per hour), monitoring blood glucose levels carefully, or salbutamol (4 µg/kg over 20 minutes; Northern Neonatal Network 1993).

Chloride balance

Chloride follows sodium from the filtrate in the tubular lumen into the tubular cell. In the neonate, hypochloraemia may result from increased losses due to loop diuretics. Hyperchloraemia can occur in infants on parenteral nutrition, resulting in metabolic acidosis.

Calcium and phosphate balance

Calcium and phosphate are essential minerals required for normal growth and development and, in particular, bone mineralisation. The fetal acquisition of calcium occurs mainly in the third trimester, with a daily accretion rate of 120–150 mg/kg (Kleigman and Weld 1986). Perinatal asphyxia can also lead to a fall in calcium levels due to altered cell metabolism and phosphorus release, with alkalosis from alkali therapy or from over-ventilation reducing the ionised serum calcium levels further (Cruz and Tsang 1992).

Calcium and phosphate absorption is regulated by calcitonin, parathormone and the active metabolites of vitamin D, namely 1,25 dihydroxyvitamin D.

Term infants are able to convert vitamin D in the liver and kidneys, but this process is limited in the preterm infant, and does not occur at significant rates until 36 weeks' gestation (Specker and Tsang 1986). Calcium maintenance requirements are 2.0 mmol/kg per 24 hours (Rennie 1992). If an infant presents with signs of hypocalcaemia, for example, tremors, twitching, irritability, laryngospasm, high pitched cry, apnoea or seizures, an increased dose of 2 ml/kg by slow bolus injection (Barnes and Cheetham 1999) may be warranted, but should be given with extreme caution under strict ECG control as bradycardia and asystole can occur if given too rapidly. In addition, calcium is notorious for severe extravasation injury and scarring, so close observation of the cannula site is mandatory.

Water management

Water management has to include not only water being administered, but also endogenously produced water. Approximately 10 per cent of body water is produced by oxidative cellular metabolism (Blackburn and Loper 1992). This has to be balanced with all that is lost via the differing routes.

Term infants' urinary losses are in the region of 5–10 ml/kg per 24 hours. Stool water losses are minimal in the first few days but are subsequently estimated at 5–10 ml/kg per 24 hours. Basal insensible water losses in the term newborn are approximately 20 ml/kg per 24 hours, with 70 per cent being lost through the skin and 30 per cent via the respiratory tract. Water losses via the skin are primarily influenced by gestational age and postnatal age. There is an exponential relationship between TEWL and gestational age, with a 15-fold increase in water losses in infants born at 25 weeks to those born at term. The losses are markedly different (steeper) between those born at 25 weeks and those born at 28 weeks of gestation (Sedin 1996). Increasing the ambient humidity of the infant's environment to 50–80 per cent substantially reduces these losses (see page 81), as does prevention of epidermal stripping. Care of the skin and maintenance of high humidity around a preterm infant are major nursing priorities.

Fluid requirements

Fluid intake is usually calculated on the infant's birthweight, until that weight is exceeded, unless weight gain is thought to be due to fluid retention and/or oedema.

The fluid requirement for growth in a preterm infant is thought to be 130–160 ml/kg per 24 hours, but until the postnatal diuresis begins much less is required and usual 24 hour starting figures are 60 ml/kg. The volume is then increased in a stepwise fashion over the next four or five days (Rennie 1992).

Some infants may require a more rapid increase in fluid intake, for example, very preterm infants with insensible losses that are not minimised, whilst other infants, for example, following asphyxial injury, require a more cautious approach and may undergo fluid restriction.

Fluid requirement should be calculated on the basis of anticipated losses and the volume required to administer an appropriate amount of nutrition. Additionally, the infant's weight, general condition and disease state need consideration. If any fluid is to be administered enterally, gastrointestinal tolerance is an important factor.

Monitoring fluid balance

Recording and monitoring the fluid balance of infants in intensive care is principally the role of the nurse caring for the infant. This aspect of care is as important as any other type of physiological monitoring, as imbalances and inaccuracies in fluid balance can cause serious deterioration or impair the infant's recovery. It is for these reasons that fluid balance recording should become second nature to neonatal nurses and observation and recording of the fluid status be meticulous.

Monitoring of fluid balance includes:

Accurate measurement of body weight

Weight correlates very well with total body water content during the first few days of life (Shaffer *et al.* 1986). Whilst infants in the NICU are physiologically unstable and warrant minimal handling, weighing is an important marker for fluid balance and should be undertaken daily in most instances. The risks of the procedure, for example, accidental extubation, temperature instability and brief disconnection from IPPV, have to be balanced against the infant's condition and the benefits from the information gained. The procedure should be well planned and executed by skilled personnel, to lessen the occurrence of potential problems.

Weight loss is normally in the region of 1–3 per cent of the body weight per day, reaching a maximum of 10–15 per cent by day 5 in preterms, with weight gain commencing by 7–10 days (Rennie 1992; Modi 1999).

Measurement of urine volume

Urine output must be accurately measured as it is a marker for not only water balance but also renal perfusion. The volume voided can be estimated in several ways. Weighed nappies provide the simplest method, but evaporation of the urine into the environment or further damping of the nappy by incubator humidity may create spurious results, and this should be considered. Commercially available adhesive bags may be used but the fixation and retention without leakage can be a problem. Additionally, when the bag is removed, significant epidermal stripping can occur. The use of the finger of a polythene glove fixed to the skin with a smear of soft paraffin is sometimes successful in small inactive males, and some success can be achieved in female infants by using the thumb of a glove applied in the same way.

Urine output should be in the region of 1 ml/kg per hour on day 1 rising to >2–3 ml/kg per hour subsequently.

Urinalysis

Urinalysis is a procedure nurses undertake from very early in their clinical practice experience and yet it is often overlooked in its importance in renal compromise. Catching volumes of the urine can be difficult in the newborn (see above), but as only a small amount is needed for urinalysis, simply placing cotton wool balls into the infant's nappy, and then aspirating the absorbed urine with a syringe, is sufficient for the task and avoids skin damage from the application of adhesive urine bags.

SPECIFIC GRAVITY (SG) SG reflects the ability to concentrate and dilute urine. As the ability to concentrate urine is limited, the maximum specific gravity is usually 1.015 to 1.020, but the minimum may be as low as 1.001 to 1.005. As other factors such as glucose and protein can alter SG, osmolality measurement of both blood and urine should be undertaken and compared if concentrating ability is being questioned. Osmolality equivalent of the above SG values are 700mosmol/l and 100mosmol/l respectively (Rennie 1992).

URINE PH The pH reflects the ability to acidify urine, but it is usually relatively alkalotic at 6.0. Many neonates can acidify the urine to 5.0.

GLYCOSURIA This estimation reflects the kidneys' ability to handle glucose and is usually associated with a high blood glucose level. High blood glucose levels associated with renal losses can contribute to osmotic diuresis and dehydration.

PROTEINURIA Proteinuria is frequently seen in small amounts and is related to gestational age. If proteinuria persists, 12–24-hour urine collection should be undertaken as it may indicate renal injury, congestive heart failure, sepsis or elevated venous pressure (Boineau and Levy 1992).

HAEMATURIA Haematuria is not a normal finding and may be associated with renal damage following asphyxia, embolisation in the renal artery from UAC in situ, renal vein thrombosis, coagulopathies or congenital abnormality, for example cystic disease or obstruction.

Glucose homeostasis

During intrauterine life the fetus is dependent upon a constant supply of transplacental glucose to provide energy for metabolic functions and substrate for growth. Fetal glucose utilisation, at 6 mg/kg per minute, is greater than the neonatal requirement of 3.5–5.5 mg/kg per minute, which is approximately twice the adult requirement.

Fetal blood glucose levels are approximately 70–80 per cent of the maternal level which allows the process of facilitated diffusion across the placenta to occur.

Whilst the stored form of glucose, glycogen, is laid down from as early as 9 weeks, the rate of deposition accelerates during the third trimester. Thus, the more preterm the infant is, the smaller the reserves are, increasing susceptibility to hypoglycaemia. The timing of the increased deposition of stored substrate is probably related to the fetal energy needs during the process of labour and delivery in order to maintain blood glucose levels during the anaerobic conditions. Should the labour become difficult or prolonged, substrate utilisation increases during anaerobic metabolism, depleting hepatic and cardiac stores rapidly, making the term asphyxiated infant also susceptible to hypoglycaemia (see p. 45).

At birth, the fetus undergoes a shift from an intrauterine anabolic-dominant state to a neonatal catabolic state (Blackburn and Loper 1992), as separation from the maternal supply occurs. All newborns, therefore, have a potential for glucose instability but the susceptibility increases in certain situations (see Table 10.3), and is probably greatest in infants requiring admission to NICU.

Glucose is the main substance required by the body to create adenosine triphosphate, referred to as the energy 'currency' (Karp *et al.* 1995). After birth, the neonate has to regulate its own glucose metabolism. To survive, the newborn infant utilises the processes of glycogenolysis, the breakdown of stored glycogen to glucose, and gluconeogenesis, which is glucose produced from non-carbohydrate sources, such as alanine from skeletal muscle. The diffusion of glucose into the cells is greatly enhanced by insulin.

Monitoring of blood glucose levels of infants, both on admission and subsequently, is a primary responsibility of the nurse if the adverse event of glucose instability is to be avoided.

Renal handling of glucose

The ability of the tubules to reabsorb glucose is decreased in the preterm infant and increases towards term. The preterm infant has a low renal

threshold for glucose conservation, and varying degrees of glycosuria are not uncommon. Glycosuria in conjunction with hyperglycaemia may cause an osmotic diuresis whereby water is lost due to the high urinary solute concentration. This situation can result in other electrolyte instabilities, such as hypernatraemia. Osmotic diuresis, however, is unlikely to occur in blood glucose levels below 12 mmol/l (Coulthard and Hey 1999).

Reducing the concentration of infused glucose to a level that will reduce the blood glucose level is often all that is required. Infants not responding to this measure may be prescribed intravenous insulin therapy. Whilst this practice appears to occur relatively frequently, there have been no controlled studies to validate it and it must therefore be undertaken with caution (McGowan *et al.* 1998; Hawdon and Aynsley-Green 1999).

Hypoglycaemia

The definition of neonatal hypoglycaemia has long courted controversy, with definitive values ranging from below 1 mmol/l to below 4 mmol/l (Koh and Aynsley-Green 1988a). Hawdon and Aynsley-Green (1999) suggest that, in light of current data, a blood glucose of less than 2.6 mmol/l should be avoided, especially in infants who are unable to utilise ketone bodies as substrate, which due to their impaired metabolic responses incorporates most infants in the NICU. Infants at risk of hypoglycaemia fall predominantly into two broad categories: those with reduced stores and those with increased utilisation (Table 10.3).

Management

Early identification and close surveillance of the infant at high risk of developing hypoglycaemia, and institution of prophylactic measures to prevent its occurrence, constitute by far the best treatment strategies for this disorder (McGowan *et al.* 1998). Commencement of early feeding, either enteral or intravenous, is advocated. Intravenous 10% dextrose at 4–6 mg/kg per minute (Digiacomo and Hay 1992) should be sufficient for most infants.

If hypoglycaemia occurs despite this, a bolus of 10% dextrose at 3–5 ml/kg should be given slowly followed by an infusion (Digiacomo and Hay 1992; Hawdon and Aynsley-Green 1999). Higher concentration should be avoided due to the risk of tissue injury and rebound hypoglycaemia. If higher concentrations are required to maintain blood glucose levels, the infusion must be administered via a central venous line in order to prevent vessel damage, extravasation injury and scarring due to the sclerosing effects of the solution (Hawdon and Aynsley-Green 1999). Vigilant observation of the infusion site and infusion pump pressures must be instituted. In hypoglycaemic conditions not responding to dextrose infusions other agents have been used, including

Table 10.3 Infants at risk of hypoglycaemia

Infant group	*Mechanism*	*Expected duration*
Decreased stores		
Preterm	Decreased stores of glycogen and fat Enteral feed intolerance Fluid (caloric) restriction Impaired hormonal responses	Transient
Intrauterine growth retardation	Decreased stores of glycogen and fat Impaired hormonal responses	Transient
Inborn errors of metabolism	Glycogen storage disease Enzyme deficiencies impairing glycogenolysis and gluconeogenesis	Prolonged
Increased utilisation		
Perinatal hypoxia	Anaerobic cellular metabolism exhausting stores	Transient
Sepsis	Increased metabolic rate	Transient
Hypothermia	Increased metabolic rate and brown fat metabolism	
Infant of diabetic mother	Hyperinsulinaemia	Transient
Beckwith–Wiedeman syndrome	Hyperinsulinaemia from islet hyperplasia	Prolonged
Erythroblastosis fetalis	Hyperinsulinaemia from islet hyperplasia	Transient
Exchange transfusion	Excess insulin secretion due to glucose level in stored blood	Transient
Islet cell dysplasias	Hyperinsulinism	Prolonged
Other causes		
Iatrogenic	'Tissued' IVs abruptly reducing supply	Transient
	Glucose infusion via UAC if tip is close to the coeliac access	Transient
Maternal drugs	Beta-agonist tocolytics	Transient

diazoxide, glucagon, somatostatin and hydrocortisone, but none of these agents appears to have been studied in a controlled fashion, and they are neither appropriate nor necessary in most situations.

Whilst the consequences of asymptomatic hypoglycaemia are not well established, Lucas *et al.* (1988) did report lower motor and mental development scores following five episodes of blood glucose levels below 2.6 mmol/l in the very low birth weight population. Neurological outcome following hypoglycaemic seizures, however, is poor (Fluge 1975; Koh *et al.* 1988b).

Hyperglycaemia

Neonatal hyperglycaemia is defined as a blood glucose level >7 mmol/l in term infants and >8 mmol/l in the preterm (Digiacomo and Hay 1992; McGowan *et al.* 1998), and is a much less common condition in the newborn than hypoglycaemia. Neonatal diabetes mellitus occurs in 1 in 400 000 livebirths (Shield *et al.* 1997), but the incidence of 'transient diabetes' (see Table 10.4) appears to be increasing as more extremely low birth weight infants are actively managed within the NICU. This is probably reflective of an immaturity of the usual regulatory mechanisms, including decreased insulin response, and the provision of a high glucose load (King *et al.* 1986; Hawdon *et al.* 1993).

The consequences of hyperglycaemia are not well defined. There may be an increased risk of intracranial haemorrhage if serum osmolality is increased due to the high serum glucose level, but this remains largely speculation (Digiacomo and Hay 1992) (see p. 221).

***Table 10.4* Infants at risk of hyperglycaemia**

Infant group	*Mechanism*	*Expected duration*
Extremely low birth weight infants	High intravenous glucose concentration Immature insulin and regulatory mechanisms	Transient
Infants receiving methyl xanthines		Transient
Infants undergoing surgery	Release of stress-related hormones Infusion of high glucose-containing solutions Transfusion of blood with high glucose levels	Transient
Neonatal diabetes mellitus	Decreased insulin production	Prolonged

Acute renal failure

Acute renal failure (ARF) is defined as 'the sudden deterioration of the kidneys' baseline function resulting in an inability to maintain the body's fluid and electrolyte homeostasis' (Bonilla-Felix *et al.* 1998). It is said to occur in up to 8 per cent of neonates admitted to the NICU (Stapleton *et al.* 1987).

Renal failure is classified into three main categories, those of: pre-renal, intrinsic and post-renal or obstructive failure (see Table 10.5). Pre-renal failure often precedes established renal failure and is reversible if prompt attention is paid to correcting renal perfusion. As the first clinical sign of pre-renal failure is the onset of oliguria, careful monitoring of urine output is mandatory for an infant requiring intensive care.

The commonest reasons for ARF are asphyxia, sepsis, necrotising enterocolitis and major surgery, but it should be considered in any neonate whose hourly urine output falls below 1 ml/kg (Modi 1999).

Investigations

When oliguria is first recognised, the bladder should be palpated to eliminate acute urine retention, which is not uncommon after asphyxial insults. Assessment of the adequacy of the circulation, blood pressure, capillary refill time and core-periphery gap must also be undertaken. A useful adjunct to diagnosis is a renal ultrasound. This will clearly identify any anatomical abnormality, obstructive problem, congenital malformations such as polycystic disease or aplasia, or renal vascular thrombosis.

Paired blood and urine analysis may assist in establishing the diagnosis of renal failure. Measurement of fractional excretion of sodium (FeNa) is said to be the best indicator to distinguish between pre-renal and established renal failure.

***Table 10.5* Causes of renal failure in infants**

Pre-renal	*Intrinsic*	*Obstructive*
Due to systemic hypovolaemia Haemorrhage Septic shock Necrotising enterocolitis Dehydration	Acute tubular necrosis Congenital malformations Agenesis Polycystic kidneys	Congenital malformations Strictures Obstructive lesions Renal calculi
Due to hypoperfusion Asphyxia Heart failure Respiratory distress syndrome	Infection Renal vascular thrombosis Nephrotoxic drugs	Fungal balls Neurogenic bladder Compression Teratomas

Adapted from Karlowicz and Adelman 1996

It is calculated from the sodium and creatinine concentrations of serum and spot urine samples. For example:

$$\text{FeNa} = \frac{\text{Urine creatinine/Serum Na}}{\text{Urine creatinine/Serum creatinine}}$$

If an infant is dehydrated with intact tubular function, sodium and water will be conserved and the FeNa will be less than 1 per cent. However, if tubular damage is causing the oliguria the FeNa will be in the region of 3 per cent (Bonilla-Felix 1998). Other indices are listed in Table 10.6.

Use of these indices in very preterm infants is unreliable and, in some instances, they overlap. The response to a fluid challenge is often helpful. This consists of administering 20–30 ml/kg of 0.9% saline over 1–2 hours, followed by a single dose of furosemide on completion of the infusion. If the infant responds with an increase in urine output, a diagnosis of pre-renal failure is likely and further management should be directed towards maintaining adequacy of renal perfusion. Furosemide may be given as a single dose of up to 5 mg/kg. As the half-life of furosemide is up to 24 hours (Northern Neonatal Network 1993), repeated doses cannot be advocated due to its potential ototoxic and nephrotoxic effects. (N.B.Furosemide is now accepted British nomenclature and should replace frusemide.)

Management

Management should be directed to correcting the underlying cause, for example, correcting poor perfusion, hypotensive states and sepsis. Successful management of these infants is pivotal upon meticulous measuring and accurate recording of urine output, in conjunction with a fastidious account of *all* fluids administered.

Table 10.6 **Indices of renal failure in infants**

	Pre-renal failure	*Established renal failure*
Urine sodium	Low (< 10 mmol/l)	High (>20 mmol/l)
Urine urea	High	Low
Urine creatinine	High	Low
Urine specific gravity	High (> 1025)	Low (approx. 1010)
Urine osmolality	High (> 500 mosmol/kg)	Low (approx. 300 mosmol/kg)
Fractional excretion Na	Low (< 1%)	High (> 2.5–3.0%)
Urea : plasma creatinine	High (> 40)	Low

All nephrotoxic drugs should be stopped where possible. The adjusting of dose schedules of all renally excreted drugs also needs consideration, with drug assays guiding the adjustments. Fluids should be calculated as insensible losses plus urine output, and any gastrointestinal losses. Furosemide may be given in doses up to 5 mg/kg (Modi 1999). Low dose dopamine 2–5 µg/kg per minute may aid renal perfusion. In hypotensive infants inotropic support may be required, but as high dose dopamine decreases renal blood flow, dobutamine 5–10 µg/kg per minute should probably be used. Acidosis may require treatment with sodium bicarbonate 4.2% or 3.6% tromethamine (THAM). Hyperphosphataemia and hypocalcaemia, if persistent, may require oral calcium carbonate 2–3 ml t.d.s. to bind intestinal phosphate. Symptomatic hypocalcaemia with serum Ca <1.7 mmol or ionised Ca <0.7 mmol/l may be treated with infusion of calcium gluconate 10% at 0.5–1 mmol/kg per 24 hours. Treatment of hyperkalaemia (see p. 214) may also be instigated. Whilst these are very sick and often unstable infants, regular weighing 12–24 hourly is essential in order to gauge fluid gains and losses. The same weighing scales should always be used.

If the condition is not improving and there is severe fluid overload, hyperkalaemia, acidosis or other electrolyte disturbances with uraemic central nervous system depression, despite the above interventions, dialysis should be considered (Boineau and Levy 1992).

Dialysis

Dialysis is the process of removing solute molecules by means of diffusion down their concentration gradients, via a semi-permeable membrane.

Whilst dialysis as a treatment of infants in renal failure is a rare occurrence in the NICU, it can be implemented by three methods:

- Haemodialysis: this is not really an option for most infants due to lack of suitably sized machines and their availability outside highly specialised centres.
- Peritoneal dialysis (PD): this is the commonest and most easily available technique.
- Continuous haemofiltration (CH): this is a relatively recent development in the neonatal population.

PD and CH will be further described.

Peritoneal dialysis

PD is useful in management of renal failure and is probably the technique most commonly available in most NICUs. PD works by the process of diffusion and ultrafiltration across the semi-permeable peritoneal membrane (see Figure 10.1).

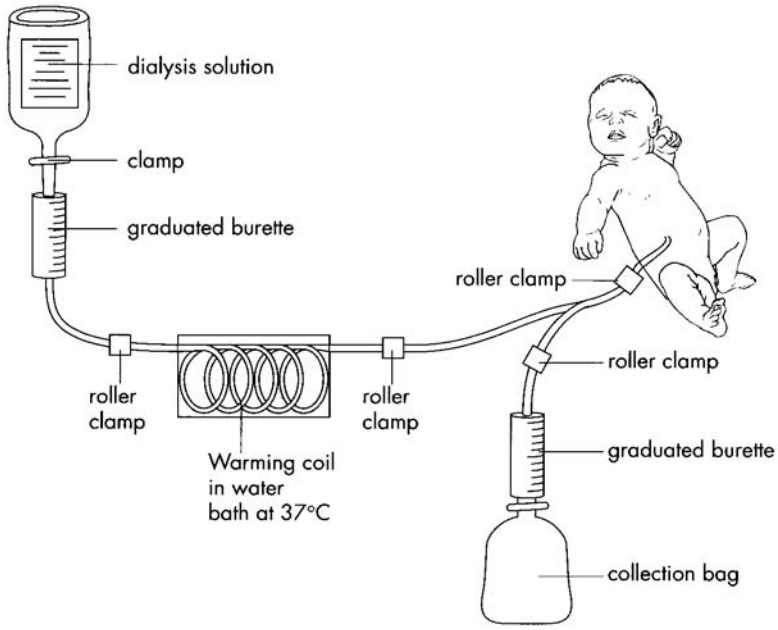

Figure 10.1 **Diagram of an infant receiving peritoneal dialysis**

A soft catheter is inserted into the peritoneal space, following systemic and local analgesia, and securely fixed in position with sutures and carefully applied tape to reduce potential skin damage. The site of insertion should be visible at all times and observed for fluid leakage, which can affect skin integrity, inflammation and signs of infection. Once the catheter is deemed patent the fluid cycles can commence. Cycle frequency, dwelling time and volume are dependent on the infant's toleration of the procedure.

A typical dialysis prescription is: 15–40 ml/kg volume of fluid infused over 10 minutes, dwelling for 35 minutes and draining for 10 minutes (Modi 1999).

Nursing management of an infant undergoing PD is focused upon prevention of complications of the procedure. These are peritonitis, fluid overload or dehydration, thermal instability and respiratory compromise. To prevent these occurring, a sterile technique should be adopted whenever changing fluids or PD circuitry. The drained PD fluid should be observed for turbidity, and additionally sampled daily and sent for culture and sensitivity. Fastidious attention must be paid to fluid balance recording of cycle volumes both in and out to establish whether the infant is in a positive or negative balance state. The circuitry should be well secured and supported, and not subject to kinking, which may impede flow either in or out.

PD fluid should be warmed by the use of a blood warmer set at 37 °C, so that dramatic cooling does not occur from cold dialysate being instilled.

Impairment of the infant's respiratory status can occur during cycles, due to increased intra-abdominal pressure causing diaphragmatic embarrassment. This

can be lessened by reducing the cycle volume, and nursing the infant in a pronounced head-up tilt position to reduce the infra-diaphragmatic pressure.

Continuous haemofiltration

Unlike PD, this requires vascular access in order to be commenced, via either the umbilical venous and arterial routes or a double lumen central venous catheter.

CH is a convection-based extracorporeal therapy, which involves a haemofilter constructed of semi-permeable membranes within a circuit of tubing (see Figure 10.2). Because the extracorporeal blood volume will be in most cases 10–20 per cent of the infant's blood volume, priming of the lines with heparinised saline (5000 IUI/l), followed by whole blood, will help the infant tolerate the connection process more readily. It is also prudent to have fluid for volume replacement immediately accessible in order to correct any precipitate falls in blood pressure during the process.

As blood flows through the haemofilter, ultrafiltrate moves across the semi-permeable membranes, mimicking the process of glomerular filtration (Dudley and Sherbotie 1992). CH provides slow, continuous and gradual removal fluid and electrolytes without adverse effects on the haemodynamic status (Dudley and Sherbotie 1992; Bonilla-Felix *et al.* 1998). It is driven by the infant's blood pressure, which needs to be in the region of 45 mmHg systolic to be

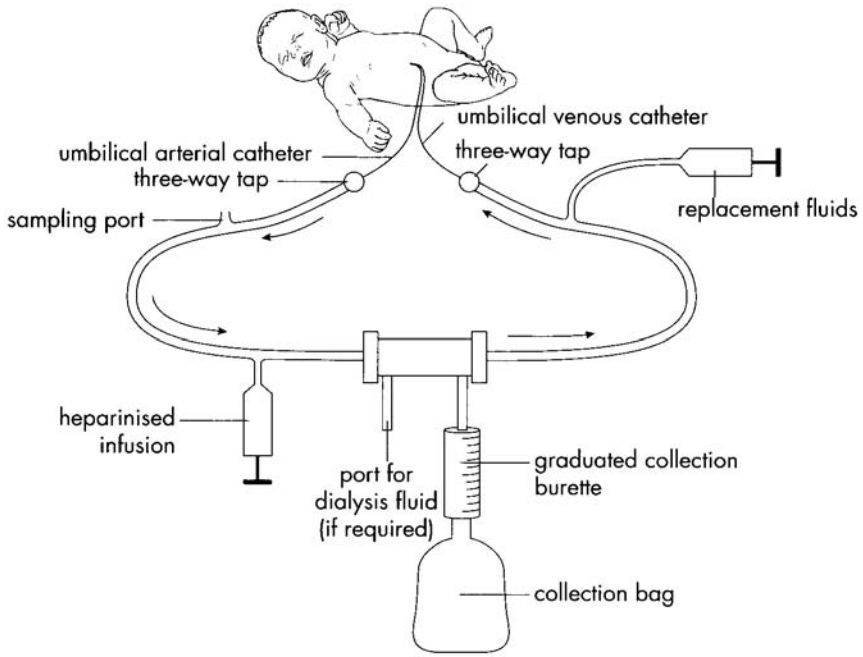

Figure 10.2 **Diagram of an infant receiving continuous arteriovenous haemofiltration**

adequate to sustain the flow. Infants may well require inotropic therapy (see p. 139) to achieve this.

Whilst the nursing management from fluid balance, infection and temperature perspectives is not that dissimilar to the infant undergoing PD, the risk of bleeding is a major consideration in these infants. Due to the extracorporeal nature of the circuit, significant heparinisation is usually required to prevent clotting within the circuit and filter. Careful observation of the skin for petechiae and for oozing needs to be undertaken. Procedures that can create trauma to tissue, for example oral or endotracheal suction and removal of sticking tape, should be carefully undertaken and kept to a minimum. Infants should not have intramuscular injections and blood sampling should be undertaken via the circuit rather than venepuncture. Clotting studies should be undertaken regularly to ascertain how much heparinisation is required.

The filter needs to be observed constantly for the flow through it. Flow ceasing abruptly suggests that the filter is blocked and needs to be changed.

Whilst in 1995 Coulthard claimed that many clinicians agreed that PD was the preferable technique, Bonilla-Felix and colleagues (1998) state that CH is now their first choice for dialysis of infants. Irrespective of which technique is chosen, the prognosis for these infants remains poor, usually due to the severity of the underlying disease processes. It is for this reason that the decision to undertake dialysis should only be taken following careful deliberations with the family regarding the infant's probable outcome, prior to commencement of either of these techniques (Coulthard and Sharp 1995; Coulthard and Vernon 1995; Modi 1999).

Conclusion

Whilst fluid and electrolyte balance may not appear as exciting or demanding as some of the newer technologies present within the NICU, maintenance of the fluid and electrolyte homeostasis has a significant role in the progress and subsequent outcome of infants. This has never been more apparent than in recent times with the management of increasingly preterm infants who, with their exquisitely fragile skin coupled with organ immaturity, make fluid and electrolyte therapy a very great challenge.

Maintaining stability and detection of potential (and real) problems is a pivotal role of the nurse and should not be underestimated.

Case study 1: fluid and electrolyte disturbances in a preterm infant

Stephanie was born at 24 weeks' gestation weighing 680 g. She is now 36 hours of age. Stephanie has been ventilated since birth for respiratory

distress syndrome and has required both albumin and bicarbonate infusions to correct hypotension and acidaemia. She is also on phototherapy for hyperbilirubinaemia which is due, in part, to extensive bruising sustained at delivery.

Her current fluid intake is 75 ml/kg of 10% dextrose infusion. Her most recent serum electrolyte result is:

Sodium	158 mmol/l
Potassium	3.6 mmol/l
Urea	7.2 mmol/l
Creatinine	90 µmol/l

Q.1. What does this result suggest?

Q.2. What are the factors that may have contributed to this state?

Q.3. What other parameters should be considered in conjunction with the above result?

Q.4. What should the plan of action include for Stephanie?

Case study 2: fluid and electrolyte disturbances in a term infant

Edward is a 3 kg male infant delivered by emergency cesarean section at 38 weeks for fetal distress. He had respiratory depression at birth and required resuscitation by intubation and positive-pressure ventilation.

Following admission to the NICU, he developed persistent seizures which required ventilation and anticonvulsant therapy. He is currently prescribed 60 ml/kg of 10% dextrose infusion.

A diagnosis has been made of grade three hypoxic ischaemic encephalopathy following birth asphyxia. The following results are obtained:

Sodium	122 mmol/l	Urea	2.0 mmol/l
Potassium	3.9 mmol/l	Creatinine	55 µmol/l
Calcium	2.0 mmol/l	Plasma osmolality	275 mosmol/l
Magnesium	0.7 mmol/l	Urine osmolality	320 mosmol/l
Phosphate	1.6 mmol/l	Glucose	2.0 mmol/l

Q.1. What are the areas of concern within this result?

Q.2. What are the contributing factors to this situation?

Q.3. What plan of action is necessary to correct this situation?

Q.4. What observations does Edward require?

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Chapter 11

Chapter 11

Nutritional Management of the Infant in the NICU

Contents

Introduction

Nutritional management of the preterm or sick term infant is essential for survival and an optimal outcome. Nutritional needs of the preterm infant differ from the term infant, but they also differ for each infant according to the infant's gestation, degree of growth retardation, postnatal age and accompanying disease (Steer *et al.* 1992). Nurses are faced with many challenges as they aim to meet the nutritional goals of the infant in their care. At the same time, nurses need to decide on the most appropriate method of feeding according to the individual infant's stage of development. There remains an uncertainty about feeding preterm infants despite a 50-year record of research in nutrition in the preterm infant. This uncertainty comes from a lack of knowledge on how preterm infants are fed in terms of their long-term outcome (Lucas 1993).

This chapter aims to examine the available evidence in support of meeting the individual infant's nutritional goals. It is anticipated that nurses will use the evidence to individualise their approach to feeding the neonate in intensive care and to support the family in their choice for feeding. Nurses need to consider the ability of the infant to feed or suck together with the environment in which such feeding occurs. The importance of the mother-infant nursing dyad needs to become part of the nutritional management of infants in intensive care.

It is beyond the scope of this chapter to provide an in-depth coverage of nutritional requirements and breast feeding practices. These topics are readily available in a range of reference material. This chapter concentrates on the infant in the intensive care unit and how the nutritional needs can be met and the nurse's role in meeting these requirements.

The chapter reviews the development and function of the gastrointestinal tract to enable the reader to make an informed choice for initiating enteral feeds. A structurally intact and functioning gastrointestinal tract is extremely important for infant development and long-term survival. The outcomes of nutrition are explored, with particular emphasis on the assessment of adequate nutrition evidenced by optimal growth and development and clinical outcomes.

The challenges of feeding the sick infant are examined with reference to current trends and research. What are considered appropriate nutritional requirements, how they can be provided and parental choices are examined. Particular emphasis on the available research for feeding methods is a major focus of the chapter. Problems associated with gastrointestinal dysfunction, immaturity and associated diseases are included. It is anticipated that nurses will use a problem-solving approach when they consider feeding an infant in the intensive care unit.

The chapter concludes by considering the important role of families, particularly mothers, in meeting the nutritional goals of their infant.

The gastrointestinal system

The gastrointestinal tract's role in ingestion, digestion and elimination is important for long-term growth and survival in infants. Development occurs in utero and the tract is structurally prepared for oral feeding by 20 weeks' gestation; however, many of the functions required for successful feeding are not fully developed. By 38 weeks the gastrointestinal tract is sufficiently mature and ready to meet the nutritional demands following birth (Weaver 1992). After birth, the newborn's gastrointestinal tract takes over from the placenta in the task of assimilation of nutrients. In healthy full-term infants this transition proceeds smoothly. When the function is impaired, however, owing to disease such as infection, shock or hypoxia, or extreme prematurity, the results may be a delay in gastric emptying and intestinal peristalsis, resulting in impaired digestion and absorption.

The most critical period of embryonic development of the gastrointestinal tract is from around 15 days to 60 days. During this time there is a period when the development may be affected by teratogens. Birth represents a challenge and demands several responses from the gastrointestinal tract, including coordinated sucking and swallowing, efficient gastric emptying and intestinal motility, regulated salivary, gastric, pancreatic and hepatobiliary secretion, effective absorption, secretion and mucosal protection. The major functions of the gastrointestinal tract are digestion, absorption of nutrients and water, and as a protective barrier against infection (Weaver 1992).

Successful transition to extrauterine nutrition requires the gut to be able to function rapidly and efficiently. Two factors are important in this transition: first, the gestational age of the neonate, and secondly, the composition of the food the neonate receives after birth. Human milk and colostrum contain all the nutrients for healthy growth and development, including ment of the newborn term infant (Lucas 1993), and is the preferred milk. It may, however, be substituted with artificial formulas. Human milk is the feed of choice for preterm infants but may need fortification. Should human milk be unavailable, then a specialised preterm formula should be used. Whatever the source, it remains important that the infant receives the appropriate nutrients in appropriate quantities for the appropriate stage of development.

Studies have shown that there is a gestationally dependent pattern of development of small intestinal motility. Increasing gestational age results in an increase of the migrating motor complex which is responsible for the forward movements of nutrients (Berseth 1996). There are different types of motor activity during feeding and fasting. During fasting, few infants display a migrating motor complex. Instead, episodes of non-migrating phasic activity of clusters of contractions followed by an absence is seen. Normal motor activity following feeding, despite an immature small intestine, suggests that infants can respond to enteral nutrition before complete maturation of their gastrointestinal motility, thereby accelerating motor development of the preterm gut (Berseth 1996).

Gastric emptying is delayed in preterm infants as compared with term infants; this may also reflect immaturity of motor function and a lack of coordination between the antrum, fundus, pylorus and duodenum. Intestinal transit is also slower in preterm infants with a range of 8–96 hours compared with 4–12 hours seen in adults (Berseth 1996). The varying times for the transit through the gastrointestinal tract can be an important factor in deciding an appropriate feeding regime for the neonate with special needs.

In the first 3–6 months of life the gastrointestinal function is immature, which places the newborn infant at risk for a variety of infectious and non-infectious diseases. Within a few hours of birth, the neonate is faced with antigens from both the diet and microbial sources. Following birth, mucosal protection can be divided into non-immune, which includes the luminal and mucosal defence mechanisms, and immune defence mechanisms, which include both humoral and cellular immune responses which are activated in response to specific antigens that are present in the lumen or surface of the gastrointestinal tract (Weaver 1992). Non-immune defence mechanisms include gastric acid, motility, pancreatic secretions, breast milk, mucus and a microvillus membrane. Immune defence mechanisms include secretory IgA, **macrophages, lymphocytes** and leucocytes.

Despite the immaturity of many of the digestive mechanisms, the well neonate is able to compensate to achieve adequate digestion of nutrients (Hamosh 1996). Sugars present in human milk and formula are assimilated by both small intestinal digestion and, especially in the case of lactose, colonic bacterial fermentation (Kien 1996). Diarrhoea may be caused by the malabsorption of sugars in the small bowel. Carbohydrate absorption is limited due to a deficiency of lactase that is reduced in the preterm infant to less than 30 per cent of that in the term infant. Gradual introduction of enteral feeds may enhance lactase production, so avoiding the osmotic diarrhoea of sugar malabsorption (Kien 1996). Therapy to reduce diarrhoea may involve slowing of motility, by using a prokinetic agent to facilitate fermentative activity. However, there have been studies which have shown that there is no benefit for some agents given intravenously (Stenson *et al.* 1998). Immaturity of other organs poses additional problems for nutrition. For example, reduced renal sodium conservation may necessitate large sodium supplements. The preterm infant is also at risk for deficiencies of some amino acids that may be achieved through supplementation.

The nutritional requirements, calories, vitamins and minerals and water are supplied by the breakdown of food through digestion and absorption. Digestion and absorption involve complex mechanisms that may differ from the adult and between the preterm and term infants. These include both inefficient protein and fat absorption. Infants who have had intestinal damage following necrotising enterocolitis or who have a prolonged ileus with a resulting atrophy may have a disaccharide intolerance and yet absorb glucose polymers. These variations between the term and preterm infant lead to caution when commencing enteral feeds in very small and preterm infants.

Illness and some diseases can inhibit the function of the gastrointestinal tract thereby making it difficult for nutritional goals to be achieved. The

administration of nutrients using the vascular system has been in clinical practice for more than 30 years. However, there is only limited data derived from randomised controlled trials to support the use of parenteral nutrition in the newborn (Heird 1992). Nevertheless, if the gastrointestinal tract is unsuitable for normal nutritional function due to illness or disease, then alternate routes need to be utilised.

Functional problems of the gastrointestinal tract may occur which can limit the ability for achieving nutritional requirements by the enteral route. One of the more common problems is gastro-oesophageal reflux (GER), which has been described in 50 per cent of healthy infants at the age 2 months (Novak 1996). The contributing pathogenesis has been identified as a decrease in lower oesophageal sphincter tone, transient sphincter relaxation, increases in intra-abdominal pressure, and delayed gastric emptying. One study (Newell *et al.* 1989) found GER was reduced during mechanical ventilation in neonates, but more studies are required before this is considered normal. Other functional problems, although rare, may be due to congenital abnormalities of the gastrointestinal tract such as oesophageal atresia or stenosis, pyloric stenosis, extrahepatic biliary atresia and malformation of the small and large intestines.

Outcomes of nutrition

The most effective method of determining whether nutritional goals have been achieved in the short term is growth. This may be assessed by measurement of weight, length, head circumference and skin fold thickness. The pattern of growth rate may influence the management of the infant and their length of stay in the NICU or step-down nursery. For graduates of intensive care it is important to know that their dietary management is appropriate as it may influence long-term growth, neurological development and subsequent illnesses such as allergies (Steer *et al.* 1992). Preterm infants seem no more prone to allergy than term infants (Klebanoff and Berendes 1988); a family history of atopy predisposes them in a similar way (Lucas *et al.* 1990). Lucas *et al.* (1998) propose that suboptimal nutrition during sensitive stages in early brain development may have long-term effects on cognitive function and they recommend that avoidance of under-nutrition in sick preterm infants is important to optimise neurodevelopmental outcomes.

Lucas (1993) describes an uncertainty that persists in clinical practice, despite a 50-year record of research in nutrition in the preterm infant. He goes on to identify the lack of knowledge on how preterm infants are fed, in terms of their long-term health, as a cause of this uncertainty. Lucas suggests that meeting the preterm infant's increased requirements may confer long-term benefits. However, this also poses a challenge for the clinicians caring for the neonate in intensive care. The increased requirements may be met by using either fortified breast milk, preterm formula or a combination of both for enteral feeding. There is evidence that human milk may have additional benefits in

terms of neurocognitive development (Lucas 1993). The choice of milk remains with the clinician, though the mother's choice for her infant must be taken into consideration in any decisions made regarding formula or milk substitutes. Involving the mother in informed discussions may be beneficial for all concerned.

Growth

The assessment of growth parameters remains one of the most practical and valuable tools to estimate the nutritional status in neonates. For full-term infants there are charts available to plot their postnatal growth. For preterm infants there are differing opinions as to whether to use intrauterine or extrauterine growth charts. Charts that are suitable for the appropriate clinical, demographic, ethnic and socioeconomic similarities of the population should be used.

Postnatal growth rates that are comparable to those of the fetus are the aim of nutritional management in the preterm infant. The majority of infants will lose up to 15 per cent of their body weight soon after birth, but can expect to regain their birth weights within 3 weeks of age. The normal intrauterine growth rate is 15–20 g/kg per day for an infant growing around the 50th centile; this is not often achieved in sick or preterm infants. Many studies have shown that rates of weight gain can exceed these rates by in excess of 50 per cent of the intrauterine growth trends (Heird *et al.* 1993). The higher growth trends appear to be associated with rates of fat accumulation in excess of the intrauterine rate. These may be useful in low birth weight infants as a support for their environmental adaptation. However, the effect of increased fat deposits on the long-term well-being of the preterm infant remains unclear.

Preterm infants are born with low body stores of nutrients that would normally accumulate in late gestation. After birth, preterm infants rapidly acquire a pattern of growth together with metabolic and nutrient handling skills which would not be seen at a corresponding postconceptional age in utero. Therefore, preterm infants need to be assessed for their adequate nutritional requirements based on their ability to adapt to their environment and any contributing disease factors that are present.

Growth patterns vary according to the population under review. There are various charts currently used in practice to help determine a neonate's nutritional status.

Postnatal head growth is a strong predictor of early developmental outcome in low birth weight infants and insufficient calories provided to small for gestational age (SGA) infants beyond the first 2 weeks of life may result in failure to initiate catch-up head growth (Georgieff *et al.* 1985). Adequate nutrition can be estimated by measuring the infant's growth through body weight, length and head circumference. The ponderal index, which is a calculation of body weight and length, gives an indication of the quality of the growth by relating the body weight to the overall length. Length and head circumference are

important to measure as they are indices of skeletal and organ growth, whereas weight may change due to fluid balance changes and fat disposition. However, these measures can be inaccurate and care needs to be taken to ensure consistency and skill in taking the measures.

The practicalities of weighing a baby in the NICU can be a source of concern for nurses caring for these infants. The reliability of weighing sick ventilated infants has not been evaluated and the possible harms of weighing the infant need to be balanced against the accuracy of the technique. The use of in-bed scales has not been adequately tested as a reliable method for assessing growth in sick and/or small infants who are ventilated. However, to provide a good estimate of the trends in the infant's weight the procedure of weighing needs to be meticulous. The infant should be weighed at the same time of the day and the same scales used. The accuracy of the scales should be checked regularly. A documented unit policy on the procedure may be beneficial in achieving some consistency, especially in intensive care, as the infant may have splints, drains and multiple lines to consider in the total weight.

Tudehope and Steer (1996) suggest that more desirable short-term outcomes of nutritional management may be clinical outcomes. Measurements of morbidity, which include rates of neonatal death, necrotising enterocolitis, infection, hypoglycaemia and bronchopulmonary dysplasia, may be useful in determining trends. Other measures would include gastrointestinal tolerance, biochemical measures of undernutrition, such as hypophosphataemia, metabolic acidosis and hyperaminoacidaemias. These clinical outcomes can provide a source of evidence that would allow clinicians to benchmark their practice with an aim to improve the nutritional management of infants in intensive care.

Assessment

Accurate assessment is an important aspect of caring for the infant in the NICU, and nutritional assessment and readiness to feed is a pivotal part of the routine assessment made by nurses.

Infants need to be observed for their readiness to feed and tolerance of enteral feeds as well as how they are achieving their nutritional goals. Obvious parameters of weight and corrected age are taken into consideration when making an assessment of the infant's readiness to feed. There is a trend these days to take into account the infant's behavioural organisation skills, including his or her ability to maintain a quiet alert state and to display clear engagement and disengagement cues. The infant is seen as a collaborator in their own care, and provides the best information base from which to design feeding routines (Als and Gilkerson 1997). Due to the wide adoption of the developmental care philosophy, more nurses are becoming proficient in reading the infant's cues and adjusting routines to suit the individual infant (see Chapter 2). By viewing feeding from the infant's perspective, nurses can optimise intake, prevent fatigue and reduce some of the dangers associated with feeding premature infants. The families can be supported and involved from the early stages of feeding so that

they can learn to recognise their infant's signs of stability or stress during feeding and intervene appropriately to promote the infant's self-regulation (Shaker 1999).

Part of the nutritional assessment is to determine if the infant is ready to feed and has effective suck and swallow coordination. Futile attempts to suck and swallow may be due to structural or functional defects, which need to be eliminated before continuing with feeding attempts (Korones and Bada-Ellzey 1993). Prematurity may contribute to the infant's inability to tolerate enteral feeds and attempts to oral feed should be delayed until the appropriate gestational age of at least 32–34 weeks (Tudehope and Steer 1996).

Once feeds have been commenced, the infant's tolerance of the feeds forms part of the nutritional assessment. In the NICU feed intolerance may indicate a worsening underlying disease or a stress response from noxious causes. Gastric aspirates have been used as an indication of feed tolerance in tube-fed infants. Kirkham (1998), in a randomised controlled trial, found the procedure of gastric aspiration had little influence on the hours of feed interruption. This could relate to the lack of consensus about what is an acceptable volume of aspirate to determine cessation or withholding of feeds. There is, however, no clinical evidence as to what constitutes an acceptable volume of aspirate that may indicate feed intolerance. Thus the monitoring of the trends in volumes of aspirate may be a more useful guide.

In Kirkham's study interruption to feeds was more likely to occur due to abdominal distension. However, the measurement of abdominal distension appears to be a practice based on ritual with no evidence that measurement using a tape is a reliable method. Indeed many texts will recommend the practice without a thorough evaluation of the technique. A more appropriate assessment would be to observe and gently palpate the abdomen to determine an increase in tension and resistance.

The signs of a soft abdomen, the presence of bowel sounds, the passage of stool and minimal gastric aspirates indicate a functioning gastrointestinal tract. Milk feeds may not be tolerated in some infants and when attempts are made to feed there are increasingly large gastric aspirates and/or vomiting. The use of a prokinitic agent such as metoclopramide may improve feed intolerance by facilitating gastric emptying and motility. Other drugs such as cisipride have been used for feed intolerance, although a systematic review of its use showed there is a lack of conclusive evidence as to its effectiveness from proper scientific methodological trials (Premji *et al.* 1997). Thus feed intolerance can be a difficult problem when trying to establish enteral feeds. Patience and perseverance on behalf of the carers and an ability to interpret the infant's cues as to his or her readiness to feed can aid the eventual success of establishing and maintaining feeds.

Feeding the NICU infant

Feeding the preterm infant is not a natural physiological process. None of the current dietary regimes can mimic the transplacental process. Individualisation

of nutritional care is important. Two factors are important in the postnatal adaptation to enteral feeding: the gestational age of the infant and the composition of feeds the infant receives. When feedings are delayed, so is the process of intestinal colonisation, leaving the infant more susceptible to enteric pathology (Yellis 1995).

Requirements

Caloric requirements for full-term infants are based on measurements of basal metabolic rates and estimates of calories required for physiological functioning. Basal metabolic requirements include energy for thermoregulation, respiration, cardiac functioning and cellular activity. Energy requirements increase in disease states such as fever, sepsis and hypoxia. Preterm infants have higher metabolic and growth rates and an increase in insensible water losses, leading to an increase in energy and nutritional requirements to support their higher growth rates. When the infant requires intensive or special care there is often an increase in their energy expenditure; therefore when providing care nurses should aim to promote energy conservation and growth by implementing practices such

***Table 11.1* Effects of neonatal diseases on specific nutrient requirements**

			CHD			
Nutrient	*RDS*	*BPD*	Cyanotic	CHF	*Sepsis*	*IUGR*
Free H_2O	↓	↓	↔	↓	↔	↑
Energy	↑	↑↑	↑	↑↑	↑	↑
Fat	↔	↑	↑	↑	↔	↑
CHO	↑	↓	↑	↑	↑	↑
Protein	↔	↑	↑	↑	↑↑	↑
Calcium	↔	+↑	++↑	++↑	↔	↑
Iron	↔	*↑	↑	↔	↓	↑
Vitamin A	↑*	*↑	↔	↔	↔	↔
Vitamin E	↔	↑	↔	↔	↔	↔

RDS, Respiratory distress syndrome; BPD, bronchopulmonary disease; CHD, (congenital) heart disease; CHF, congestive heart failure; IUGR, intrauterine growth retardation; CHO, carbohydrate.
* = in <1500 g infant.
\+ = especially if on calciuric diuretic (furosemide).
++ = especially postoperatively.

Source: Wahlig and Georgieff 1995, reproduced with permission

as minimal handling, maintaining a neutral thermal environment and providing developmentally supportive environmental care (Darby and Loughead 1996).

An optimal diet for preterm infants is one that supports growth at intrauterine rates, without imposing stress on the infant's immature metabolic and excretory functions (American Academy of Pediatrics 1985). Goals for nutritional support during the first few days of life are the maintenance of fluid status, glucose homeostasis, and normal serum electrolytes and mineral concentrations. Extremely premature infants have decreased body stores of nutrients and, consequently, a limited capacity to tolerate starvation, especially if coupled with metabolic demands imposed by illness (Pereira 1995).

The nutritional requirements for the healthy term infant have been fairly well defined. There is less evidence available, however, on the nutritional requirements for the preterm or sick infant requiring intensive care. Metabolic changes occur in acute and chronic disease and these will have an effect on the nutritional requirements and management. Wahlig and Georgieff (1995) have translated this information into recommendations for sick and physiologically unstable infants (Table 11.1).

The resting energy expenditure varies from infant to infant and will be dependent on factors such as environmental temperature, activity, diet and rate of growth. In enterally fed infants the daily energy expenditure averages 50–60 kcal/kg, with slightly less expenditure in parenterally fed infants. This difference has been attributed to smaller losses from excreta and possibly reduced expenditure.

Parenteral nutrition

Parenteral nutrition may be used as a supplement to enteral nutrition or it may be used exclusively. It has become a popular choice for providing nutrients to the infant requiring intensive care. It may be administered using a variety of routes, the most popular being via a percutaneous central venous line inserted in the NICU. Units that have facilities for providing paediatric anaethesia may use central venous catheters inserted subcutaneously into a central vein.

Amino acids administered parenterally in conjunction with sufficient non-nitrogen energy were found to improve weight gain in low birth weight infants (Heird 1992). Practices have tended to provide maintenance amounts of amino acids in the first few days of life increasing gradually for optimal growth. There are no studies to support this practice, thereby indicating an area for further research.

Heird (1992), through a review of the available clinical trials, has shown that parenterally delivered nutrients support normal to supranormal rates of growth. He recommends daily amino acid intakes of 3 g/kg and energy intakes of 80–90 kcal/kg for intrauterine rates of nitrogen retention and weight gain. The pattern of the amino acid mixture is an important

determinant of the efficiency of the total mixture. Lipid infusion plays an important role in preventing fatty acid deficiency and in ensuring sufficient non-nitrogen calories without exceeding glucose tolerance. Rates of 2–3 g/kg daily appear to be well tolerated when given by continuous infusion. Van Beek *et al.* (1995) recommend starting parenteral feeding in preterm infants, including amino acids and fat as soon as possible after birth; however, they do recommend caution in infants with sepsis, respiratory disease or extreme prematurity. Preterm infants have poor tolerance of both glucose and fat when infused above physiological levels (Brans *et al.* 1988; Jones *et al.* 1993), therefore nurses need to ensure that the infusion rate remains constant and avoid the temptation to 'catch-up' with volumes when the infusion has been disrupted for whatever reason.

Despite the available research and evidence for the use of parenteral nutrition for neonates there remain many unanswered questions (Heird 1992). These questions are important for clinicians when making decisions for the use of parenteral nutrition in the NICU. Issues such as cost-effectiveness, complications of routes of administration, effect on morbidity, ratio of energy to amino acid requirements, and the relationship of early enteral feeding and necrotising enterocolitis are a few that need to be considered.

When parenteral nutrition is used in the NICU specific care is required to ensure the complications associated with its use are avoided. Infants should be monitored regularly for their fluid intake and output, their tolerance of dextrose by measuring the blood glucose, and their tolerance of lipids by monitoring plasma turbidity, either by observing a sample of settled blood or spinning a sample of blood. If signs of turbidity persist, then a sample should be sent for analysis in the laboratory. Other complications include sepsis, metabolic disturbances and catheter-related problems.

Enteral nutrition

Human milk is the preferred diet for enteral feeds. There is wide variation in the nutrient and non-nutrient content of the milks currently available. Formulas designed for preterm use differ in composition from milks designed for term infants. Human milk also varies in composition during the course of lactation, diurnally and during a feeding. The milk from mothers who have delivered prematurely has a different composition from mothers who have delivered at term. If formula is to be used for term infants then standard formulas based on cow's milk with a protein concentration of 13–15 g/l provide the best nutritional source (Atkinson 1992).

Atkinson (1992) found that there is a lack of properly controlled randomised trials concerning optimum infant nutrition. Based on a comprehensive review of the literature, Atkinson states that breast feeding will be most successfully achieved in mothers who receive adequate education and support from knowledgeable health professionals. The breast-fed term infant can be adequately nourished for the first 6 months of life by breast milk alone. Supplementation is only required if the maternal diet is inadequate, resulting in malnutrition. This

becomes an important goal for infants requiring intensive care. Mothers need to be supported as they express their milk, often for many weeks, to provide an opportunity for breast feeding once their baby is able to coordinate sucking and swallowing. This applies to the preterm infant as well as the disorganised term infant.

Early nutrition of low birth weight infants can influence later neurodevelopment (Lucas 1993) so it remains vital that early nutritional needs are met. Whereas breast milk supplies adequate nutrients to meet the nutritional demands of the term infant, the nutrient component of premature human milk provides insufficient quantities to meet the estimated needs of the premature infant. Fortification of human milk with multicomponent supplementation has been used to increase the rate of growth in very low birth weight infants. Kuschel and Harding (1998) undertook a systematic review which demonstrated that human milk fortification with more than one nutritional supplement resulted in a small gain in weight, linear growth and head growth in the short term. They found no long-term advantages in terms of growth or neurodevelopmental outcome. Recommendations for further research were made to compare the proprietary products currently used for both short- and long-term outcomes.

Methods

Feeding practices vary between clinicians and institutions, although McClure *et al.* (1996) found there is a tendency towards greater uniformity in feeding practices. It remains an issue for nurses, however, who often draw on their experiences when initiating and planning the feeding regime for infants in their care. Feeding the infant in the NICU needs to be planned on an individual basis taking into account the infant's illness, development and family presence. McCain (1997) found in her study that awake behaviour dominated in the successful feeders and concluded that awake and quiet behaviours represent optimal feeding states. Therefore, before initiating a feeding regime nurses need to integrate more assessment of infant behaviour state in determining feeding readiness.

An effective route and schedule of feeding the low birth weight infant is an important part of the nutritional management. Infants who are less than 32 weeks' gestation do not effectively suck, lack coordination of sucking, swallowing and breathing, and have delayed gastric emptying (Cavel 1979). Evaluation of sucking behaviour may be helpful in identifying those infants who are disorganised or poor feeders (Medcoff-Cooper *et al.* 1993).

It was thought that non-nutritive sucking (NNS), by providing sucking opportunities on a pacifier before, during or after tube feeding, might accelerate the sucking reflex, enhance gastric emptying and stimulate intestinal transit and improve digestion.

Studies have shown no difference in respect to energy balance (DeCurtis *et al.* 1986), fat absorption or nitrogen balance. Furthermore there is no effect on gastric emptying and motility in term infants (Szabo *et al.* 1985), nor on gastric residual volume after 3 hours (Widstrom *et al.* 1988). Field *et al.* (1982)

found no difference in feeding performance, regurgitation, volume of intake, or feeding time in infants who were offered non-nutritive sucking opportunities. On a positive note, non-nutritive sucking did decrease the number of days to initiation of nipple feeds (Measel and Anderson 1979; Field *et al.* 1982) and the number of days in transition from gavage to full oral feeds (Bernbaum *et al.* 1983). This may be due to acceleration in the maturation of the sucking reflex (Bernbaum *et al.* 1983). A systematic review of 19 studies on non-nutritive sucking in premature infants (Pinelli and Symington 1998) found a significant decrease in the length of hospital stay, and that NNS appeared to facilitate the transition to full oral/bottle feeds as well as facilitating behavioural state. Therefore, based on the available evidence there does seem to be some benefit in support of NNS in premature infants, although many of the claimed benefits of effect on weight gain, energy intake, oxygen saturation, intestinal transit time, time to full oral feeds and energy expenditure remain unproven. The use of a pacifier may be beneficial, however, as a self-consoling strategy by promoting calming behaviour in an infant unable to take sucking feeds (see p. 245).

Another popular feeding practice is for minimal enteral nutrition in parenterally fed neonates. Tyson and Kennedy (1998), in their systematic review, found the clinically important effects of minimal enteral nutrition were difficult to determine and concluded that it remains unclear whether minimal enteral nutrition is beneficial in high-risk neonates.

Once a decision has been made to commence enteral feeds the frequency and volume of the feeds need to be taken into consideration. The immediate feeding of low birth weight infants soon after birth (within 2–4 hours) has resulted in more aspiration and death (Wharton and Bower 1965). This serves to reinforce the need to consider each infant and their underlying disease when initiating enteral feeds. Wu *et al.* (1967) found symptomatic hypoglycaemia is reduced with early enteral feeds, and the rapid advancement of enteral feeds has shown a reduction in the incidence of hypoglycaemia (Russell and McKay 1966). Most of these early studies tended to demonstrate adverse events, but as clinical practice has changed over time many of the practice changes have not been evaluated in relation to adverse events of feeding sick and premature infants. Therefore clinicians need to use the evidence from these early studies to remind them of the caution required when feeding small or sick infants. The use of parenteral nutrition in combination with the slow introduction of enteral feeds may avoid some of the problems previously identified.

There is little evidence evaluating the benefits and risks of contrasting policies on volume of feeds or concentrations of formula (Steer *et al.* 1992).

Mandich *et al.* (1996) found that apnoea appears to influence the length of time it takes for a premature infant to begin receiving full oral feedings. This may be due to the frequency and the pattern of grading of feeds. Demand-fed infants have been shown to require fewer gavage feeds and were able to be discharged sooner (Collinge *et al.* 1982). The degree of maturity has been shown to influence the transition time to bottle or breast feeding. This is more likely to be successful after 34 weeks' gestation, and if the infant is given more opportunities to breast feed (Meier and Riordan 1985).

Infants requiring intensive care are more likely to commence enteral feeds via a tube. There is little consensus about the ideal method to provide tube feeding. Popular methods have been via intermittent gastric feeds, continuous transpyloric and continuous gastric tube feedings. The placement of the gastric tube, either orally or nasally, has also been part of the debate on appropriate methods (see p. 286).

The use of either the orogastric or nasogastric route for tube placement has been studied by several researchers. Van Someran *et al.* (1984) found a decrease in the incidence of central apnoea and periodic breathing in infants with orogastric tubes. There was, however, no difference in obstructive apnoea. Studies have shown that infants with nasogastric tube placement have lower minute ventilation and tidal volume during continuous and intermittent sucking (Shaio 1994). Therefore, there appear to be less benefits for the use of nasogastric tube placement, although depending on the individual infant the placement of gastric tubes nasally may be more suitable. None of the studies compared different tube sizes and this may be significant for some smaller infants or those with underlying respiratory problems.

In a survey of current practices Shiao and Difore (1996) found that both orogastric and nasogastric tubes are used in clinical practice in 66 per cent of NICUs. The reasons cited for choosing a particular route were often associated with unit policy or tradition. The feeding stage of the infant tended to determine the practice regarding continuous tube placement. Practices of intermittent placement were less common. The preferences of physicians and nurses were used in several units. In practice, tube sizes varied from 5 to 8 French, and 73 per cent of nurseries had a unit policy regarding tube use. Weibley *et al.* (1987) found that there were errors in tube placements for two standard techniques. This is a concern if the complication of aspiration is to be avoided in the preterm infant. There appears to be no evidence from clinical trials to support practices regarding tube placement.

Intermittent (or bolus) feeds into the stomach at intervals from hourly to 4-hourly have been used for feeding premature infants for many years. There has been debate in practice as to whether the tube should be left in situ or removed after each feed. Frequent insertion and removal of tubes may be associated with vagal stimulation that can result in frequent apnoea and bradycardia in small or sick infants. Continuous gastric tube feedings have been used for small sick infants when intermittent feeds have been poorly tolerated. Studies comparing the two methods have shown no difference in weight gain or in length, head circumference or skin thickness (Toce *et al.* 1987) and very low birth weight infants had similar growth, macronutrient retention rates and comparable length of stay (Silvestre *et al.* 1996). Continuous feeding may be preferable for immature infants with delayed gastric emptying, but practice is more likely to be determined by the individual infant's needs and the preference of the clinicians. Care needs to be taken when using human milk with continuous tube feeds. Fat losses can occur due to the settling of the milk; the syringe needs to be rotated or carefully positioned so rising fat is advanced down the tube.

Another alternative to gastric feeding is transpyloric feeding where a tube is passed through the pylorus into the distal end of the duodenum or jejunum (see p. 287). This method was popular in the 1970s and is not used as often today. The procedure is technically more complex in that a tube cannot always be passed and may result in additional X-rays to verify tube position (Laing *et al.* 1986). Major complications of tube feeding, such as aspiration, necrotising enterocolitis and diarrhoea, are not reduced by using transpyloric tube feeds and the death rate increased in some instances. Steer *et al.* (1992) state that transpyloric feeding should not be used as a routine method of tube feeding small preterm infants.

Other methods of providing enteral feeds can include the use of a gastrostomy tube in specific cases. Studies have shown that in infants receiving gastrostomy feeds death was more frequent before 21 days (Vengusamy *et al.* 1969). Routine gastrostomy feeding should not be used in very low birth weight infants (Steer *et al.* 1992).

A particular challenge for nurses in the neonatal nursery is the problem feeder. Once enteral feeds have been commenced several problems may arise that need to be considered and potential complications avoided. Gastrointestinal reflux has been identified as a fairly common occurrence (Novak 1996) and can be very distressing for the infant, mother and caregiver trying to establish oral feeds. The prone position has been recommended (Vandenplas and Sacre-Smits 1985) to reduce the effects of vomiting. The use of a pacifier should be avoided as the incidence of reflux has been shown to be significantly increased (Orenstein 1988). Recent data indicate that oesophageal reflux may be less and of shorter duration in infants who are breast fed compared to formula fed (Tan and Jeffery 1995).

The lack of normal feeding behaviour during critical periods may result in the loss of the ability to feed (Skuse 1993). Therefore encouragement with oral stimulation and pleasant sensations may help the infant to adjust. Very low birth weight infants should be monitored during feeding or breathing pauses, as bradycardia and desaturations have been reported when a nasogastric tube is in place (Shaio 1994).

Specific problems in the NICU

Respiratory disease is a common problem in the NICU and infants with an acute respiratory illness require an appropriate nutritional regime due to their increased metabolic demands. Oxygen consumption and therefore energy expenditure is increased (Wahlig and Georgieff 1995). Preterm infants are frequently ill, and this may be a major factor on the practical management of feeding and on nutritional requirements. Infants with respiratory disease often tolerate enteral feeds poorly and feeds may need to be delayed or kept at minimal levels until they have overcome the severity of the disease.

Infants with cardiac disease or failure may require restricted volumes, making it difficult to achieve their nutritional requirements. These infants pose

particular difficulties when it comes to establishing a feeding regime as they often require increased energy intake. A challenge of feeding these infants is to maintain their extra calories while at the same time reducing the energy expenditure of feeding (Wahlig and Georgieff 1995). Complementary feeds of breast and tube may be necessary to ensure the infant receives adequate rest following feeding episodes.

Infants with bronchopulmonary dysplasia need more energy and a relatively low carbohydrate to fat ratio to reduce carbon dioxide production and retention (Steer *et al.* 1992). Ryan (1998) recommends increasing the energy density of the feeds so that sufficient energy is delivered in tolerable volumes.

Lucas and Cole (1990) found that necrotising enterocolitis was more likely to occur in infants who were exclusively formula fed when compared to infants receiving some expressed human milk (EHM). They suggest breast milk has an important protective role and can be used to supplement formula-fed infants. Thus, nurses would be advised to use this knowledge to encourage mothers to express milk for their preterm infants so that it may be used for the early enteral feeds as a protection against necrotising enterocolitis and to aid in the neurocognitive development.

Neonatal surgery can have a significant effect on the feeding regimes and patterns in the NICU. Infants who have undergone gastrointestinal, or other major surgery may have their enteral feeds delayed for several weeks. How the feeds are commenced and graded up in volume is often at the discretion of the surgeon. However, nurses can contribute to the discussion and take an active role in the feeding plan to suit infant and family. There remain no hard and fast rules for postoperative feeding, but the resumption of intestinal motility and peristalsis play a vital role in the timing of the commencement of feeding. A more difficult problem concerns the measurement of tolerance and the grading to full oral feeds.

There are many infants admitted to the NICU where feeding routines play an important part in their recovery. It is the role of the nurse to develop a plan of care that includes the infant's nutritional goals. As part of the plan the mother's preferences and involvement in the feeding schedule are an integral part of the infant's recovery phase.

Family support

The ability to feed is a vital part of the infant's recovery from intensive care. Mastering oral feeding, by whichever method, is an important part of the maternal/infant interaction. Families often become frustrated at the difficulty and time involved in establishing a feeding routine. Nurses have an important role to play in alleviating parental anxiety and supporting the mother and infant while they establish their routines.

The delivery of a sick or preterm infant can be an anxiety-producing event, which may physically and psychologically impede the mother's ability to breast feed. Support from well-informed staff can alleviate some of these anxieties and help facilitate a successful breast feeding experience.

There have been several studies looking at mothers' intention to breast feed their infants when they are admitted to the NICU. Byrne and Hull (1996) found 64 per cent and Griggs *et al.* (2000) found 71 per cent of mothers intending to breast feed on admission. The breast feeding rates on discharge were 49 per cent; this drop may reflect their experiences in the NICU.

Byrne and Hull (1996) interviewed mothers whose infants had been in the NICU. From the interviews, they concluded that a feeling of tenseness rather than embarrassment inhibited lactation. The mothers requested information on breast feeding and expressing milk, comfort during their feeding attempts and facilities that support breast feeding. Nurses need to supply information on how to express milk and how to breast feed immature and sick infants. The information should be clear, simple and unequivocal and, most importantly, should be agreed by all who advise the mothers.

Nyqvist and Sjoden (1993) asked mothers about the advice they would give to the NICU staff to facilitate breast feeding for mothers and infants during their stay. In the study, the mothers identified an unsupportive environment in the NICU, the quality of advice given on breast feeding, work organisation practices and the behaviours of the nurses as inhibitors for the initiation of breast feeding. Mothers felt uncomfortable in the hectic and technological environment with other sick infants, nurses, other parents and equipment. Griggs *et al.* (2000) also found that mothers stated that they felt they were in the way of equipment or the staff were too busy attending other sick infants. Interestingly, the mothers suggested that it was mothers who should feed their infants and not the nurses. Practices may vary between units, but nurses need to consider ways of involving the families when feeding is commenced. For example, the feeding schedules could be agreed with the mothers and practical assistance may be offered to encourage the mothers to hold the syringe barrel during tube feeds.

In the same study the mothers advised other mothers to be persistent with their attempts to express and feed as early as possible. They also encouraged other mothers to maintain physical contact with their infant and not to let nurses take over the maternal role functions by feeding the baby (Nyqvist and Sjoden 1993). This advice is useful for nurses when planning ways of involving the mother in her infant's care and feeding routines.

Before the infant can take oral feeds, expressed human milk (EHM) can benefit both the infant and mother. Mothers have reported that seeing their infant receive their expressed milk by tube feeds reinforces their breast feeding efforts (Meier *et al.* 1993).

From the studies and interviews with the mothers of infants in the NICU nurses should be able to provide strategies to assist in the successful transition from parenteral to tube to oral feeds. The environment should be warm, comfortable and private (Byrne and Hull 1996). Meier *et al.* (1993) found the NICU environment influenced the mothers' ability to breast feed—mothers wanted to breast feed or express in a quiet room. They also did not want to be left alone during breast feeding. Griggs *et al.* (2000) found mothers requested opportunities to live in and be on call for feeding opportunities. The presence

of an experienced and supportive nurse would be seen to be of benefit to these already stressed and anxious women.

Tan and Jeffery (1995) identified several factors that may influence mothers' choice of infant feeding. These include urbanisation and poverty environments, working women, commercial influences, attitudes of health professionals, social attitudes and support. Mothers were not always aware of the benefits of breast milk—this may be due to health professionals not wishing to put pressure on mothers to produce milk (Byrne and Hull 1996). Mothers also identified inconsistent and poor advice as discouraging feeding opportunities (Griggs *et al.* 2000).

Infants admitted to the NICU come from a diverse range of cultural backgrounds. When initiating feeds the particular cultural traditions and beliefs may not be able to be met. Nurses working with the families need to be considerate and sympathetic to the possibility of cultural clashes. Western ideals may not be always acceptable, therefore special consideration needs to be given to instructing mothers regarding expressing their milk and breast feeding in a busy intensive care environment.

Conclusion

Providing adequate nutrition to infants in the neonatal intensive care unit presents a major challenge to the doctors and nurses caring for the infants. The goal of feeding is to meet the metabolic requirements of a number of the developing organ systems. Presently the adequacy of nutrition is measured by plotting the infant's growth. A challenge is to consider other ways of measuring the outcome. Assessment of the infant's readiness for feeds and the infant's ability to feed normally are a major focus of the nursing care in the NICU. Nurses are in powerful positions to influence how neonates attain their goals and to assist the infant-mother dyad. By using an evidenced-based approach to their practice, optimal outcomes for each individual can be achieved.

Much of the evidence on neonatal nutrition and feeding practices dates back several decades. Practices have evolved that are based on tradition, ritual and personnel preferences. Therefore neonatal nutrition remains a challenge for the neonatal nurse; obtaining the evidence to support practice and changes within practice is a task that reflects on history. It remains a challenge for all neonatal nurses to question rituals associated with infant nutrition and to search out the evidence for changes in their practice.

Case studies

The aim of these case studies is to assist you in applying the theory covered in this chapter to practice. The cases have been developed to contrast the differences, and in some instances the similarities, between the preterm and

term infant. It must be stressed that each infant in the NICU is an individual, and evidence is used to support the decisions for care. Nutrition remains an important aspect of intensive care and a challenge for nurses working with these unpredictable infants.

Case study 1: nutritional requirements of a preterm infant requiring assisted ventilation

Ahmed was born at 27 weeks' gestation. He was the first child of a young married couple. At birth, Ahmed breathed spontaneously and required minimal resuscitation. He was transferred to the NICU where he weighed in at 980 g. An arterial blood gas and chest X-ray indicated respiratory distress syndrome and he was intubated and ventilated. Ahmed required assisted ventilation for the first week of life, he was then extubated and nursed in a double-walled incubator and was receiving 25% oxygen via a head box.

During the course of his RDS Ahmed received two doses of surfactant. He required antibiotics for an episode of sepsis and he required volume expanders for a period of hypotension.

Ahmed's mother visited each day with her husband and family. During her visits she appeared anxious and often relied on the information concerning Ahmed's progress to be relayed to her by her husband.

Answer the questions below giving the rationale for your answer from the evidence supplied in the chapter.

Q.1. What would be the most appropriate method to supply Ahmed's nutritional requirements for the first week of his life?

Total parenteral nutrition
Parenteral nutrition with minimal enteral feedings
Parenteral nutrition with non-nutritive sucking
Enteral feeds of mother's expressed milk

Q.2. Which are the risk factors that may impact on Ahmed commencing enteral feeds?

Decreased motility
Delayed gastric emptying
Inability to suck and swallow
Mother unable to express enough milk

Q.3. Once Ahmed is to commence enteral feeds, what regime would be appropriate for him?

Continuous gastric feeds
Intermittent gastric feeds
Continuous transpyloric feeds
Small sucking feeds with bolus tube feeds

Q.4. How would you assess the outcome of Ahmed's nutritional goals?

Compare to intrauterine growth standard
Minimal gastric residuals on aspiration
Presence of hypoglycaemia
No abdominal distension and passing stools

Case study 2: nutritional requirements of a term infant with a congenital heart defect

Maggie was born at term to a 38-year-old woman. At birth Maggie's Apgar scores were 6 and 8 at one and five minutes. During a breast feed on the postnatal ward Maggie's mother noticed that she became blue. On examination a heart murmur was present. Further investigation revealed Maggie had a congenital heart defect. She was transferred to the NICU and was scheduled for surgery later in the day.

Maggie tolerated her surgery well and was extubated and nursed in an open care system. One week following surgery Maggie remained nil by mouth, had a orogastric tube in place and was having 3 ml of aspirate every 4 hours. Her mother asked if she could breast feed Maggie as she had been expressing regular volumes of milk.

Answer the questions below giving a rationale for your choices based on the evidence in the chapter.

Q.1. How does Maggie's disease affect her nutritional needs?

Increases her energy requirements
Decreases her total fluid requirements
Increases her need for amino acids

Q.2. How should Maggie be fed?

Small frequent oral feeds
Intermittent gastric feeds
Combination of breast and tube feeds
Combination of parenteral and enteral feeds

Q.3. Are there any contributing factors that may complicate Maggie's progression with oral feeds?

Decreased intestinal motility
Inability to coordinate sucking and breathing
Presence of an orogastric tube
None

Q.4. What assistance may her mother find beneficial?

Talking to other mothers about feeding
Consistent advice from the NICU staff
Explanation of how the breast pump works
Privacy to feed Maggie

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Chapter 12

Neonatal Infection

chapter 12

Contents

Introduction

The incidence of early onset neonatal sepsis is said to have dramatically declined over the past 10 years, attributed to increased use of antenatal antibiotics and more effective management of premature rupture of membranes (Bedford-Russell 1996). Infection still remains an important cause of morbidity and mortality in the neonatal period due to the increase in late onset nosocomial infection. This increase is due, in part, to the improved survival of extremely low birth weight (ELBW) infants, with its associated long stay in hospital, immunological incompetence and the increased usage of parenteral nutrition through central venous catheters (Philip 1994; Greenough 1996; Avila-Figueroa *et al.* 1998). Nosocomial infection is a common, costly and clinically important measure of outcome and may be a valuable indicator of performance in neonatal intensive care (Fowlie *et al.* 1996).

This chapter will review the current information regarding neonatal susceptibility to, and acquisition of, infection, the organisms commonly implicated, investigation and treatment modalities. It should serve to increase the neonatal nurse's ability to detect and manage the infected infant. Terminology used in the chapter is defined at the outset in Table 12.1.

***Table 12.1* Definitions used in neonatal infection**

Timing of infection	
Very early onset	< 24 hours
Early onset	1–7 days
Late onset	> 7 days
Nosocomial infection	Hospital acquired infection
Bacteraemia	The presence of viable bacteria in the blood.
Septicaemia	Systemic disease caused by the multiplication of organisms circulating in the blood
Sepsis	The presence of various pus-forming and other pathogenic organisms, or their toxins in the blood or tissues

Source: Bone 1991; Philip 1994; Berger *et al.* 1998

Acquisition of antenatal infection

The intrauterine environment should be sterile, with the fetus surrounded by amniotic fluid which has bacteriostatic or bacteriocidal properties against many organisms (Isaacs and Moxon 1991). In addition, enclosure within the amniotic membranes serves to provide the fetus with a physical barrier against invading organisms, which may reside in the maternal genital tract.

As a consequence of these mechanisms, most fetuses are effectively protected from infection whilst in utero. Should an ascending organism breach these defences, however, amnionitis may occur, which infects the fetus due to the direct aspiration into the bronchial tree initiating pneumonia and bacteraemia (Blumberg and Feldman 1996). The organism group B streptococcus (GBS) is highly implicated in this transmission route, and is the commonest organism for early onset septicaemia and meningitis (Philip 1994; Boyer 1995; Blumberg and Feldman 1996; Greenough 1996; Berger *et al*. 1998).

The Mycoplasmas, *Mycoplasma hominis* and *Ureaplasma urealyticum,* commonly inhabit the female urogenital tract and have also been implicated in the premature onset of labour and the development of chorioamnionitis. The role of these organisms, especially *Ureaplasma urealyticum,* in the aetiology of chronic lung disease has provoked significant interest in recent years (Ridgeway 1990; Wang *et al*. 1995; Lyon 1996), and further investigation into its spread and management is warranted. Chorioamnionitis is widely believed to be responsible for premature rupture of membranes and preterm labour (Fox 1993), possibly due to its invoking an increase in the synthesis of prostaglandins. GBS and mycoplasmas are also thought to have the ability to cross the maternal placenta, or breach the membranes, as they have been isolated when membranes have been intact.

Other organisms that may ascend from the genital tract and contaminate the amniotic fluid are *Bacteroides, Escherichia coli, Clostridium* and *Peptococcus*. As *E. coli* is highly implicated in the development of neonatal meningitis (22–47 per cent of cases: cited Rennie 1995), suspicion of infection with this organism should be taken seriously.

Infection via the maternal bloodstream (transplacental haematogenous spread) is also recognised, as alluded to above, whereby organisms affecting the fetus are present despite intact membranes. *Listeria monocytogenes* causes placentitis (Fox 1993), which then serves as a focus from which the fetus is subsequently infected. The transplacental mode of spread may also affect placental function, resulting in a growth-retarded fetus. The placental transfer of viruses, for example, cytomegalovirus and rubella, is well documented (Logan 1990; Miller 1990) in their relation to growth retardation and congenital defects when acquired in the first trimester. Likewise, documented evidence exists of placental transfer of hepatitis B and herpes simplex and varicella zoster, but these are said to be rarer events, and the more likely source of infection is intrapartum (Logan 1990; Lyall and Tudor-Williams 1997).

Intrauterine infection with parvovirus B19 is associated with severe fetal anaemia and development of hydrops fetalis due to inhibition of the erythroid progenitor cells (Logan 1990). Women presenting with fetal hydrops should have serum screening for this organism antenatally, in order to optimise postnatal management.

The vertical transmission of human immunodeficiency virus (HIV) from mother to fetus is known to involve several routes, but transplacental acquisition is well documented, and thought to be related to the maternal viral burden, disease stage and the mother's own immune response (Johnstone and Mok 1990;

Mueller and Pizzo 1995; Lindsay and Nesheim 1997). There are questions as to the timing of acquisition of HIV and degree of risk of fetal infection, the answers to which are unclear (Kline 1996), but this has obvious implications for the neonatal nurse attending deliveries, and admitting any newly delivered infant into the NICU.

Acquisition of intrapartum infection

Factors that increase the likelihood of transmission of infection during the delivery process are the presence of preterm labour, maternal pyrexia and prolonged rupture of membranes (Rennie 1995). The most commonly acquired organism via this route, again, is GBS, due to the human gastrointestinal tract being the most common reservoir for this organism. Its secondary transmission to the female genitourinary tract is said to occur in 5–40 per cent (average 20 per cent) of pregnant women (Blumberg and Feldman 1996; Glantz and Kedley 1998). The acquisition of herpes simplex virus (HSV) infection is highest at this time due to contact with the genital secretions during delivery, with the attack rate for infection being greater if the mother has a primary infection at the time. The risk of infection is lower if recurrent lesions are present (Logan 1990; Kohl 1997). Likewise, hepatitis B virus (HBV) is also more likely to be transmitted around the time of birth by contact with the maternal vaginal secretions (Kane 1997). HIV transmission during delivery is also attributed to contact with the infected secretions.

The length of time in contact with the birth canal is implicated in transmission of infection, with studies suggesting that the first born twin has a 2.8 higher infection rate than the co twin, due to more prolonged exposure to blood and cervical secretions in the maternal genital tract during delivery (Kline 1996).

Chlamydial infection of the fetus is not, in general, acquired by ascending infection, and infection with this organism is rare following cesarean section with intact membranes. It is presumed, therefore, that infection is acquired during vaginal delivery, with the transmission rate from mother to infant being in the order of 50 per cent. The total number of sexually transmitted infections (syphillis, gonorrhoea and chlamydia) appears to be on the increase in the UK, with chlamydia and gonorrhoea rising by 20 per cent and 5 per cent respectively in 1996–7 (Hughes *et al.* 1998). This worrying rise in chlamydia has implications for the neonatal population in that chlamydial conjunctivitis is said to be five times as common as gonococcal ophthalmia (Ridgeway 1990), and in chlamydial infections left undiagnosed, between 10 and 20 per cent of those affected will develop pneumonitis which may be serious enough to require ventilation (Isaacs and Moxon 1991). Any infant presenting with conjunctivitis and purulent discharge should be taken very seriously, screened and treated accordingly.

It can be seen then that the vertical transmission of infection from the mother to the fetus is high, and since it is impossible to identify sero-positivity for HBV and HIV along with other organisms, in all women, all healthcare workers who

come into contact with patients' (both maternal and infant) blood and body fluids should implement universal precautions routinely as a matter of good practice (DoH 1990).

Late onset and nosocomial infection

Studies suggest that the face of neonatal infection has changed over the past decade, with a discernible decrease in early onset sepsis, to one of late onset which is invariably hospital acquired (Philip 1994; Berger *et al.* 1998).

The term nosocomial infection (NI) is defined as 'related to a hospital', or 'hospital acquired'. The source of the infection may be from contact with the mother, hospital personnel, or inanimate objects.

Late onset sepsis may occur due to the invasion of bacteria that have previously colonised the infant's upper respiratory tract, conjunctivae, mucosal surfaces, umbilicus or skin. This process occurs due to alteration in the host's native microflora which is affected by antibiotics, disease processes and reduced host immunity (Greene 1996). The time between colonisation and invasion will vary but predicting when this may occur is an important surveillance challenge to clinicians within neonatal units. The commonest organisms in late onset infection are *Staphylococcus, Klebsiella, Escherichia coli* and *Pseudomonas* (Greene 1996).

Nosocomial infection in the NICU is reported to occur in up to 27 per cent of admissions (Drews *et al.* 1995), and is most prevalent in the very low birth weight (VLBW) population, with the infection rate, length of stay and mortality rate being five times higher in this group, with the most common sites for infection being pneumonia (32.3 per cent), bloodstream (27.4 per cent) and skin infections (11.3 per cent).

Coagulase negative staphylococci (CONS) appears to be the most prevalent cause of late onset infection in neonatal units, and is an important cause of morbidity, particularly in the VLBW infant, and is commonly associated with catheter placement for intravenous feeding (Philip 1994; Greenough 1996; Avila-Figueroa *et al.* 1998; Berger *et al.* 1998). Spafford *et al.* (1994) suggest that this risk may be reduced by the addition of continuous low dose vancomycin to the parenteral fluids; however, serious concerns about the emergence of vancomycin-resistant organisms preclude this approach currently (Fanaroff *et al.* 1998). Avila-Figueroa *et al.* (1998) further suggest that it is not necessarily catheter placement that is the issue, but the lipid infusions the infant is receiving, and propose that 'lipid emulsions may fuel a proliferation of colonizing bacteria and facilitate bloodstream invasion'. This suggestion warrants further studies to detect ways of reducing this risk.

CONS is not only implicated in central lines and lipid infusion, having been reported to be a significant contaminant of hospital stethoscopes, which may serve as a vector for its transmission around the NICU. Wright *et al.* (1995) highlight the fact that VLBW infants have immature, and often abraded skin with a variety of portals for infection from lines and surgical incisions, and as a consequence have an increased susceptibility to infection from stethoscopes that

may be placed near these sites. They report a fall in contamination rates following the instigation of a more rigorous stethoscope cleaning policy, which reduces the bacterial load to which the infants are exposed.

Staphylococcus aureus is another frequently reported pathogen in neonatal nosocomial infection, with the infant less than 1500 g, invasive procedures and longer hospitalisation denoting the most 'at risk' groups (Yamauchi 1995). 'Outbreaks' of infection within NICUs are invariably related to lapses in good infection control procedures. Such a situation is reported by Johnson *et al.* (1996), whereby seven infants were affected by *S. aureus* following babies 'being handed from one member of staff to another without handwashing'. Whilst none of the infants was adversely affected by this in the long term, it is a salutary reminder of how important basic principles of practice are. Handwashing practices have been extensively researched among nurses, with authors agreeing that poor handwashing practices contribute to nosocomial infection (Larson 1995; Callaghan 1998). Callaghan further notes that nurses' awareness of transmission of nosocomial infections was generally poor, and that the general standard of handwashing hygiene was particularly low. This view is reflected by Royal *et al.* (1999), where they report control of a potentially serious outbreak of *Klebsiella pneumoniae* in their neonatal unit. Whilst they also changed empiric antibiotics, they suggest the main feature of control of the outbreak was strict attention to handwashing, with the nursing team undertaking the role of advocate and ensuring handwashing was adhered to before and after touching infants. Anyone refusing to comply with this simple request was reported to the infection control team!

Handwashing is not the only area implicated in cross infection. The laryngoscope has been identified as a potential vector responsible not only for colonisation of infants with *Pseudomonas aeruginosa,* but has also been implicated in the subsequent deaths of five infants in two separately reported outbreaks (Foweraker 1995; Neal *et al.* 1995). This again can be avoided by strict attention to maintaining cleanliness and decontamination of equipment.

There is thought to be a substantially greater mortality and morbidity amongst infants who develop nosocomial infections (Fanaroff *et al.* 1998). Whether this is as a result of the septicaemia or a reflection of infant vulnerability is yet to be determined. Prevention of NI is an important and integral part of the neonatal nurse's role. Its control and incidence may, potentially, be reduced by enforcing strategies that reduce contamination and cross infection, education of all members of the team and parents regarding handwashing techniques, and by liaising with, and taking advice from, the infection control team.

The risk factors for hospital acquired infection are listed in Table 12.2.

The susceptible host

Infection is more common in the neonatal period due to exposure to a large number of organisms, but also a failure of the host defence mechanisms to clear micro-organisms from the blood and tissues. The 'immune deficiency' of

***Table 12.2* Risk factors for nosocomial infection**

Infant

Weight < 1500 g

Depressed immunological function

Presence of underlying disease

Raised gastric pH

Poor nutritional status

Need for total parenteral nutrition (esp. lipids)

Multiple antibiotic usage

Multiple sites of access
- ETT tube
- IV cannulae
- central catheter placement

Long stay in hospital

Environment

Poor compliance with infection-control techniques
- handwashing
- shared equipment (e.g. laryngoscopes)
- scales
- scanning equipment

Presence of opportunistic organisms

Overcrowding

Inadequate staffing levels

Poor disposal of contaminated waste

Source: adapted from Sproat and Inglis 1992

the newborn is relative rather than absolute (Isaacs and Moxon 1991), and due to its naiveté rather than defects *per se*. The immune system is made up of several component parts which are all necessary to maintain defence mechanisms and recognise invading foreign material. The physical defences include the skin, gastric secretions, tears, intact mucous membranes, ciliated epithelium and urine pH. All, or certainly some, of these aspects will be potentially affected in the sick newborn infant by either immaturity due to poor keratinisation of the skin, or by interventions preventing their efficacy during procedures such as endotracheal intubation and ventilation which will inhibit mucociliary movement.

Non-specific defences include **phagocytes** (neutrophils, monocytes), natural killer cells (NK), inflammatory responses and the antimicrobial proteins, including complement activation. Phagocytes are derived from a common precursor myeloid stem cell, often referred to as colony forming units (CFUs). These units are stimulated by colony stimulating factor (CSF) and cytokines. Polymorphonuclear neutrophils are the most numerous of the white blood cells and are chemically attracted to bacteria and fungi which they are able to destroy by first engulfing the organism and then inducing the 'respiratory burst', in which oxygen is actively metabolised to release hydrogen peroxide, hydroxyl radicals and superoxides which are powerful microbiocidal agents (Roitt 1991). This process is dependent on the cells' ability to be able to 'home in' on the organism by chemotaxis with the activation of complement, which amplifies the response and increases adherence to the surface by opsonisation. Serum opsonins are plasma protein substances that enhance phagocytosis by making the organism more visible or 'attractive' to the macrophages, which then allows engulfment and destruction to occur. In the newborn infant this process can be affected, as there is little or no transplacental crossage of complement. Whilst infected newborns do have an ability to release complement, it has both quantitative deficiencies and qualitative differences which affect its function (Lewis and Wilson 1995). Concentration increases postnatally but does not reach adult levels until between 6–18 months of age. It is also recognised that term and preterm infants have a relative deficiency in opsonin activity to a variety of organisms (Crockett 1995). It follows therefore that both phagocytosis and opsonisation are affected, and a study undertaken by Kallman *et al.* (1998) shows this to be particularly pertinent in the case of GBS infection, with the problem being further compounded in the preterm population.

Macrophages develop from monocytes and are crucial to the body's defence system. They reside in specific parts of the body, mainly the alveoli and liver. From these strategic positions macrophages filter out blood-borne **antigens** and degrade them without producing an immune response. They also serve to provide a further function by becoming antigen presenting cells (APC), whereby a fragment of the antigen is displayed on the macrophages' surface, which the immune system then recognises as non-self and brings into force the cell and antibody mediated immune responses.

NK cells are derived from lymphocytes, and are closely related to T lymphocytes. They play an important part in the defence of intracellular pathogens, particularly herpes group viruses. NK cells destroy by the binding to the membrane of the target, and releasing a cytolysin, which destroys by **apoptosis**. **Interferons** augment NK cells' toxicity, and since interferons are produced by virally infected cells, an integrated feedback defence is created (Roitt 1991). Although NK cells are said to appear early to mid gestation, they are immature and have decreased cytotoxic activity which diminishes their functional ability. This functional ability is further reduced due to the newborn's inability to produce sufficient amounts of ? interferon (Harris 1992).

Specific defence mechanisms involve two distinct lymphocytes. Both of these cell types are generated from bone marrow, but where they gain immunocompetence determines their ultimate development into **T cells,** which

are responsible for **cell mediated immunity,** or **B cells,** which confer so called **humoral immunity,** from which antibodies are produced.

T cells are 'educated' within the thymus gland. During this time they differentiate into three cell types: T helper cells (CD4 cells), T cytotoxic cells (CD8 cells) and T suppressor cells. T helper cells are the most numerous, and their function is to recognise antigens on the surface of APCs and stimulate a proliferation of cytotoxic T cells and B cells by the release of interleukins. Cytotoxic T cells bind to the targeted cell and perforate the cell membrane, which allows for an influx of extracellular calcium, killing it. Suppressor T cells are inhibitory in their action and are thought to suppress activity of both T and B cells once the antigen has been inactivated. This mechanism is also thought to be important in preventing autoimmune disease. Whilst there is no evidence to support maternal transfer of T cell specific immunity to the fetus, the neonate can produce a T cell response to an antigen challenge. However, overall function is impaired due to diminished cytotoxicity and decreased cytokine (interleukin) production (Lewis and Wilson 1995).

Within humoral immunity it is recognised that maternal transfer of some **immunoglobulins** (antibody) does occur, and affords the neonate some protection in the initial stages. Maternal IgG transfer begins at approximately 22 weeks of gestation, transporting larger quantities after 30 weeks (Lewis and Wilson 1995). This is made possible by the presence of receptor sites on the placenta. As the placenta does not have such sites for IgM (the acute phase immunoglobulin), it cannot cross to the fetus, so immunity to acute maternal infection around the time of delivery is not incurred. Elevated IgM levels in the newborn indicate congenital intrauterine infection.

The more preterm the neonate is the less placental transfer will have occurred, so passive immunity is diminished. Neonatal resistance to bacterial pathogens to which the mother has little or no IgG is particularly compromised by the inability to produce antibody, due to immaturity or a lack of B cell (Lewis and Wilson 1995). In conjunction with this, passively acquired IgG is rapidly catabolised, with a half-life of approximately 20 days. Losses will occur much more quickly in the sick infant who is repeatedly bled and transfused with washed packed erythrocytes (Lawton 1992), so the benefit of the maternal transport system is quickly lost.

B cells are the antibody-producing component of the immune system. Once an antigen (organism) has been recognised within the body, B cells become activated to produce plasma cells which secrete antibody specific to the antigen. Some B cells differentiate to become memory cells which remain in the system so that a more rapid immune response occurs should the same antigen present itself at a later date.

The five immunoglobulin types have differing structures depending on their site and function:

- IgG is the most abundant antibody in the plasma. It provides passive immunity to the fetus, it is able to neutralise toxins and bind organisms to enhance phagocytosis. It also activates complement and increases opsonisation.

- IgM is the largest immunoglobulin and is an efficient agglutinating and cytolytic agent.
- IgA is secretory in nature and present in saliva, tears, intestinal secretions and maternal colostrum. It inhibits adherence of organisms to the surface of mucosal cells, thereby preventing entry to the body. It also activates complement.
- IgD function appears in the main to be present on the surface of lymphocytes to control activation and suppression of B cells.
- IgE is present in very low quantities in the serum but is present in higher quantities in certain sites, for example, the respiratory and gastrointestinal tracts. It triggers the release of histamine and other vasoactive agents when presented with antigen and creates the body's 'allergic' responses (Roitt 1991).

It can be seen, therefore, that the newborn infant is equipped with mechanisms to cope with organisms following birth, but the immune system and its responses are restricted by physiological differences and functional deficiencies, which are further compromised in the premature or physiologically stressed infant.

Signs of neonatal sepsis

The neonatal nurse, with expert clinical skills and selection of appropriate monitoring, is pivotal in the early recognition of sepsis. It is imperative that sepsis is preempted or detected as early as possible due to the recognised inadequacies of the immune system response, and its inability to contain microorganisms. If not detected early enough this may lead to an initial infection of a specific body system being rapidly disseminated, producing an overwhelming septicaemia or meningitis, or in the worst scenario, death. Indicators for potential infection should be apparent from the maternal history, for example gestation of fetus, rupture of membranes, maternal pyrexia or infection. These facts can be elicited during a pre-delivery visit to the labour ward, if time allows, or by communication with the delivery unit team.

Despite any overt history, the interventions that most infants receive following admission to the NICU increase the likelihood of acquiring infection, due to breaches of physical defence systems following intubation and ventilation, the siting of intravenous and arterial lines, and by exposure to multiple members of the NICU team.

Respiratory signs

Infants presenting with tachypnoea, grunting, recession and apnoea (Isaacs and Moxon 1991; Roberton 1992) may have early onset infection or respiratory distress syndrome. It is impossible to differentiate these conditions clinically or even following chest radiography as the chest X-ray reveals similar findings.

These infants need careful consideration, as both problems may coexist, and without antibiotic therapy the septicaemia can kill in hours (Greenough *et al.* 1992).

The respiratory symptoms from GBS infection are due, in part, to the organism-mediated release of the vasoactive agent thromboxane A_2, which causes severe pulmonary vascular constriction. This combined with the infant's reduced white cell ability to kill the organism leads to rapid dissemination of the disease process and profound generalised physiological instability.

Late onset sepsis and meningitis may manifest with respiratory signs due to central effects on the respiratory centre leading to tachypnoea or apnoea.

Thermal signs

Temperature instability, after environmental factors have been eliminated, may be an early indicator of infection. Approximately half of the babies with proved sepsis are febrile, one-third are normothermic and 15 per cent have hypothermia (Isaacs and Moxon 1991). The further the deviation from the normal range, be that high or low, the more significant the finding is.

Cardiovascular signs

Cardiovascular changes of tachycardia, hypotension and poor capillary refill times may indicate sepsis. A heart rate of less than 160 bpm is often present in early sepsis. If cardiac output is compromised or peripheral vasoconstriction is present, the capillary refill time (CRT) may be prolonged. The sites for estimating CRT are optimally the forehead and the sternum, with the refill time being less than 3 seconds (Strozik *et al.* 1998). Pallor or mottling of the skin may also be apparent.

Skin signs

The skin should be carefully scrutinsed for lesions of petechiae, which may result from congenital infection, or a raised papular 'pin point' rash can be present in an infant with congenital *Listeria* infection (Rennie 1995). Necrotic skin lesions are associated with *Pseudomonal* sepsis or sepsis due to fungi. Necrotising fasciitis can be caused by *Staphylococcus aureus* and Gram negative bacteria. Jaundice occurs in one-third of septic infants, and is more associated with Gram negative infections, but may occur in GBS. It is probably due to increased haemolysis and endotoxic effects on the liver and is characteristically unconjugated (Isaacs and Moxon 1991). Disseminated intravascular coagulation (see p. 180), with petechiae and bleeding from puncture sites, the gut or kidneys, is a rare,

late and worrying sign. It is thought to occur following triggering of the clotting cascade as a result of diffuse endothelial damage from the circulating endotoxins (Emer 1992).

Abdominal signs

Gastrointestinal signs of distension and ileus are a common and important sign of sepsis and need to be differentiated from intestinal obstruction or necrotising enterocolitis (NEC), all of which may present similarly with poor toleration of milk and bile-stained gastric aspirates. Whilst no single organism has been found to be the cause of NEC, *Klebsiella, Glostridium difficile and E. coli* have, amongst others, been implicated in its development. However, 'epidemic' NEC presumed to be infectious agent-related is responsible for only 5 per cent of all cases (Clark and Miller 1996).

Neurological signs

Lethargy, irritability or 'not handling' well occurs in approximately one-half of infants with sepsis, and should always be further investigated. Meningitis may be heralded by increasing irritability, alterations in consciousness, poor tone and tremors, with up to 75 per cent of infants having some seizure activity (Klein and Marcy 1995).

Table 12.3 shows a composite of the overall likelihood ratios of clinical findings for neonatal bacterial infection. This table demonstrates not only the diversity of signs but also how poorly many of them correlate to infection. Given, however, that most infants will present with many of the listed signs, the likelihood of infection increases.

Investigations

Despite the current trend in nursing and medicine towards 'evidence-based' decision-making and management, experience and intuition are often the precursors of the initiation of screening for infection when the infant 'doesn't handle well'. This intuitive aspect is probably as effective a screening tool as any when one considers Fowlie and Schmidt's (1998) systematic review of diagnostic tests for bacterial infections which concluded that the reported accuracy of tests is generally poor. There is, of course, a necessity to try to confirm diagnosis and identify the causative organisms in order to ensure that the most appropriate antibiotic therapy is prescribed. Once sepsis is suspected, a battery of laboratory tests can be put in place to confirm or refute suspicions.

Surface swabs and site cultures

Surface swabs and cultures from deeper sites are often obtained initially on admission to the NICU or when late onset sepsis is suspected. The

Table 12.3 **The likelihood ratios of clinical findings for neonatal bacterial infections**

Clinical finding	*Likelihood ratio*
Common signs	
Pallor	14.4
Poor feeding	8.7
Tachycardia/arrhythmia	5.6
Decreased peripheral perfusion	5.4
Unstable blood pressure	4.0
Abdominal distension	3.5
Apnoea	3.1
Lethargy	2.3
Hyperbilirubinaemia	2.0
Retractions	1.7
Grunting	1.6
Abnormal tone	1.6
Tachypnoea	1.3
Cyanosis	0.3
Temperature instability	0.7
Uncommon signs	
Purpura	47.0
Omphalitis	32.5
Vasomotor instability	8.1
Bleeding	6.5
Pustules	6.1
Bulging fontanelle	5.4
Splenomegaly	4.1
Rash	4.0
Diarrhoea	3.6
Seizures	2.3

Source: Radetsky 1995

sensitivity of surface swabs in the detection is high, in the order of 90 per cent (Thompson *et al.* 1992), but the specificity is said to be poor, at around 50–78 per cent (Evans *et al.* 1988; Thompson *et al.* 1992). There appears to be little value in obtaining swabs on admission from multiple sites, for example nose, throat or rectum, as the optimal site for culture is reported to be the deep ear swab alone (Evans *et al.* 1988; Thompson *et al.* 1992), and obvious focal lesions in later onset suspected infections. The benefit of taking multiple surface swabs lies in the detection of the colonised infant. Isaacs and Moxon (1991) suggest that a heavily colonised infant with a strong clinical suspicion of infection should be treated with antibiotics despite negative blood cultures.

Gastric aspiration and culture does not help identify which babies are likely to develop infection, as the polymorphonuclear leucocytes and bacterial content

of the specimen are of maternal origin and reflect fetal exposure to infection, but not necessarily neonatal infection (Borderon *et al.* 1994; Powell and Marcy 1995).

Tracheal aspirates obtained following long-term ventilation, similarly, may not reflect infection, but rather colonisation of bacteria within the system (Powell and Marcy 1995). The treatment of such cases with antibiotics demands caution as it may contribute to an increase in resistant strains of organisms.

Haematological tests

Blood culture is mandatory before starting treatment with antibiotics (Yoxall *et al.* 1996), and is considered the definitive laboratory test (Radetsky 1995). The blood should be drawn from a peripheral vein or artery following strict cleansing of the overlying skin, or from newly inserted umbilical catheters. Blood taken from indwelling catheters that have been present for some time may reflect contamination or colonisation of the intravascular device (Isaacs and Moxon 1991; Yoxall *et al.* 1996), and as a consequence is not recommended.

Other haematological tests include the total neutrophil count. This test is a more reliable indicator of neonatal sepsis than the total white cell count as the normal range is wide and varies with gestation and postnatal age. Further to this, other pathology such as asphyxia, meconium aspiration, periventricular haemorrhage and pneumothorax can give a raised total count (Powell and Marcy 1995). Both neutropenia (less than $2–2.5\times10^9$) and neutrophilia (greater than $7.5–8.0\times10^9$) are indicators of infection, with neutropenia being particularly suggestive of severe sepsis as this reflects depletion of the granulocyte pool in the bone marrow (Isaacs and Moxon 1991). Immature neutrophils are seen in the peripheral blood of healthy newborns, but their presence is increased in sepsis as an increased number are released from the bone marrow. This is described as the 'left-shift'. Measuring the immature to total neutrophil ratio (ITR) is said to be a sensitive predictor of sepsis, with a ratio of greater than 0.2 being a good indicator of infection (Roberton 1992).

Measurement of acute phase proteins, especially C-reactive protein (CRP), is often undertaken, with levels of greater than 6 mg/l being indicative of sepsis. Its level also rises in necrotising enterocolitis and following surgery.

Whether it is a sensitive enough predictor for sepsis, in isolation, is debatable but its value in monitoring the course of infection and infant response to therapy is recognised (Roberton 1992; Powell and Marcy 1995; Benitz *et al.* 1998), as its level should return to normal in less than 96 hours if the stimulus to its production has been removed (Yoxall *et al.* 1996).

Platelet count measurement in the newborn is well established, with the normal count, regardless of birthweight, rarely being less than 100×10^9/l in the first 10 days of life or less than 150×10^9/l during the next 3 weeks. Thrombocytopenia due to increased platelet destruction, aggregation and adhesion occurs in 50 per cent of cases of sepsis, but is usually a late finding and not until some time after the infant is obviously septic (Powell and Marcy 1995).

Cerebrospinal fluid

Lumbar puncture (LP) (see p. 307) has been considered mandatory in the evaluation of newborns for infection (Radetsky 1995) to rule out bacterial meningitis, which occurs more frequently in the neonatal period than at any other time in life, and carries a significant morbidity amongst survivors (Yoxall *et al.* 1996). Positive culture yield is said to be low in infants in whom the procedure is performed on a 'routine' basis as part of admission screening (Klein and Marcy 1995; Radetsky 1995). This factor, coupled with the technical difficulties, discomfort and potential hypoxia encountered in the ventilated and physiologically unstable infant, means that many units will not perform this procedure unless the infant is manifesting with central nervous system signs. The use of local anaesthesia has been suggested in reducing pain, but this does not prevent the other physiological changes encountered by infants (Klein and Marcy 1995) and its use potentially increases handling and duration of the procedure. Controlled pre-oxygenation may be employed to ameliorate the hypoxia, but this needs careful assessment and attention by the nurse assisting the procedure.

Some units continue to undertake this procedure despite the associated problems, the opinion being that the high incidence of neonatal meningitis with its associated mortality and morbidity justifies its inclusion in routine infection screening (Yoxall *et al.* 1996).

The obtained cerebrospinal fluid (CSF) should be 'gin clear'. Yellow coloration (xanthochromia) is often seen on neonatal specimen due to bilirubin staining or old intraventricular haemorrhage. A CSF white cell count of more than 30/mm^3 with more than 66 per cent neutrophils, or a CSF protein level greater than 1 g/l in a term infant or 2 g/l in a preterm infant, are suspicious and meningitis should be seriously considered. A CSF glucose level should also be measured and correlated to the plasma glucose level. The plasma glucose sample should *always* be taken prior to the LP as the level will probably increase due to stress, which can make the result less easy to interpret. The CSF glucose is approximately 70–80 per cent of the blood glucose (Rennie 1995).

Urine

Urine should be obtained for culture, but therapy should not be withheld if there is difficulty obtaining the specimen. If a bag is to be used to collect the specimen the surrounding area should be thoroughly cleaned to minimise contamination from organisms colonising on the skin. The bag should be removed as soon as urine is voided for the same reason. Positive bag urine cultures are often viewed with suspicion, and a suprapubic aspiration may be performed. Whilst this procedure often appears drastic (see p. 309), the long-term implications of a urinary tract infection with its potential development of renal scarring and atrophic kidneys in later life (Klein and Long 1995) probably justifies its use.

Urine antigen detection (late agglutination test) may be available to facilitate early detection of GBS. It is reported to be between 88 and 100 per cent sensitive (Radetsky 1995), but is not available in many hospitals.

Other laboratory tests, including fibronectin levels, which fall in infection, and cytokine concentrations such as IL1ß, IL6 and tumor necrosis factor, which are thought to be endogenous mediators of immune response to bacterial infection, are being currently researched but further clinical data are needed to determine whether these are truly useful in diagnosing and following the progress in neonatal infection (Powell and Marcy 1995).

Management of the infected newborn

Supportive therapy and broad spectrum antibiotics should commence as soon as infection is suspected, as a delay can result in mortality or significant morbidity.

General supportive therapy

Ventilatory support

Many babies will become apnoeic when infected. Depending on the underlying cause, respiration may be further compromised due to abdominal distension, which may invoke diaphragmatic splinting, resulting in poor oxygenation and carbon dioxide retention (see p. 109). Elective intubation and ventilation is an important supportive measure that may be neglected, following prescription of antimicrobials in the misguided hope that they will be sufficient, leaving the infant to collapse and require emergency resuscitation later (Isaacs and Moxon 1991).

Ventilation will be mandatory in the infant with GBS due to its association with pulmonary vasoconstriction and the development of persistent pulmonary hypertension (see pp. 104 and 137).

Cardiovascular support

Maintenance of the circulation cannot be over-emphasised. The septicaemic infant is likely to be shocked, so assessment and management of blood pressure and perfusion are of paramount importance. The result of a reduction in circulating blood volume can cause pre-renal renal failure unless aggressive management is adopted (Rennie 1995).

Peripheral perfusion and capillary refill time needs to be frequently estimated. Blood pressure should be maintained within normal limits for the infant's gestation and age (see p. 138). Both are often compromised due to poor cardiac output, acidaemia and low circulating volume (Greenough *et al.* 1992). Volume

replacement with blood or fresh frozen plasma (FFP), saline or human albumin solution (HAS) is indicated. Blood and FFP are the most beneficial as they, theoretically, not only replace volume but also contain immunoglobulins and clotting factors (Isaacs and Moxon 1991), but there are no studies providing evidence that the outcome from infection is better if FFP is used (Bedford-Russell 1996). Human albumin solution or normal saline may be given, saline being as effective in treating hypotension as albumin (So *et al.* 1997), but neither contains the postulated added value of the other products. The transfusion should be given at 10–20 ml/kg over 20 minutes, being mindful that the hypoxic, acidaemic infant has poor myocardial contractility and is prone to overload and cardiac failure (Roberton 1997), so careful monitoring is required.

If volume replacement is not sufficient to treat the hypotension, an inotropic agent may be included, for example dopamine or dobutamine (see p. 139).

Acid base balance

The acid base state may be compromised in septic shock. Whilst the instigation of ventilatory support to correct respiratory acidaemia, and support of the cardiovascular system with volume and inotrops should correct the blood pH, further correction may be necessary by the use of intravenous alkali therapy. The use of bicarbonate continues to be contentious (see p. 52), but the effect of acidaemia on myocardial contractility, surfactant production and pulmonary vascular resistance warrants its careful and considered use in the infant with a pH less than 7.25 and a base deficit of less than 10 mmol/l, if other modalities have failed to correct the situation (Greenough *et al.* 1992).

Fluid and electrolyte balance

Management of fluids in this infant needs to be meticulous, with a record of intake of all infusions and boluses of colloid and crystalloid being strictly made. Fluid balance may be further complicated in infection due to the inappropriate secretion of antidiuretic hormone (IADH) brought about by severe hypoxia or meningitis (Rennie 1995) (see Chapter 10).

Urine output should be measured by the weighing of nappies, urine collection bags/devices or catheterisation, each having its advantages and disadvantages.

Nappies need frequent changing to prevent evaporative losses, which, though not as much a problem now with hyper-absorbent disposables, still means more regular handling. Collection devices either fall off if not sufficiently secured or can cause skin excoriation in the very premature or fragile newborn, and catheterisation is usually undertaken with a tube not specifically designed for this purpose increasing the likelihood of trauma and introduction of infection in this already compromised infant.

Hyponatraemia may also transpire secondary to IADH secretion, and both sodium and potassium losses increase in NEC or the diuretic phase of acute

tubular necrosis (Roberton 1992), which may occur following low renal perfusion states. Hypoglycaemia may occur as a result of increased metabolic demands coupled with low substrate (Karp *et al.* 1995) so regular monitoring is imperative if acute and long-term sequelae are to be avoided. Fluid volumes and concentrations of dextrose need to be carefully titrated to maintain blood glucose within the acceptable normal range.

Haematological management

A careful running total of blood taken for testing needs to be recorded as sick infants, especially those less than 1500 g, tolerate anaemia badly (Greenough *et al.* 1992). Regular top-up transfusions may be required to correct deficits.

Bruising, oozing and petechiae due to consumptive coagulopathy (see p. 180) need to be promptly recognised and managed with platelet transfusion, fresh frozen plasma, cryoprecipitate and vitamin K, as appropriate (Rennie 1995).

Exchange transfusion may be advocated in extreme circumstances, as it corrects bleeding by simultaneously washing out coagulation inhibitors and supplying missing coagulation factors. It is also a source of opsonins and has the potential to increase the oxygen-carrying capacity of the blood (Roberton 1992; Perez and Weisman 1997). However, the procedure potentially carries with it dramatic haemodynamic changes which may be poorly tolerated (see p. 175).

Antimicrobial therapy

Because of their susceptibility to vertically and nosocomially acquired infection, newborn infants are often prescribed antibiotics on presumptive sepsis before cultures are reported (Saez-Llorens and McCracken 1995). Initially, broad spectrum cover is prescribed, which is usually dependent on local policies and the unit flora (Roberton 1992), changing to specific antibiotics when dictated by the culture and sensitivity results. Whilst this is accepted as the most appropriate and safe practice for the infant, administration of broad spectrum agents for empirical treatment of presumed sepsis is highly implicated in antimicrobial resistance, which world wide is a problem reaching crisis dimensions (Goldmann and Huskins 1997).

It has also been postulated that indiscriminate use of antibiotics may actually predispose infants to NEC rather than reducing the risk (Clark and Miller 1996). The use of antibiotic therapy within neonatal units therefore needs to be multidisciplinary, involving the prescribing clinician, clinical microbiologist, the infection control team and the nurse caring for the infant, if potential problems are to be avoided.

In acute infection, intravenous administration of antibiotics is the route of choice. Oral medication is clearly unreliable in babies who are shocked, acidotic

and obviously unwell, with the risk of delayed absorption being too great (Northern Neonatal Network 1998).

As neonatal nurses are invariably the ones who administer these drugs to this susceptible population, an understanding of the choice of drugs and their potential side-effects needs to be considered if the UKCC's *Standards for the Administration of Medicines* (UKCC 1992) is to be adhered to. Advice and information should be sought from the unit's pharmacist and reputable texts, for example, *The Neonatal Formulary* (Northern Neonatal Network 1998) or *Medicines for Children* (Burns 1999) regarding reconstitution solutions, displacement volumes, recommended dosage and storage of individual drugs.

Penicillins

With a 40-year record of safety and efficacy in neonates, penicillin remains the drug of choice for proved infections caused by groups A and B streptococci, susceptible pneumococci, meningococci, gonococci and *Treponema pallidum* (Edwards 1997). Penicillin G does not penetrate CSF well, even in bacterial meningitis, so other antimicrobials are recommended.

Ampicillin is effective against most strains of enterococci and *Listeria monocytogenes*. Despite the increasing prevalence of methicillin-resistant *Staphylococcus aureus* (MRSA), some antistaphylococcal penicillins are useful for infections caused by susceptible *S. aureus* and *S. epidermidis*. The withdrawal of methicillin from commercial use has necessitated substitution with nafcillin (Saez-Llorens and McCracken 1995; Edwards 1997). Ticarcillin, a semi-synthetic penicillin, is useful against *Pseudomonas aeruginosa*.

As penicillins are primarily excreted by the kidneys, renal function should be carefully monitored when the drugs are given parenterally. In high-dose penicillin treatment, bone marrow suppression can occur, so the WBC should also be monitored during treatment (Edwards 1997).

Aminoglycosides

The aminoglycosides—gentamicin, netilmicin, tobramicin—are widely employed in the newborn with a possible or proved infection resulting from Gram-negative enteric pathogens (Isaacs and Moxon 1991; Edwards 1997). They are also proposed to have a synergistic effect when used with penicillin against GBS (Saez-Llorens and McCracken 1995).

Adverse effects from aminoglycosides include renal toxicity and otoxicity, although the latter is difficult to determine due to other variables within the NICU setting. It is thought that gentamicin toxicity is more likely to occur with a dosage interval of 12 hours, as this leads to many infants having toxic trough levels. Dosage intervals need careful consideration. Intervals of 24 hours in infants greater than 30 weeks, with an increased interval to 36 hours when less than 30 weeks, have been recommended (Davies and Cartwright 1998). Serum levels

should be determined after five half-lives (at steady state) or after three to five doses have been administered (Edwards 1997).

Aztreonam

The aminoglycoside-like activity of aztreonam, with good CSF penetration, and its absence of nephrotoxic or ototoxic effects may make it useful against *E. coli* in the setting of renal compromise.

Cephalosporins

The cephalosporins are grouped into generations based on their spectrum of activity. The third generation cephalosporins (cefotaxime, ceftazidime) are the most useful in the treatment of neonatal infections, with activity against Gram-negative bacilli, meningococci, gonococci and *Haemophilus influenzae*. Ceftazidime is particularly effective against *Pseudomonas*. These drugs are generally well tolerated by neonates (Saez-Llorens and McCracken 1995), but serum creatinine levels, white blood cell counts and differential should be performed regularly to monitor for potential renal or bone marrow toxicity (Edwards 1997).

Vancomycin

This glycopeptide antibiotic is active against staphylococci, streptococci, enterococci and other Gram-positive bacteria. It is the drug of choice for the treatment of infections owing to MRSA (Edwards 1997). Infants receiving vancomycin should have renal function monitored and the trough level should be measured after the fifth half-life, or third to fifth dose.

Metronidazole

Metronidazole has excellent activity against Gram-negative, obligate anaerobic bacilli such as *Bacteroides*. It is particularly useful in necrotising enterocolitis (Edwards 1997).

Chloramphenicol

Chloramphenicol has been used for many years in the treatment of meningitis. Due to its toxicity in causing vascular collapse, bone marrow suppression and 'grey baby syndrome', many authors suggest its use should now be abandoned in the treatment of neonatal infection (Isaacs and Moxon 1991; Rennie 1995;

Saez-Llorens and McCracken 1995; Edwards 1997). As much safer and effective drugs are now available, its usage should be confined to exceptional circumstances.

Adjunctive therapies

The use of intravenous immunoglobulins in prevention or treatment of neonatal infection remains controversial (Bedford-Russell 1996). In the 1980s studies suggested that it significantly reduced the incidence of nosocomial infection, mortality and morbidity (Haque *et al.* 1986; Chirico *et al.* 1987), but in a further study Weisman *et al.* (1994) did not see any significant alteration of rate of sepsis or mortality when given prophylactically. Due to its lack of pathogenspecific antibody and large donor pool of three to five thousand donors, its use is not currently advocated in prophylaxis or routine treatment of neonatal infection (Perez and Weisman 1997).

As septic neonates are at risk of neutropenia and storage pool depletion, white blood cell transfusion has been attempted. The isolation of the buffy coat is difficult and also requires irradiation to prevent the infant developing graft versus host disease. As a consequence, this treatment modality remains unproved and experimental (Rennie 1995; Perez and Weisman 1997).

Other developments have centred on the use of granulocyte colony stimulating factor (GCSF), and granulocyte macrophage colony stimulating factor (GMCSF), to enhance endogenous defence mechanisms (Bedford-Russell 1996). Whilst studies in these areas are encouraging, large, prospective, randomised controlled studies are needed to test the treatment efficacy further (Perez and Weisman 1997).

The role of prostaglandin, thromboxane and leukotriene inhibition in the treatment of sepsis has not yet been established. The suggestion that non-steroidal anti-inflammatory agents such as ibuprofen or indomethacin may be utilised in order to ameliorate the effects of thromboxanes and tumour necrosis factor, which contribute to pulmonary hypertension and endotoxaemia (Rennie 1995; Perez and Weisman 1997), is being further investigated.

Conclusion

Congenital and hospital acquired infection remains a significant cause of neonatal mortality and morbidity. In increasing knowledge of the common causative organisms and the antenatal, intrapartum and postnatal factors associated with their acquisition, the neonatal nurse is well placed to advocate for the most effective management strategies to help optimise the infant's outcome.

Case study: susceptibility to infection in a preterm infant

A 26-year-old primigravid woman is admitted to the delivery unit at 27 weeks of gestation with ruptured membranes and complaining of abdominal pain.

Q.1. What information would you likely gain when visiting this woman prior to her delivery, and how would this help in deciding an immediate course of action after delivery?

Following a brief labour, a boy is delivered vaginally. He is pale and gasping with a heart rate of 80 bpm, but responds well to immediate intubation and positive-pressure ventilation.

Q.2. What are the factors that make this infant susceptible to infection?

Q.3. On admission to the NICU, what should the immediate management of this infant be?

Forty minutes after admission he looks pale and shocked and is becoming increasingly hypoxic and difficult to ventilate.

Q.4. What are the potential causes of his deterioration, and what management strategies would you instigate in order to regain stability?

Q.5. On day 2 a central venous catheter is inserted in order to give total parenteral nutrition. Why might this procedure adversely affect this infant's outcome?

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Chapter 13

Diagnostic and Therapeutic Procedures

Chapter 13

Contents

Introduction

Infants admitted into a neonatal intensive care unit will undoubtedly undergo a barrage of interventions in order to diagnose, stabilise and provide ongoing management of specific problems. This chapter incorporates many of the diagnostic and therapeutic procedures that may be undertaken within the NICU situation. Included are interventions that were previously described as 'medical' procedures, as many nurses now undertake these practices as part of advanced nursing roles (see p. 5). Each unit should have specific protocols for practice, but the content of this chapter may provide for development of protocols for procedures if they are not already in existence.

Gastric tube placement

Rooting and sucking are important components of infant feeding but do not begin to develop until 28 weeks' gestation and are not well established until 32–34 weeks (Tappero and Honeyfield 1993) (see p. 236). This makes feeding through a gastric tube an essential component of feeding the preterm infant (see p. 245). Term infants who are very sick, severely asphyxiated or have suffered cerebral haemorrhage will also require tube feeding, and any infant with an illness that compromises the gastrointestinal tract will require a gastric tube in place to permit gastric decompression to take place (see p. 345).

Indications

- Prematurity
- Neurological impairment
- Sick term infants
- Gastrointestinal tract anomalies

Access to the gastrointestinal tract may be through:

- Nasogastric tube
- Orogastric tube
- Transpyloric tube

Naso/orogastric tube

Equipment

- Size 6 or 8fg feeding tube
- 2 ml syringe
- Blue litmus paper
- Securing tape

Procedure

The length that the tube has to be inserted needs to be determined before placement. Measure from the bridge of the nose to the xiphisternum plus 1 cm (see Box 13.1). Place the infant in the supine position with the head in the midline and pass the measured length of tube backwards over the tongue into the oropharynx and advance slowly to the predetermined length and secure with tape.

For a nasogastric tube, measure from the tip of the ear to the bridge of the nose and down to the xiphisternum (see Box 13.1). With the infant in the above position and the head slightly extended to push the nose upwards, direct the measured tube backwards and downwards through the nostril, advancing slowly to the predetermined length and secure with tape.

To verify the position of the tube aspirate a small volume of the stomach content and test for an acid reaction on the blue litmus paper. In a sick infant who will be having X-rays for other reasons it is advisable to ensure that the gastric tube is in place beforehand; the position of the tube can then be verified at the same time. An alternative method of verification is to inject 1–2 ml of air down the tube whilst simultaneously auscultating over the stomach. This can be misleading, however, as the rush of air can be heard when the tip is in the distal oesophagus (Gomella *et al*. 1994).

Do not use any gastric tube without first verifying the position.

Box 13.1 **Gastric tube length estimation in centimetres**

Orogastric tube

Measure from the bridge of the nose to the xiphisternum and add 1 cm

Nasogastric tube

Measure from the tip of the ear to the bridge of the nose and down to the xiphisternum

Transpyloric tube

Measure from the bridge of the nose to the ankle, with the infant supine and legs extended

Transpyloric tube

Babies who suffer from marked gastro-oesophageal reflux or protracted vomiting when milk is given into the stomach may benefit from a transpyloric tube.

Equipment

- Size 5fg silicone duodenal feeding tube
- Size 6fg orogastric feeding tube
- Blue litmus paper
- 2 ml syringe
- Ampoule of sterile water
- Securing tape

Procedure

To determine the length of tubing to allow passage into the jejunum place the infant supine with the legs extended and measure from the bridge of the nose to the ankle (see Box 13.1). To advance the measured tube, pass it through the nostril and down into the stomach and test the gastric contents with litmus paper to verify the position. Turn the infant onto the right hand side and distil 2 ml of sterile water down the tube and continue advancing until resistance is felt, which indicates that the pylorus has been reached. It may take several hours for peristalsis to carry the tube through the pylorus to the jejunum, but once it has been reached secure the transpyloric tube and pass an orogastric tube into the stomach, check the position of both tubes radiographically before use. The orogastric tube serves to ensure the stomach remains decompressed and to monitor any reflux of milk.

Complications

- Vagal stimulation bradycardia—associated with placement of both naso- and orogastric tubes (Van Someran *et al.* 1984; Greenspan *et al.* 1990).
- Increased work of breathing, especially if nasogastric tubes are used in respiratory compromised infants (Stocks 1980).
- Aspiration of feed into the lungs through tubes inadvertently dislodged or passed into the lungs or only as far as the **distal** oesophagus. To continue administering feed when there is stasis of milk in the stomach can also lead to milk **aspiration;** it is important to check the amount of residual feed in the stomach at set intervals for this reason.
- Perforation of the oesophagus, posterior pharynx, stomach or duodenum —though rare, can result if too much force is used in passing the tube.
- Small bowel perforation.
- A risk of necrotising enterocolitis has been associated with transpyloric tubes (Sinclair and Bracken 1992) (see p. 248). The position of the naso/orogastric tube should be checked at the change of every nursing shift and also in between times if the securing tape has become loose.

Endotracheal tube placement

Endotracheal intubation may be approached from the orotracheal or nasotracheal route, with each requiring a slightly different technique to place the tube but with both achieving the same end result.

Indications

- To perform resuscitation
- To remove meconium from the trachea
- To provide mechanical respiratory support
- To aid bronchial lavage and removal of secretions

Equipment

- Paediatric laryngoscope with a good light source
- Suction apparatus
- Endotracheal tube and means to secure it (stylet if used)
- Magill forceps (if nasal intubation used)
- Bag and mask apparatus
- Stethoscope

Procedure

Place the infant flat in the supine position with the head in the midline and slightly extended—the 'sniffing the air' position. With the right hand, gently open the mouth, and with the left hand, insert the laryngoscope blade into the right side of the mouth and sweep the tongue to the left. Advance the blade a few millimetres into the **vallecula** and, by lifting the blade vertically, elevate the epiglottis to bring the entrance of the larynx and vocal cords into view (Figure 13.1). The view can be enhanced by exerting pressure on the thyroid cartilage with the third or fourth finger of the left hand. Cautious suction at this point will ensure that all the landmarks are clearly visible. Insert the orotracheal tube into the right side of the mouth and through the vocal cords. The nasal tube is passed through the nostril until visible in the pharynx. It is then guided through the vocal cords with the Magill forceps. Advance the tip of the tube 1–1.5 cm through the cords to position it midway between the thoracic inlet and the **carina,** i.e. approximately to the second **thoracic vertebra**.

To confirm the position of the endotracheal tube (ETT), attach it to the bagging equipment and commence mechanical ventilation. Equal air entry on **auscultation** of both sides of the chest will indicate a well-placed tube. As a guide, the length of the tube at the lips should be 6 cm plus the weight in kilograms and the size of ETT that will be required is as shown in Table 13.1.

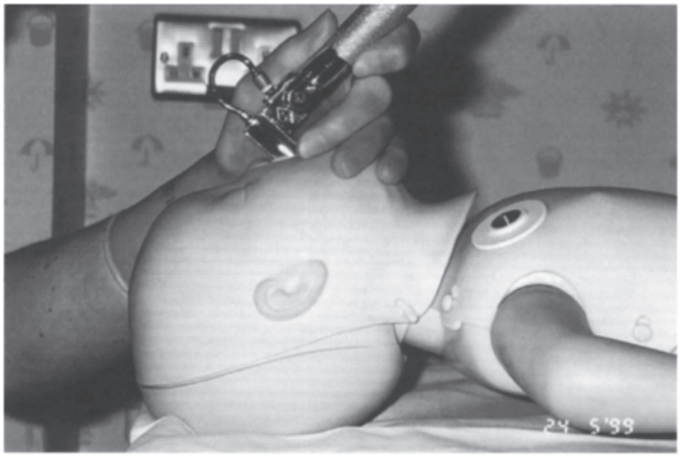

Figure 13.1 **Endotracheal intubation—head in midline and slightly extended**

Table 13.1 **Endotracheal tube size estimation**

Tube size (mm)	*Weight (g)*	*Gestation (weeks)*
2.5	<1000	<28
3.0	1000–2000	28–34
3.5	2000–3000	34–38
4.0	>3000	>38

Length of tube at the lips should be estimated in centimetres as the infant's weight in kilograms plus 6.

The most common problem is a tube advanced too far and into the right main bronchus. To overcome this, slowly withdraw the tube until equal breath sounds are restored. Auscultation over the stomach will also help identify a tube inadvertently passed into the oesophagus.

A chest X-ray taken in the anteroposterior view will confirm the position of the tube. Care must be taken to ensure that the head is in the midline and not flexed and that there is correct centring of the X-ray beam on the chest to ensure satisfactory location of the tube. An X-ray taken with the head deviated to the side or with the beam centred on the abdomen, especially in a term infant, may distort the position of the tube or make it appear marginally higher (Meerstadt and Gyll 1994). To locate the position of the tube using the X-ray, count down the vertebrae from where the posterior ribs arise, commencing at the first thoracic vertebra (T1). The tip of the tube should be advanced 1–1.5 cm through the vocal cords to just above the carina (Wilkinson and Calvert 1992). Following X-ray verification of an ETT position a record of the number of centimetres that take the tube to the

lips is useful. It provides a guide to how far a suction catheter should be advanced to reach the tip during ETT suction and also where to place the tube on any subsequent reintubation.

Stylets are often used to introduce the softer endotracheal tubes. They help keep the tube rigid and aid intubation. The stylet should not protrude beyond the tip of the tube, where it can cause damage or perforation of the trachea or oesophagus if misplaced. Care must be exercised in removing the stylet after intubation to ensure that a tube is not inadvertently withdrawn with it.

Complications

- Tracheal perforation—a rare complication avoided by careful use of the laryngoscope, endotracheal tube and stylet.
- Oesophageal perforation—the result of a traumatic intubation and can lead to a stricture formation that will require surgical correction.
- Laryngeal oedema—apparent after extubation and may require reintubation to allow a short course of steroids to be given to reduce the oedema.
- Subglottic stenosis—associated with long-term intubation and usually requires surgical correction.
- Palatal grooves—the result of long-term intubation.
- Brain penetration—a complication of nasal intubation (Cameron and Lupton 1993).

Chest drain placement

A small number of ventilated preterm infants will develop a pneumothorax and require a chest drain. This may constitute a medical emergency, or at least require expeditious action.

Indications

- Pneumothorax
- Tension pneumothorax
- Pleural effusion

A **pneumothorax** leads to increased work of breathing, hypoxia and hypercapnia. A **tension pneumothorax** not only compromises the respiratory status but leads to decreased cardiac output through reduced venous return to the heart, resulting in hypotension and the possible adverse outcome of intraventricular haemorrhage (see p. 156). **Pleural effusion** can seriously impede lung inflation.

A tension pneumothorax can be relieved temporarily by means of a size 21 butterfly needle attached to a 10ml syringe via a three-way tap. With this inserted

into the second intercostal space in the mid-clavicular line air can be aspirated and eliminated without the need to disconnect the syringe. Cautiously advance the butterfly needle to ensure that the lung is not punctured. An aspiration of this kind should always be followed up with the insertion of a standard pleural chest drain.

Equipment

- Sterile chest drain pack, gown and gloves
- Skin cleansing agent
- 1 % plain lignocaine
- 2 ml syringe and orange needle
- Scalpel with straight blade
- Fine dissecting forceps
- Size 10 or 12fg pleural drain
- Straight connector, Heimlich valve or underwater seal drain
- Suturing material
- Sterile transparent dressing
- Low grade suction apparatus

Procedure

Conduct the procedure under aseptic technique and where a systemic analgesic infusion is in situ give a bolus to cover the procedure. Alternatively, administer a single systemic bolus of analgesia and never insert a chest drain without giving a local anaesthetic: 0.3 ml/kg of 1% lignocaine infiltrated into the tissue provides local anaesthesia in 1–2 minutes (Northern Neonatal Network 1998). Avoid infiltrating the lignocaine into a blood vessel as it can induce cardiac arrhythmia. Give the lignocaine before starting to prepare the equipment to allow local anaesthesia to take place.

Remove any butterfly that may be in situ and have the infant positioned supine or turned slightly to one side with the affected side towards the operator and the arm up at an angle of 90 degrees. This ensures that the site of approach is accessible. The drain is usually inserted in the fourth intercostal space in the anterior axillary line (Figure 13.2). The second intercostal space in the mid-clavicular line is best avoided for cosmetic reasons, especially in girls. The nipple and areolar tissue lie at the fourth rib in this line—not always evident in the very preterm infant.

Infiltrate the site of insertion with the lignocaine, starting superficially and working down into the intercostal muscle and on into the parietal pleura. Once local anaesthesia is achieved, make the incision through the skin and into the intercostal muscle, no wider than the width of the drain to be inserted. Be aware that the intercostal artery, vein and nerve run just beneath the rib. By working above the superior aspect of the lower rib that makes up the space this neurovascular structure can be avoided.

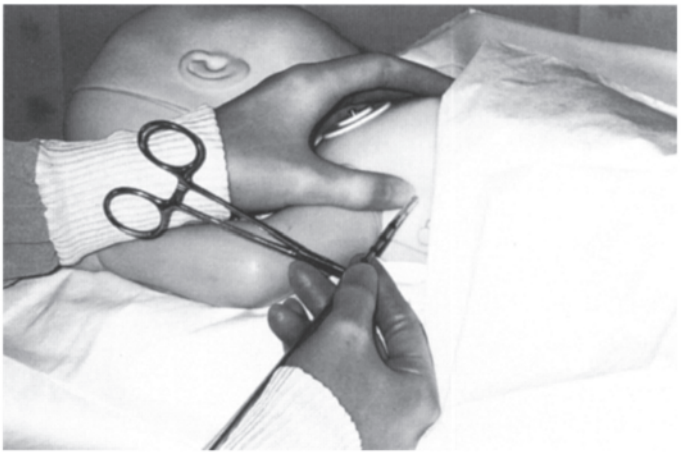

Figure 13.2 **Insertion of a chest drain**

Using fine blunt forceps separate the muscle fibres with a dissecting action until the pleural space is entered. Under no circumstances should the trochar be used to dissect the muscle or pierce the pleura. It must be withdrawn 0.5 cm into the drain and clamped in that position before insertion. Gently ease the drain in along the track that has been created, keeping it tangential to the lung and guiding it anteriorly to where the air tends to accumulate in a supine infant. Insert the drain 2–3 cm for a small preterm infant and 3–4 cm for a term infant, ensuring that the side holes of the drain lie within the pleural space.

When the drain is in the pleural space the trochar can be slowly withdrawn until there is sufficient space to clamp the drain near the skin, after which the trochar can be removed. Misting of the lumen of the drain will indicate that it is in the pleural space. The clamp must remain in position until the drain is connected to a one-way blow off system that will permit air to be removed out of the pleural space but not to enter in. A **Heimlich valve** or an underwater seal drain can be used. If required suction can be applied to these at a negative pressure of -10 to -15 cm H_2O.

Suture the drain in position by first taking the needle in through the skin and out through the incision site and knotting the silk. Repeated blanket stitches around the drain will anchor the drain in position and help to minimise scarring. Secure the blanket stitching by knotting the silk to the tail created by the first knot. Apply a sterile transparent dressing to keep the site clean and allow it to be observed.

Because air is a lighter medium, it collects in the upper part of the chest. In the case of a pleural effusion the fluid gravitates to the lower dependent areas or can become **loculated** in other areas. In order to remove pleural fluid the drain needs to be inserted in the mid- or posterior axillary line using the same technique.

The movement of air through the chest drain needs to be recorded on a hourly basis as part of observations. This will help to determine tube patency and the appropriate time to clamp off or remove the drain.

Complications

- Bleeding from damaged intercostal, mammary, pulmonary, or axillary vessels.
- Intercostal nerve damage.
- Phrenic nerve damage leading to eventration of the diaphragm.
- Horner's syndrome—due to damage of nerve roots C8 to T1
- Thoracic duct injury—any drain that impinges on the structures in the mediastinum should be pulled back.
- Puncture of the spleen, liver or heart.
- Lung damage—a drain wrongly inserted to remove pleural effusion can cause a pneumothorax or a bronchopleural fistula.
- Syncope—occurs when fluid is removed too quickly.
- Secondary infection—always use aseptic technique.

Blood sampling

Obtaining blood samples is an important part of the diagnostic process and the subsequent treatment. Bearing in mind that the total volume of blood in the neonate averages about 80 ml/kg and that 4 ml of blood would represent 5 per cent of that volume in a 1 kg infant, the least amount of blood possible for any test should be taken. Depending on what the blood is required for, three options are available:

- Capillary blood
- Venous blood
- Arterial blood

Capillary blood sampling

Capillary blood is obtained from the heel from the areas beyond the lateral and medial limits of the calcaneus (Figure 13.3).

Indications

- Most routine laboratory tests requiring less than 1 ml of blood
- Metabolic and cytogenetic screening tests
- Blood glucose and lactate analysis
- Acid base balance status

Procedure

Clean the heel with an alcohol swab and allow to dry before applying a thin layer of soft paraffin. This helps the blood to form into globules making collection

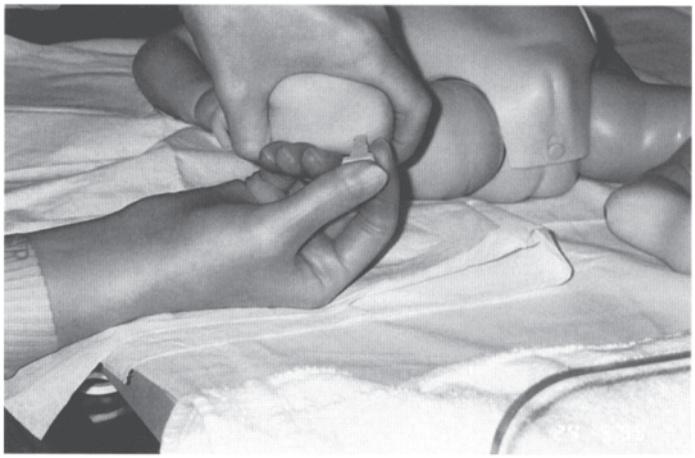

Figure 13.3 **Capillary blood sampling**

easier. Each infant must have their own tube of sterile soft paraffin to prevent infection and cross infection.

With the foot dorsiflexed, pierce the heel to a depth of 1–2 mm in the medial or lateral aspect of the plantar surface. Modern lancets with retractable blades allow the stab to be made to a predetermined depth only and also protect the operator from stick injuries. Gentle squeezing of the heel allows the blood to collect in globules on the paraffin-coated skin which can then be collected into the blood bottle. Samples for acid base balance gravitate into the heparinised tube by a capillary action. Avoid excessive squeezing of the heel and scraping of the blood into the bottle as this causes haemolysis of the sample and leads to inaccurate results. Sample from alternate heels to prevent them becoming very sore.

Complications

- Haemolysis of the sample
- Infection from the puncture site
- Sore heels

Precautions

Capillary pH and PCO_2 do correlate with arterial samples, but PCO_2 values are unreliable. It is important therefore to record the site from which the sample was obtained (Wilkinson and Calvert 1992).

Platelet count and creatinine can be unreliably low and potassium levels unreliably high in a haemolysed sample.

Venous blood sampling

This is preferable under certain circumstances.

Indications

- A large volume of blood is required
- A non-haemolysed sample is of the utmost importance
- Poor peripheral perfusion does not permit capillary sampling
- Blood is required for viral, fungal or bacterial culture
- To accurately diagnose polycythaemia

Procedure

The most common site for venepuncture is the dorsum of the foot or hand. The veins of the antecubital fossa should be avoided if long-term cannulation is anticipated and the femoral vein should not be routinely used because of the risk of damage to the hip joint and the introduction of infection into the joint. Before the vein is punctured the area must be swabbed with a cleansing agent (alcohol- and iodine-free if the infant is very preterm) to avoid the risk of nosocomial infection (see p. 263).

A 19 or a 21G needle can be used or a 21G butterfly. The broken needle technique whereby the hub of the needle is removed before use does make collection straight into the bottle easier. If blood is required for culture an intact needle or cannula should be used and the blood collected from the hub with a needle and syringe or from a butterfly attached to a syringe.

Position the hand or foot so that there is some stasis of blood in the vein and keep the skin taut to prevent the vein moving, then gently insert the needle into the vein at an angle of 45 degrees, ensuring that the flat aspect of the needle tip is applied to the skin and the angled aspect is facing upwards. Once blood flow commences collect the sample without unnecessary squeezing. To remove the needle place a sterile swab over the puncture site and withdraw the needle. Apply gentle pressure until haemostasis is achieved.

Complications

- Infection—this is minimised by asepsis.
- Venous thrombosis—often unavoidable, especially if multiple samples are taken from the same vein.
- Haematoma or haemorrhage—avoidable by applying pressure until haemostasis is achieved.

Arterial blood sampling

There are times when the infant's condition will indicate the need for arterial blood sampling, usually when an arterial pH and blood gas analysis are required but an indwelling arterial sampling device is not justified or when these have failed. The most common sites for sampling are the radial and posterior tibial artery; the dorsalis pedis and temporal arteries can also be used, but the brachial artery has a minimal collateral circulation and there is the risk of damage to the median nerve, so it should be avoided. The femoral vein should also be avoided because of the risk of trauma or the introduction of infection to the hip.

Procedure

The radial artery is the most frequently used. The right radial should be used when a preductal arterial gas is required. Before the sampling is attempted it is imperative that a good collateral circulation is in evidence. This is achieved by elevating the arm and simultaneously occluding the ulna and radial arteries at the wrist. Gentle pressure on the palm of the hand will cause it to blanch; releasing the pressure on the ulnar artery should be followed by the return of normal colour to the hand within 10 seconds (Allen's test).

Locate the artery by palpating it in the normal manner or by transillumination from the opposite side of the wrist with a cold light source. After cleansing the area, puncture the artery just proximal to the transverse wrist crease with a 25G hypodermic needle at an angle of 45 degrees to the skin and slowly advance the needle in the opposite direction of the blood flow (Figure 13.4). The vessel is superficial and can be easily transfixed by the needle. When this happens blood flow can be established by slowly withdrawing

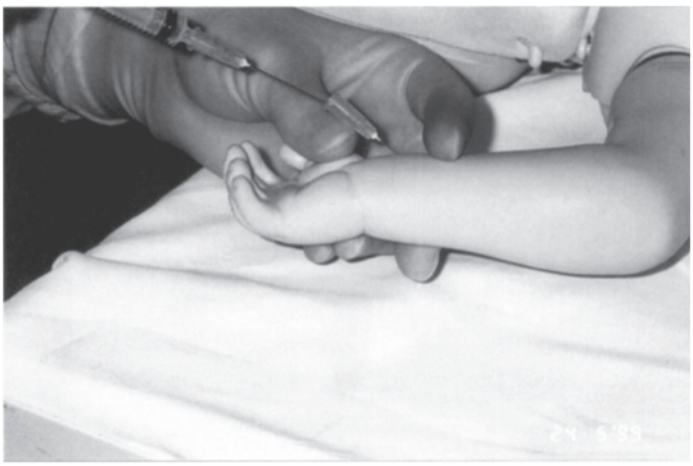

Figure 13.4 **Arterial stab (right radial artery)**

the needle. Over-extension of the wrist can also cause occlusion of the vessel. The blood to be collected into a heparinised syringe requires very little suction on the plunger. To collect directly into a heparinised capillary tube place it at the hub of the needle and allow it to fill by capillary action and gravity. Collect the amount of blood required before withdrawing the needle and then immediately covering the puncture site with a sterile swab and applying gentle pressure until haemostasis is achieved.

Complications

- Arteriospasm or ischaernia of surrounding tissue—minimised by using the smallest gauge needle and keeping punctures to a minimum.
- Haematoma and haemorrhage—prevented by correct withdrawal of the needle and ensuring haemostasis has occurred.
- Infection—rare and minimised by using strict sterile procedure.
- Inaccurate blood gas result. Too much heparin may lead to a falsely low pH and PCO_2. Air bubbles in the sample can result in a falsely high PaO_2 and low $PaCO_2$. Even in an infant who does not cry, intolerance to the procedure can cause a fall in the PaO_2 during sampling. This is important if there is a concern about hyperoxia in the premature infant (Wilkinson and Calvert 1992).

Peripheral vein cannulation

This is the most commonly used method of access to the circulatory system and any superficial vein can be cannulated. The most preferred sites are the dorsum of the hand (Figure 13.5) and foot and the forearm. The basilic and cubital vein at the bend of the elbow, and the greater saphenous at the ankle, also provide

Figure 13.5 **Venous cannulation in the dorsum of the hand**

good access but should be left untouched in those infants likely to require a centrally placed long line. Scalp veins provide the same access but are usually only used when no other vein is accessible.

Indications

- Administration of intravenous medication and fluid, blood and blood products
- Administration of parenteral nutrition

Equipment

- 24G cannula, or a 22G cannula for very premature infants
- T-piece extension set attached to a 2 ml syringe, both primed with water for injection or 0.9% sodium chloride
- Cleansing agent with sterile cotton wool balls
- Transparent sterile dressing
- Splint and securing tape

Procedure

Prepare all the equipment beforehand and ensure that the needle that runs down the centre of the cannula pulls out easily before attempting to use it. If the infant is very vigorous ask for assistance before starting the procedure. Identify the vein to be cannulated and cleanse the area over and around the site of access.

With one hand hold the skin taut to immobilise the vein, then puncture the skin a few millimetres proximal to the vein with the tip of the needle protruding out of the cannula, ensuring that the bevelled side is facing upwards. Enter the vein from the anterior surface or from the side by advancing the needle 1–2 mm in the direction of the flow of blood; this will ensure that the cannula is in the vein lumen. A flashback of blood will confirm the position. Hold the hub of the needle with the thumb and third finger and gently ease the cannula off the needle and into the vein with the index finger.

Once the cannula is in position the needle can be completely removed. This should be accompanied by a flow of blood into the cannula hub. The primed T-piece can now be attached to the cannula and used to flush it; this will confirm that the vein has been successfully cannulated.

Before applying the transparent dressing protect the skin with one of the available skin preparations. If the site of cannulation is over a joint in a limb immobilise the joint with a splint.

Resistance to or evidence of flush entering the surrounding tissue will indicate the cannula has been misplaced. It should be removed. Hourly observation of the cannula site is imperative. This will help to minimise the number of times

that extravasation occurs, which can result in damage to the surrounding tissue and scarring.

Complications

- Infiltration of the subcutaneous tissue—avoid advancing the cannula with the needle still protruding through the end as this will damage the vein lumen.
- Emboli or clot—always flush the cannula with a fluid-primed T-piece.
- Haematoma—caused by damage to the vessel during cannulation.
- Vasospasm—occurs rarely and resolves spontaneously.
- Infection—can be minimised by adopting an aseptic technique and cleansing the skin before cannulation.
- Phlebitis—can occur when highly irritant substances are infused through the cannula or when it remains in situ for a protracted length of time.

Peripheral artery cannulation

There are occasions when sequential arterial blood gas analysis is essential but can only be obtained from a cannula placed in a peripheral artery.

Indications

- Umbilical arterial catheterisation is not possible
- Arterial access is required for blood gas analysis and invasive blood pressure monitoring

Equipment

- 24G cannula, or 22G for a very premature infant
- T-piece extension attached to a 2 ml syringe, both primed with water for injection or 0.9% sodium chloride
- Three-way tap with Luer lock device
- A 50 ml syringe of 0.45% or 0.9% sodium chloride with heparin (1 unit: 1 ml) primed through an infusion line with a Luer lock device
- Infusion pump, pressure transducer and manometer tubing
- Skin cleansing agent
- Transparent dressing

Procedure

The arteries most commonly used for this procedure are the radial and posterior tibial because both have collateral supplies from the ulna and dorsalis pedis arteries

respectively. Locate the artery as described for arterial puncture (see p. 297) and verify a collateral circulation is evident before cannulating the artery in the opposite direction of the blood flow. Once the cannula is in the artery blood flow will be brisk. The T-piece extension is now connected to the cannula and the artery is slowly flushed clear of blood before clamping the T-piece. Secure the cannula in place with a transparent sterile dressing and splint if appropriate. Ensure that the digits are left visible to enable them to be observed. Quickly attach the heparinised infusate to the T-piece via the three-way tap and commence the infusion at 0.5 ml per hour. Attach to monitoring equipment if blood pressure assessment is required.

Complications

- Vessel damage. Slow infusion of heparinised **isotonic** solution will help to minimise damage to the intima of the vessel and reduce clots. Do not use the cannula for infusion of other fluids or administration of drugs.
- Arterial spasm. Avoid excessive suction when sampling from the cannula. Allow the blood to flow freely if possible. Any persistent blanching or discoloration of the extremities requires immediate removal of the cannula.
- Infection. Use aseptic technique during cannulation.

Peripheral venous longline

This kind of central venous access is usually required in the very preterm or sick infant. The advantages over the venous cannulae are that these lines last longer and can safely be used for inotropes, hypertonic and hyperglycaemic solutions and are most useful when prolonged use of parenteral nutrition is required. There are several kinds of lines available. The smallest comes with a guide wire in situ and can be threaded through a 22G cannula; the most frequently used is a 23G Silastic catheter that is threaded through a 19G butterfly. The largest is introduced using the Seldinger technique; this will not be discussed here.

Indications

- Administration of inotropic medication
- Administration of hypertonic or hyperglycaemic solutions
- Administration of parenteral nutrition
- Administration of antifungal medication

Equipment

- Sterile longline pack, gown and gloves
- Prepared sterile pack containing the Silastic catheter or finer wired catheter and 22G cannula

- Cleansing agent
- A 2 ml syringe with sterile water or 0.9% sodium chloride (used to prime the Silastic catheter only)
- Sterile transparent dressing

Procedure

Box 13.2 **Peripheral venous longline length estimation in centimetres**

Measure from the site of insertion to the approximate position of the right atrium

Before commencing the procedure, identify the vein to be used. A large vein such as the long saphenous, medial and lateral antecubital veins or the superficial temporal vein are best. To estimate the length of line required measure in centimetres the distance from the site of insertion to the approximate position of the right atrium. Adopting a strict aseptic technique, cleanse the area over the selected vein and any area that will ultimately lie under the transparent dressing and drape the surrounding area.

To insert the fluid-primed Silastic line, first access the vein with the 19G butterfly needle and thread the line through it. Blood flow is usually brisk and can push the line out of the butterfly. Gentle pressure applied to the vein can help overcome this. Using non-toothed forceps, advance the line up to the proximal right atrium. Be aware that the butterfly needle is approximately 5 cm in length and this dead space needs to be taken into account when advancing the line. Once it is in place, disconnect the line from the blue compression mounting and slide the butterfly out of the vein and off the line. Applying gentle pressure with a swab at the site of the butterfly insertion will prevent the line coming out with the butterfly.

Reassemble the connections and flush the line with the priming fluid. This will indicate that it has not become impacted during insertion. Coil the remaining line into loops on the skin and apply a small piece of sterile gauze under the blue compression mounting before covering the whole area with a sterile transparent dressing. Always check the position of the line radiographically with 0.5 ml of a radio-opaque fluid in situ before putting any infusate through it.

The finer wired lines are placed in position in exactly the same way with the exception that they are inserted through a 22G cannula and are not primed with fluid beforehand—this would necessitate the removal of the guide wire. They need to be examined radiographically immediately after insertion with the guide wire in situ, after which it is removed. Do not attempt to remove the guide wire and inject a radio-opaque medium as this will occlude the line. An infusate should be commenced immediately the position has been verified. Some makes of this line allow the cannula to be split to

enable it to be removed; for those that don't, remove the cannula from the vein and pull it loosely up the line and on to the hub before including it under the transparent dressing.

Complications

- Prolonged bleeding from the insertion site—always ensure there is stasis of blood before applying the dressing.
- Shearing off of the line on the point of the butterfly—never pull back a line with the butterfly in situ.
- Infection—insert under strict asepsis. Any unexplained pyrexia or signs of infection may necessitate the removal of the line, as will any erythema or swelling along the track of the line.

Umbilical vessel catheterisatien

The availability of these vessels places the infant in a unique position and provides a direct route to the great vessels of the body. The umbilical vessels allow ease of access for arterial blood gas analysis and other blood sampling, blood pressure and central venous pressure monitoring and a route through which blood and blood products, fluid and drugs can be administered (Klaus and Fanaroff 1993).

Umbilical vein catheterisation (UVC)

This is the easier of the vessels to catheterise and the vein will usually accommodate catheters at the larger end of the spectrum.

Indications

- Emergency infusion of resuscitation drugs and blood in infants depressed, shocked or asphyxiated at delivery (see p. 51)
- Exchange transfusion (see p. 175)
- Central venous pressure monitoring

Equipment

- Umbilical vessel catheterisation pack, containing instruments and cord ligature
- Sterile gown and gloves
- Scalpel and blade
- Umbilical catheter size 4 or 5fg attached to a three-way tap (marked blue as venous) with a Luer lock and primed with fluid from a 2 ml syringe

- A 50ml syringe of 0.45% or 0.9% sodium chloride with heparin (1 unit: 1 ml) attached to and primed through a line with a Luer lock device
- Pressure transducer and monitoring equipment
- Cleansing agent

Procedure

Prepare all the equipment beforehand, with the catheter attached to the three-way tap and both primed with the 2 ml syringe of fluid. Place the tap in the off position to the baby. This procedure will require an aseptic technique. Place the infant in the supine position. A vigorous infant may need to have the lower limbs restrained. Ensure that the facility to monitor the infant's heart rate and saturation is in place, and that the infant's face can be observed for colour changes throughout the procedure. Illuminate the area with a good source of light before you begin.

Clean around the cord and umbilical area with an aqueous-based cleaning agent. Iodine and agents with an alcohol base can very easily burn the skin, especially in the very preterm infant, if applied liberally and allowed to run down to the lumbar area unobserved. Cover the chest and abdomen in sterile drapes, but ensure that you can observe the infant's face for changes in condition at all times.

To estimate the number of centimetres that the catheter needs to be passed calculate 1 1/2 times the infant's weight in kilograms and add 5 1/2. Place the cord ligature around the base of the cord and tighten just sufficiently to stem any oozing of blood from the cord once it is cut. If pulled too tight it can damage the vessels. Apply artery forceps across the cord about 2 cm from the base and cut across on the underside of the forceps with the scalpel. The vessels run a spiral route down the cord which makes them difficult to access if the cord is left too long. The umbilical vein is found in the 12 o'clock position and is the largest vessel, with a gaping appearance, and is more prone to bleed at this point. The two arteries lie inferior to the vein in the 8 and 4 o'clock positions. They are smaller and white in appearance. Stabilise the cord with the artery forceps and apply a gentle caudal traction. Place the tip of the primed catheter into the vein and advance in an upwards direction through the ductus venosus into the inferior vena cava proximal right atrium. There is usually no need to dilate the vein. If resistance is felt the catheter may have entered the portal system. Withdraw the catheter 1–2 cm, twist it and re-advance it. Once the catheter is advanced to the desired length a free flow of blood can be aspirated, but the position needs to be checked by X-ray.

Box 13.3 **Umbilical venous catheter length estimation in centimetres**

(1.5×weight in kg) + 5.5

The catheter needs to be anchored by first securing the suture to the cord and then applying several blanket stitches to the catheter before tying it off to the tail of the initial suture. A 'goal post' device constructed from adhesive tape attached to the abdomen at each side of the cord and across the top incorporating the catheter provides a further means of attachment. A continuous heparinised infusate needs to be attached to the catheter with a manometer if venous pressure monitoring is required and the catheter tip needs to be placed 1 cm above the diaphragm precisely to ensure that the measurement is accurate (Wilkinson and Calvert 1992).

Complications

- Air embolus can occur if the line is left open to the atmosphere.
- Hyperosmolar solution (>300 mOsm/kg H_2O) can cause hepatic necrosis, thrombosis and portal hypertension if infused into the liver. Remove an umbilical vein catheter that is placed in the liver (Wilkinson and Calvert 1992).

Umbilical artery catheterisation (UAC)

The size of catheter used in the artery varies from 3.5 to 5.0fg and is determined by the size of the infant. End hole catheters are generally the catheter of choice because they are less likely to cause damage to the endothelium of the aorta and block less easily than side hole catheters. These do, however, give a more reliable blood pressure reading (Wiendling 1989). Complications usually arise at the tip of the catheter so precise placement is essential. This is achieved by placing the catheter tip above the diaphragm at thoracic vertebrae 6–10 for the high position and lumbar vertebrae 3–5 for the low position. These positions avoid the major branches of the aorta. Thrombotic complications have been reported with catheters placed in both the high and low positions (Goetzman *et al.* 1975; Mokrohisky *et al.* 1978). The prolonged use (more than one week) may increase the incidence of abdominal symptoms and alter intestinal blood flow (Kempley and Gamsu 1992).

Indications

- Frequent arterial blood estimations of pH, PaO_2 and $PaCO_2$ (some special catheters permit continuous PaO_2 and SaO_2 recording to be made and others that have the addition of pH monitoring are currently being marketed)
- Continuous arterial blood pressure monitoring
- Exchange transfusion (see p. 175)

Equipment

- Sterile umbilical vessel catheterisation pack, containing instruments and cord ligature
- Sterile gown and gloves
- Scalpel and blade
- Umbilical catheter size 3.5–5.0fg (choice will depend upon the size of the infant) attached to a three-way tap (marked red as arterial) with a Luer lock and primed with fluid in a 2 ml syringe. Close the three-way tap to the catheter before insertion
- A 50 ml syringe of 0.45% or 0.9% sodium chloride with heparin (1 unit: 1 ml) attached to and primed through a line with a Luer lock
- Pressure transducer and monitoring equipment
- Cleansing agent

Procedure

Prepare as described for UVC. To estimate the number of centimetres that the catheter needs to be passed multiply the infant's weight in kilograms by 3 and add 9, and then add the number of centimetres of cord left after cutting. This will give the length required to place the catheter in the high position.

Box 13.4 **Umbilical arterial catheter length estimation in centimetres (high position)**

(3×weight in kg) + 9 + length of umbilical cord stump in cm

Proceed as for an umbilical venous catheterisation. Identify the two arteries but only attempt to catheterise one. This will enable a second person to make an attempt should the first be unsuccessful. Stabilise the cord with the artery forceps but ensure that they do not impinge on any of the vessels, and then gently tease the lumen of the artery open with the non-toothed forceps followed by the probe. Patience is needed as the intima is easily damaged and undue-force can lead to a false passage. Once the vessel is sufficiently dilated the tip of the prepared catheter can be introduced into the artery and gently but firmly advanced to the estimated position.

Some resistance may be felt at the level of the umbilical wall and the internal iliac junction. A sustained but gentle pressure can sometimes overcome this. Once in place, open the three-way tap to the catheter and aspirate with the 2 ml syringe to obtain arterial blood. Arterial pulsation should be seen. Secure in the same way as described for venous catheterisation and attach to the heparinised solution and manometer as soon as possible.

The position should be verified by X-ray, which must include the whole chest and abdomen. The positions of the UAC and UVC are more clearly defined on a lateral film, but one taken in the anteroposterior position will show the UAC passing down the anterior abdominal wall into the pelvis before ascending the abdominal and thoracic aorta, along the left of the vertebrae. The UVC passes straight upwards through the ductus venosus into the inferior vena cava along the right of the vertebrae. The exact location of the tip can be determined by counting down the thoracic or lumbar vertebrae to where it is positioned (Meerstadt and Gyll 1994).

Regular zeroing and flushing of blood from the blood pressure dome will ensure that blood pressure monitoring remains accurate, as will ensuring that the dome is placed at the approximate level of the heart. Damping of the transduced waveform will indicate partial occlusion of the line and can usually be remedied by flushing. Observing the lower limbs for discoloration or blanching will pre-empt any ischaemic injuries.

Complications

- Infection—insert the catheter using strict asepsis. If the catheter is not placed in the correct position no attempt should be made to advance it further as this represents an infection risk.
- Vascular accidents—vasospasm, thrombosis, or infarction can lead to ischaemic injury to the lower limbs and buttocks.
- Haemorrhage—loose connections in the line can lead to loss of blood and exsanguination of the infant.
- Vessel perforation—never force the catheter into position; surgical intervention may be required to correct a perforation.

Lumbar puncture

Some controversy surrounds the use of lumbar punctures, with some units carrying out the procedure every time there is an infection screen work-up, yet others only doing so when a central nervous system disorder or infection is suspected (see p. 273).

Indications

- Diagnostic purposes for central nervous system disorders, such as meningitis and subarachnoid haemorrhage
- To drain cerebrospinal fluid and measure pressure in a communicating **hydrocephalus**
- To administer **intrathecal** medication
- To monitor assay levels of drugs used to treat central nervous system infections

Equipment

- Sterile dressing pack, gown and gloves
- Cleansing agent
- Spinal needle (22G, 1 inch length)
- Lignocaine 1% with syringe and needle (optional)
- Plain sterile bottle×2, glucose bottle×1
- Transparent dressing spray

Procedure

The position adopted for this procedure in the neonate is the left **lateral decubitus,** with head and knees flexed. With an assistant holding the infant in position, the interspace between lumbar vertebrae 4 and 5 can be identified by following a line down from the iliac crest to lumbar vertebra 4.

Adopting an aseptic technique, cleanse the area using a widening circle starting at the area identified as the site of entry for the spinal needle and taking it up over the iliac crest. Drape the area leaving only the selected interspace exposed and identify the interspace again.

Local anaesthesia is provided by 0.3 ml/kg of lignocaine 1%, injected subcutaneously. This will provide local anaesthesia within 1–2 minutes (Northern Neonatal Network 1998), but may be more painful than a well-performed procedure and is therefore not always used.

To advance the needle, hold the thumb of the left hand on L4 and with the right hand insert the needle in the midline of the interspace below. With steady force, direct it slowly towards the umbilicus. The 'give' as the ligamentum flavum and dura are penetrated is not always evident in the neonate and this necessitates the frequent withdrawal of the trochar from the needle to ensure that it is not advanced too far, leading to a bloody specimen. For routine investigation collect 5–6 drops of cerebrospinal fluid in each of the specimen bottles for:

- Gram stain, culture and sensitivity
- Glucose and protein levels
- Cell count and differentials

Replace the stylet and withdraw the needle and with a fresh sterile swab apply pressure over the area until any flow of CSF has stopped. A transparent spray dressing can now be applied.

Complications

- Blood in the sample. If it clears as the CSF flows it indicates a traumatic tap; if it persists and forms a clot a blood vessel has probably been punctured. In an infant who has had significant intraventricular haemorrhage blood will appear in the sample.

- Infection—reduced by using strict asepsis.
- Interspinal epidermoid tumour—the result of using a needle without a trochar. The needle displaces a plug of epithelial tissue into the dura as it is advanced.
- Herniation of cerebral tissue through the foramen magnum—uncommon because of the open fontanelle in neonates.
- Spinal cord and nerve damage—prevented by using an interspace below L4.
- Hypoxia, apnoea and bradycardia resulting from respiratory compromise when the head is too flexed.

Urine sample collection

Urine can be obtained in one of three ways:

- Straight from cotton wool balls placed between the thighs
- Urine bag placed on the perineum
- Suprapubic aspiration of the bladder

Indications

- Biochemical analysis:
  (a) Semi-quantitative stick testing for glucose, ketones, urobilinogen, reducing substance, bilirubin, blood and pH estimation. A 'cotton wool ball' sample is adequate for this.
  (b) A quantitative analysis of osmolality, urea and electrolytes. This is more accurate if taken from a freshly voided bag specimen.
- Microbiological analysis, microscopy, culture and sensitivity. This sample should be collected avoiding any contamination of the urine. A 'clean catch' specimen or urine obtained from a suprapubic aspiration provides the best sample. A sample from a bag placed on a cleansed perineum can also suffice, but contamination is possible.

Suprapubic aspiration

Equipment

- A 10 ml syringe attached to a 21G needle
- Skin cleansing agent and sterile gauze squares
- Sterile gloves
- Sterile universal container

Procedure

Ensure that the bladder is full—ultrasound scanning will be helpful here. Place the infant supine and have the legs held in the frog position by the assistant. Cleanse the area above the symphysis pubis, then with the needle and syringe held at an angle of 90 degrees enter the bladder through the skin and abdominal wall 0.5–1 cm above the symphysis pubis in the midline (Figure 13.6). This may be done under ultrasonography. Advance the needle to a depth of 1–2 cm whilst aspirating the syringe. Once urine enters the syringe stop advancing the needle; this will help to avoid perforation of the posterior wall of the bladder. When the sample is collected, withdraw the needle and apply pressure at the site. The fresh sample should be sent straight for processing.

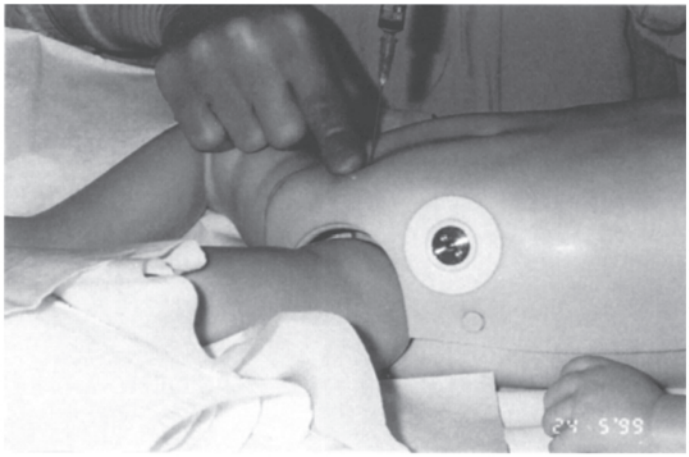

Figure 13.6 **Suprapubic aspiration**

Complications

- Microscopic haematuria can occur after aspiration but is usually transient. The procedure should not be carried out on any infant with a bleeding disorder.
- Bowel perforation can occur if the needle is advanced through the posterior wall of the bladder. Faecal matter may become evident on aspiration. The procedure should not be carried out on any infant with abdominal distension or dilated loops of bowel.

Case studies 1–3: interpretation of three neonatal chest X-rays

The following three case studies with the accompanying X-rays serve to illustrate some of the guidance given within the text when undertaking procedures in the neonatal clinical setting.

Case study 1

Justin was born at 28 weeks' gestation. He was a second twin delivered by emergency cesarean section after the mother presented in advanced labour and the infants were noted to be of unstable lie. The mother received one dose of antenatal steroids approximately 30 minutes prior to delivery.

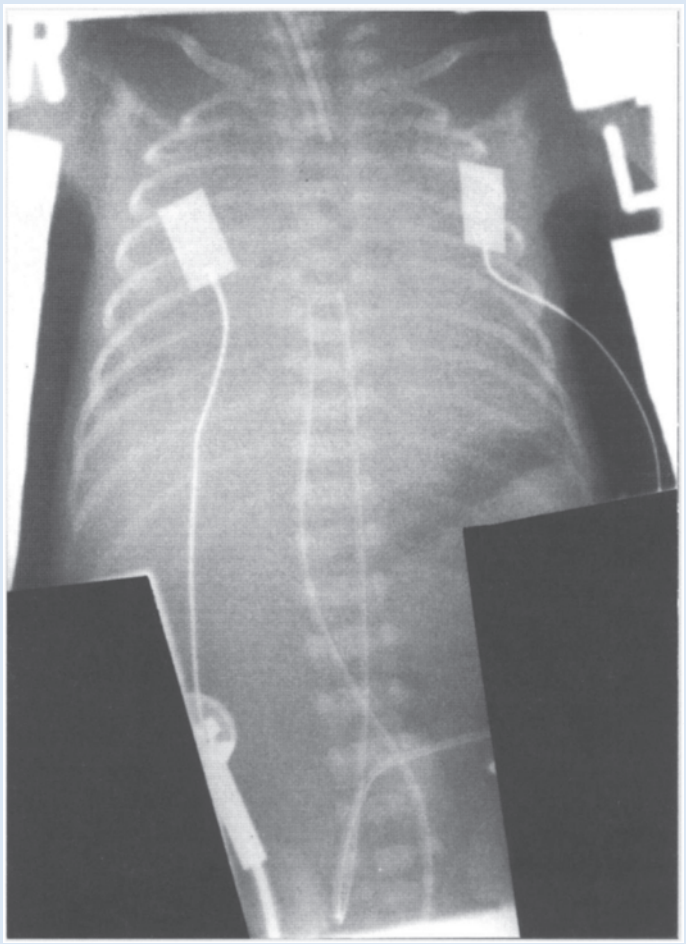

***Figure 13.7* Case 1**

Justin was delivered in a reasonable condition but at 2 1/2 minutes of age became apnoeic and bradycardic and the response to bag and mask ventilation, though good, was not sustained. He was intubated at 3 1/2 minutes of age and given the first dose of endotracheal surfactant at 5 minutes of age. On transfer to the neonatal unit umbilical arterial and venous catheters were sited. The chest and abdominal X-ray (Figure 13.7)

shows a classic respiratory distress syndrome appearance. The UAC enabled ventilatory requirements to be more precisely managed. The UVC was sited to enable inotrope support to be given in the first 24 hours of life should it have been required.

The endotracheal tube is well placed at the second thoracic vertebra (T2). The UAC seen in Figure 13.7 on the left of the spinal column enters the umbilical artery and runs down the anterior abdominal wall into the pelvis where it enters the internal iliac artery and ascends up through the abdominal and thoracic aorta to be well positioned at the level of T7.

The UVC seen on the right side of the spinal column enters the umbilical vein which runs up the falciform ligament to the liver where it joins the ductus venosus. This is drained by the hepatic vein into the inferior vena cava just before passing through the diaphragm. The tip lies at T7 in the proximal right atrium. There is no evidence of a gastric tube in position, although it is advisable to pass the gastric tube prior to the film being taken as the position can then be verified at the same time.

Case study 2

Rosa was born by normal vaginal delivery at 29 weeks plus 4 days' gestation and was her mother's third baby, the two previous siblings being born at full term. Labour was precipitous and no antenatal steroids were given. Rosa was in 25–28% head-box oxygen for the first 7 hours of age and had mild grunting with nasal flaring only when disturbed. By 8 hours the oxygen requirement had started to increase rapidly and an arterial stab blood gas analysis showed a respiratory acidosis. Intubation was carried out on the basis of the blood gas and endotracheal surfactant was given. An X-ray (Figure 13.8) carried out at this time showed a left-sided pneumothorax. A chest drain was sited in interspace T3–4 which initially drained the pneumothorax. Re-accumulation of air necessitated a second drain insertion, which was sited slightly low in the T6–7 interspace, and a third drain was sited in the T9–10 interspace when Rosa developed a oxygen requirement of 100% and transillumination of the chest with a cold light revealed a large anterior collection of air. The air leak finally resolved when the middle and lower chest drains were removed and a new drain was inserted in the middle position but angled in a more apical direction. The endotracheal tube is situated at T2–3. The gastric tube should have been passed prior to the film being taken to allow concurrent verification of the position.

Case study 2 continued

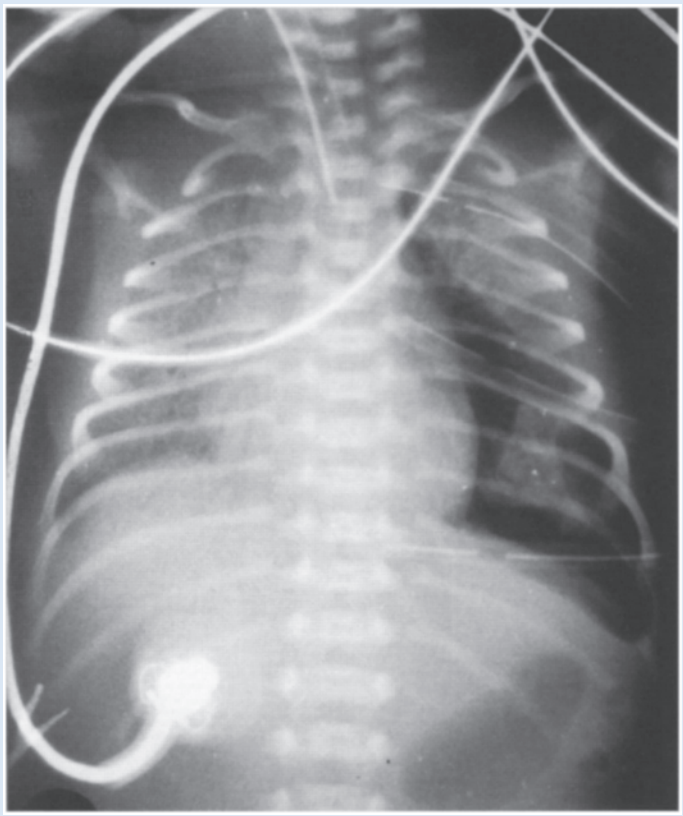

***Figure 13.8* Case 2**

Case study 3

Selina was delivered at 30 weeks' gestation by normal vaginal delivery. Her mother had experienced spontaneous rupture of membranes 56 hours before delivery. Two doses of antenatal steroids had been administered. Despite this, Selina exhibited signs of respiratory distress at delivery and was electively intubated and given endotracheal surfactant. An X-ray (Figure 13.9) was taken to ascertain the position of the central venous longline sited in the right antecubital fossa on day 2 of life to enable total parenteral nutrition to be given. The line was noted to be below the

diaphragm in the inferior vena cava and was pulled back 4 cm. The X-ray shows that the UAC lies at the level of T10, just adequately positioned (noted definitely to be a UAC on a previous film). The orogastric tube can be seen to reach only as far as the distal oesophagus; this was replaced. The endotracheal tube is high at T1, but good ventilation was achieved and it was therefore left in that position.

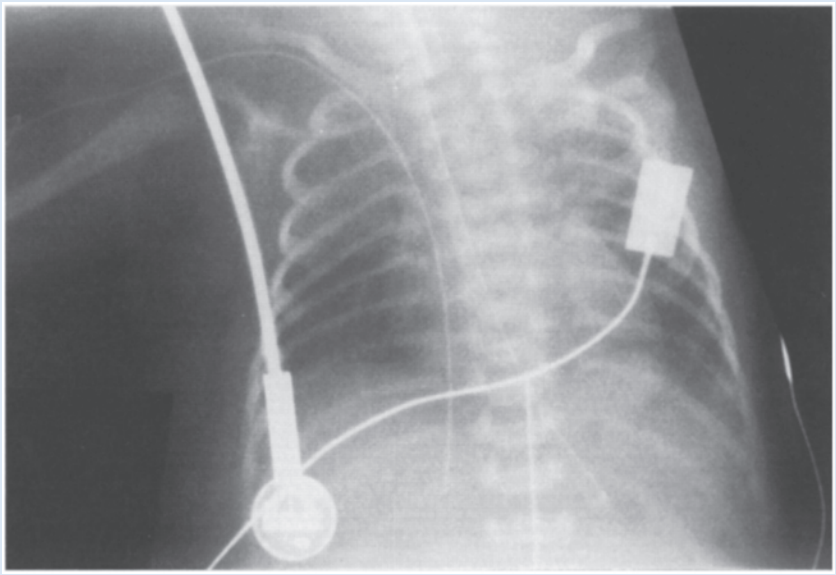

***Figure 13.9* Case 3**

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Chapter 14

Chapter 14

Neonatal Anaesthesia

Contents

Introduction

The demand for neonatal anaesthesia has grown enormously in recent decades, due to the innovations in neonatal medicine and surgery. This has resulted in large numbers of premature infants presenting for urgent surgical procedures, many of which are to correct serious congenital abnormalities or to deal with conditions peculiar to the neonatal period (for example, severe necrotising enterocolitis).

The technical skills required to anaesthetise a neonate safely are very demanding and difficult to acquire: only a minority of anaesthetists possess such skills. In addition, there are marked differences in the anatomy, physiology and pharmacology of neonates compared to adults or even older children. The neonatal anaesthetist should have these technical skills and specialised knowledge and be acquainted with the differing patterns of disease in the neonatal age group.

Preoperative assessment

All neonates must be carefully assessed preoperatively by the anaesthetist, with the emphasis on detection of the following:

Problems of prematurity affecting anaesthesia

Pulmonary disease

The incidence of respiratory distress syndrome (RDS) and chronic lung disease increases with decreasing gestational age at birth, with infants of less than 32 weeks' gestation being at particular risk. Occasionally it is necessary to perform urgent surgery on an infant during the initial respiratory illness. Many of these infants will be on mechanical ventilation, continuous positive airway pressure (CPAP) or require oxygen supplementation preoperatively. Careful note should be made of mechanical ventilatory settings, since minor ventilatory changes perioperatively can result in serious sequelae such as pneumothorax and pulmonary hypertensive crises. Even after minor surgery, infants with lesser degrees of RDS may require a period of postoperative ventilation.

Ex-premature infants with chronic lung disease may require preoperative physiotherapy, and are prone to bronchoconstriction which may benefit from preoperative inhaled bronchodilators or steroids. In addition, they may have subglottic stenosis as a complication of long-term intubation and in particular recurrent intubations (Ratner and Whitfield 1983). Any evidence of inspiratory stridor or upper airway obstruction may suggest this problem.

Cardiovascular disease

There is an increasing incidence of patent ductus arteriosus (PDA) with decreasing birth weight, with an incidence of 80 per cent in preterm infants weighing less than 1000 g (Hollinger 1987). Clinical features include a dynamic precordium, a systolic murmur and signs of congestive cardiac failure. A significant PDA should be treated before surgery for other conditions if at all possible (see p. 140).

Persistent pulmonary hypertension results from constriction of the pulmonary vascular bed causing a delay or reversal of the transition from fetal to adult circulation. It most frequently occurs in response to acidosis, hypoxaemia or hypotension, all common in the sick surgical neonate (Rutter 1993). The result is a right-to-left shunt of blood at atrial and ductal level leading to worsening hypoxaemia and acidosis. Attempts should be made to reduce pulmonary vascular resistance before surgery by treating the underlying causes and by using pulmonary vasodilator drugs if necessary.

Glycaemic control

Carbohydrate reserves in all neonates are low but preterm infants have particularly poor reserves because glycogen is not stored until 36 weeks' gestation. Hypoglycaemia is therefore likely to accompany any significant period of preoperative starvation and must be avoided. In very preterm infants normoglycaemia is maintained by commencing an intravenous glucose infusion at the commencement of the starvation period.

Thermoregulation

The ability of neonates to maintain a normal core temperature is limited by a large surface area: volume ratio, immature sweating, poor insulation (due to less body fat) and a high basal metabolic rate, with preterm infants being at greatest risk of developing hypothermia (Hey 1972). The range of ambient temperatures over which the core temperature of the infant is maintained at between 36.7 and 37.3 °C while metabolic heat production is kept at a minimum is known as the neutral thermal environment (Sauer *et al*. 1984). This may be as high as 36 °C in a 1000 g infant soon after birth (see Chapter 4).

Associated congenital/chromosomal abnormalities

Surgery in the neonatal period is frequently for the correction of congenital abnormalities. Many of these may be associated with other abnormalities, which should be actively sought preoperatively. The following are of particular relevance to the anaesthetist:

Congenital heart disease

Some cardiac abnormalities may not present until several days or even weeks after birth, with symptoms often arising following closure of the ductus arteriosus. A high index of suspicion is therefore necessary, particularly if a heart murmur is detected on clinical examination and there are accompanying signs of cardiorespiratory impairment.

Important considerations in a baby with congenital heart disease include the direction of any intracardiac shunt, the presence of cyanosis or cardiac failure, the dependence of the circulation on flow through the ductus arteriosus and drug therapy. The baby's condition should be optimised before surgery in conjunction with neonatologists and paediatric cardiologists if necessary (see p. 139).

Airway abnormalities

The presence of a cleft lip and palate or of a more severe facial abnormality such as Pierre Robin syndrome or cystic hygroma (see p. 369) may make tracheal intubation difficult (Figure 14.1).

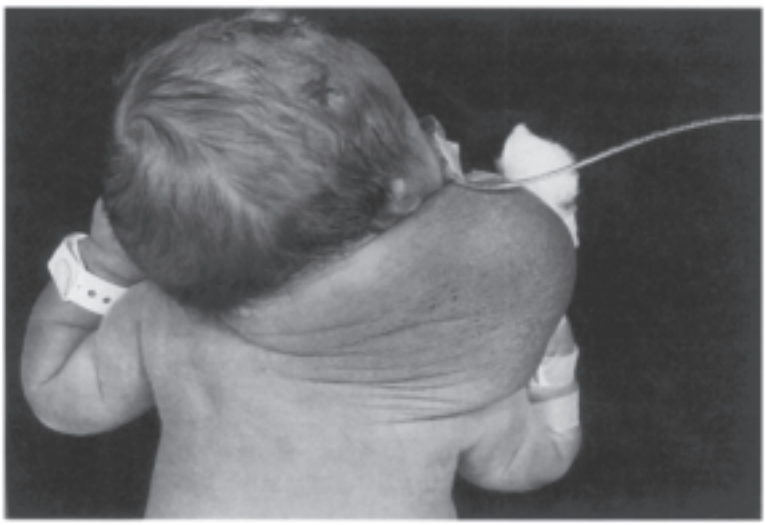

Figure 14.1 **A neonate with a cystic hygroma. These lesions arise from the posterior triangle of the neck and can produce considerable airway management problems**

Down's syndrome (trisomy 21)

Common abnormalities include congenital heart disease, upper cervical spine instability and a large tongue (which may make airway management difficult) (Mitchell *et al.* 1995).

Familial anaesthetic problems

Most neonates will be undergoing their first anaesthetic. A history of any familial anaesthetic complications—particularly the development of malignant hyperpyrexia or suxamethonium apnoea under anaesthesia—should therefore be sought by the anaesthetist.

Preoperative investigations and preparation

Investigations

The need for preoperative investigations will depend on the age and clinical condition of the baby and the nature of the proposed surgery.

WEIGHT An accurate weight preoperatively is essential in order to calculate appropriate drug doses and fluid volumes.

HAEMOGLOBIN The haemoglobin concentration (Hb) should be measured in all infants except healthy term babies undergoing minor surgery. The Hb at birth varies with gestational age (Roberton 1986), with a normal value of 14.5 g/dl at 28 weeks rising to 15.5–17.0 g/dl at term. The Hb subsequently declines, the fall being greater in preterm babies due to lower red cell survival and poorer production of red cells (Chessell 1979). An Hb less than 10 g/dl is abnormal and should be corrected before surgery. Anaemia in preterm infants is associated with an increased incidence of postoperative apnoea.

CLOTTING STUDIES These should be carried out in neonates with sepsis (for example, necrotising enterocolitis) or jaundice, and abnormalities corrected where possible preoperatively.

BLOOD ELECTROLYTES Serum sodium and potassium concentrations should be measured in any infant receiving intravenous fluids or diuretic therapy. Ionised calcium levels fall during the first week of life with a slow rise to adult levels during the second week. The fall is greatest in low birth weight infants. Calcium levels should be checked preoperatively in any preterm or sick infant and levels below 1.75 mmol/l corrected. This is because severe hypocalcaemia can cause seizures and generalised hypotonia.

BLOOD GLUCOSE Blood glucose concentration should be monitored closely perioperatively and then at least 4-hourly until satisfactory glucose homeostasis is achieved.

BLOOD CROSS-MATCH This is essential if major surgery is proposed for any infant and for more minor surgery in very low birth weight infants, in whom small amounts of intraoperative blood loss may be very significant.

OXYGEN SATURATION AND BLOOD GAS ANALYSIS Oxygen saturation is easily measured non-invasively with a pulse oximeter and should be recorded in all infants preoperatively. Blood gas analysis should be performed in babies with severe cardiorespiratory disease or with sepsis.

CHEST X-RAY A chest X-ray is required in babies with cardiorespiratory disease, those on mechanical ventilation and those with a tracheo-oesophageal fistula or a diaphragmatic hernia.

ECHOCARDIOGRAPHY Should be performed preoperatively in any neonate with a heart murmur, particularly if associated with signs of cardiorespiratory impairment.

Preparation

The baby's condition should be optimised before surgery, with correction of fluid deficits, electrolyte, acid base and haematological abnormalities wherever possible. In conditions associated with intestinal obstruction, a nasogastric tube should be inserted and aspirated at frequent intervals to minimise the risk of pulmonary aspiration of gastric contents. Babies with lung disease may benefit from preoperative physiotherapy.

Prolonged preoperative starvation is dangerous in neonates as they are at increased risk of hypoglycaemia (see above). Appropriate exclusion periods for milk feeds and clear liquids are now well defined (Emerson *et al.* 1998) (Table 14.1).

Premedication

Atropine

This drug is still advocated by some centres as a routine premedicant in infants (Parnis and van-der-Valt 1994). In a dose of 20 μg/kg intravenously or intramuscularly it modifies the vagal response to anaesthesia (particularly with

***Table 14.1* Preoperative fasting guidelines for neonates and infants**

	Duration of fast (hours)	
Type of feed	*Neonates*	*Infants*
Clear fluids	2	2
Breast milk	4	4
Formula milk	4	6
Solids	N/A	6

Source: Emerson *et al.* 1998

halothane), laryngoscopy and intubation which can produce dangerous bradyarrhythmias. In addition, atropine reduces excessive airway secretions which can produce troublesome airway complications during the perioperative period.

Sedatives

These drugs are unnecessary in the neonatal age group in whom preoperative anxiety is not a factor. Prolonged effects of sedative drugs may be hazardous in this age group (see below).

Caffeine

Intravenous caffeine has been shown to reduce the incidence of postoperative apnoea in former preterm infants and has been advocated preoperatively by some authorities (Wellborn *et al.* 1989).

Transfer to the operating theatre

Neonates should be transported to the operating theatre in a warm incubator, with monitoring appropriate to the severity of illness. All neonatal intensive care units have a transport incubator equipped with an appropriate ventilator and the capability to monitor oxygen saturation, ECG and arterial blood pressure. It should also carry all drugs and equipment required for neonatal resuscitation (see p. 382).

The operating theatre environment

It is essential to maintain a baby in an environment that limits heat loss and thus reduces metabolic heat production. Morbidity and mortality have been shown to increase if a baby cools, with increased tendency to hypoxaemia, acidosis, coagulopathy and intraventricular haemorrhage (Bennett *et al.* 1977). The operating theatre should therefore be warmer than for adult surgery, with a temperature of 25 °C and humidity of 50%. (Additional measures that may be taken to reduce thermal stress are discussed in the section on anaesthetic equipment.)

Intraoperative management

Induction of anaesthesia

To minimise disturbance to the baby, anaesthesia should be induced in the operating theatre. Parental presence is not necessary for the infant, as such young babies are not obviously disturbed by separation; however, if parents express a

desire to be present at induction their wishes should be respected. The choice of an inhalation or intravenous induction depends on the clinical situation and whether an intravenous cannula is in situ.

Intravenous induction

This is the preferred method for induction of anaesthesia in most infants. Following intravenous cannulation, but before giving any drugs, the baby is given 100% oxygen via a face mask. This reduces the possibility of the baby becoming hypoxaemic when apnoeic, the risk of which greatly outweighs the risk of oxygen toxicity at this time (see below). A sleep dose of an induction agent is then given, typically thiopentone 2–3 mg/kg. Sleep doses are smaller in neonates due to increased endorphin and progesterone levels and a deficient blood-brain barrier.

Following induction of anaesthesia, muscle relaxants may be given to facilitate tracheal intubation. If rapid intubation is required (for example in babies at risk of aspiration of gastric contents) suxamethonium 2–3 mg/kg is used. In other situations, muscle relaxants with a slower onset of action such as atracurium, vecuronium and pancuronium are commonly used.

Inhalation induction

This is a useful alternative to intravenous induction, particularly in vigorous babies in whom intravenous cannulation may prove difficult. The new volatile anaesthetic agent sevoflurane has now superseded halothane as the most popular choice for inhalation induction in infants. Sleep is achieved smoothly and rapidly (within 60 seconds) and there is less tendency for a bradycardia to occur than with halothane (Lerman *et al.* 1994). Sevoflurane is added to oxygen or an equal mixture of oxygen and nitrous oxide and administered via an anaesthetic face mask in concentrations of up to 8% until the baby is deeply enough anaesthetised to tolerate intravenous cannulation. Muscle relaxants may then be administered to facilitate tracheal intubation.

Airway management

During general anaesthesia for neonates it is usual to intubate the trachea and use positive-pressure ventilation during all but the shortest surgical procedures. The airway may be difficult to maintain using a face mask and inhalational anaesthetic agents produce marked respiratory depression in this age group. Hypoxaemia due to reduced lung volumes and inefficient ventilation is therefore likely to occur unless controlled ventilation is used. In addition, gastric dilatation occurs following assisted ventilation using a face mask, making tracheal intubation essential.

Awake intubation is still advocated by some anaesthetists. It has however been demonstrated that it is associated with a higher incidence of significant desaturation than intubation following induction of anaesthesia (Kong *et al.* 1992). In addition awake intubation produces a significantly greater rise in anterior fontanelle pressure than following general anaesthesia and theoretically carries a risk of causing an intraventricular haemorrhage in high risk babies (Millar and Bissonnette 1994). It is, therefore, nowadays accepted that tracheal intubation should generally take place after induction of anaesthesia.

Maintenance of anaesthesia

Adequate anaesthesia during surgery has been demonstrated to reduce the infant's stress response and may improve outcome (Anand *et al.* 1988). High inspired concentrations of inhalational anaesthetics often cause severe cardiovascular depression in neonates. Consequently, a 'balanced' technique is usually favoured, combining a low concentration of inhalational anaesthetic in nitrous oxide and oxygen or an air-oxygen mixture to ensure that the infant is asleep, combined with muscle relaxants and adequate analgesia (see below). Ventilation is maintained intraoperatively, either manually or via a mechanical ventilator.

Oxygen and retinopathy of prematurity

The use of oxygen during anaesthesia in preterm neonates should be carefully controlled. Preterm babies are at risk of developing retinopathy of prematurity if blood oxygen levels are too high, even for short periods of time (Betts *et al.* 1977). Hyperoxia causes vasoconstriction and allows the release of angiogenic substances and oxygen-free radicals which may be toxic to many body systems. It is wise, therefore, to limit the inspired oxygen concentration to give a partial pressure of oxygen in arterial blood (PaO_2) of between 7 and 10 kPa. Pulse oximetry does not accurately detect hyperoxia but can be used as a guide (Brockway and Hay 1998); the lowest inspired oxygen concentration that maintains an oxygen saturation of 90–94% should be used.

Emergence from anaesthesia and extubation

If extubation is anticipated, the inhaled anaesthetic should be discontinued shortly before completion of surgery. When the surgical drapes are removed, the infant should be kept warm. The effects of muscle relaxants should be reversed completely and ventilation maintained with an air-oxygen mixture until the infant awakens and re-establishes spontaneous ventilation. The nose and mouth should be cleared of secretions and the nasogastric tube aspirated and removed if not required postoperatively. Extubation should only take place if the infant

is fully awake and breathing regularly and adequately. After extubation, oxygen should continue to be delivered via a face mask.

Analgesia

Until recently, postoperative paediatric analgesia was poorly managed. Neonates in particular were assumed to be incapable of perceiving pain and were seldom given adequate postoperative analgesia. However, advances in neonatal neurobiology have prompted a reconsideration of this approach. There is now no doubt that even the most premature infants respond to noxious stimuli, with well-developed physiological and behavioural responses (Anand and Hickey 1987). The modern view is that all infants must receive appropriate pain relief after surgery. Most of the methods of pain control used in adults can be modified for neonatal use; a balanced regimen using paracetamol, local anaesthesia and opioids is central to this approach.

Opioids

The term opioid is used to describe a group of drugs which act at specific receptors in the central nervous system resulting in profound analgesia. The most commonly used drugs in neonatal anaesthesia are morphine and fentanyl (see pp. 204 and 435).

Opioids must be used cautiously in neonates because immature metabolic pathways, particularly in the liver, may result in very unpredictable or prolonged responses to these drugs. The most major concern is the risk of opioid-induced respiratory depression. If the neonate is to receive respiratory support post-operatively then morphine or fentanyl can be used quite safely. Indeed, many paediatric anaesthetists would plan to electively ventilate a neonate after major surgery that necessitated ongoing opioid analgesia. Whether ventilated or breathing spontaneously, all neonates who have received opioids must be managed in a high dependency environment, with continuous pulse oximetry and apnoea monitoring being mandatory (Pounder and Steward 1992).

Morphine

Morphine has a longer duration of action and greater potency in neonates than in older children and adults (Lynn and Slattery 1987) (see pp. 204 and 435). This is mainly because of immature hepatic function resulting in delayed metabolism of opioid drugs. It should therefore be given less frequently and in a smaller dose. If postoperative ventilation is planned then up to 50 µg/kg may be given intraoperatively. Postoperatively, up to 20 µg/kg of morphine per hour may be used by continuous intravenous infusion but most neonates are well analgesed by 5–10 µg/kg per hour (Purcell-Jones *et al.* 1987). Morphine should

be used with extreme caution in neonates who are breathing spontaneously after surgery. Many authorities avoid morphine in this situation or limit the infusion to 5 µg/kg per hour.

Fentanyl

This synthetic opioid is useful in neonates with haemodynamic instability, particularly those with pulmonary vascular problems. It is a much more potent drug than morphine but has a shorter duration of action after a single bolus dose. After prolonged infusions, however, the drug will accumulate, resulting in excessive sedation, hypoventilation and risk of apnoeic episodes. For intraoperative use during major neonatal surgery up to 10 µg/kg is commonly used, necessitating postoperative respiratory support. Larger doses may be used but can potentially induce chest rigidity, particularly in preterm infants. Postoperatively an infusion of 4–8 µg/kg per hour may be used in ventilated neonates, but fentanyl should not be used in spontaneously breathing babies (see pp. 204 and 435).

Paracetamol

Paracetamol has been safely used in preterm and term babies and is a useful adjunct to opioid analgesia and nerve blockade after both major and minor surgery. Its analgesic effects are due to the inhibition of prostaglandin synthesis in the central nervous system. Single doses of 15 mg/kg orally achieve therapeutic serum concentrations and may be repeated 6-hourly (Hopkins *et al.* 1990), but rectally a loading dose of 40 mg/kg is required followed by 30 mg/kg 12-hourly if adequate serum levels are to be achieved (Anderson *et al.* 1997). Doses should be reduced in neonates with evidence of impaired hepatic function, particularly those with significant jaundice.

Non-steroidal anti-inflammatory drugs

These drugs are not recommended in the neonatal period as they can interfere with blood clotting mechanisms and cause a deterioration in renal function.

Local anaesthesia

Local anaesthetic techniques are invaluable in neonates, reducing or avoiding the need for opioids with their attendant risks in this high-risk group. All of the local anaesthetic techniques utilised in adults are feasible in neonates. The free (biologically active) fraction of local anaesthetic drugs is higher than in older infants due to a reduced concentration of plasma binding proteins; doses should

thus be adjusted accordingly. Bupivacaine is the most commonly used drug for which the dose should not exceed 1.5 mg/kg.

Wound infiltration

Wound infiltration is a simple and safe method of providing postoperative analgesia. It has been found to be effective following pyloromyotomy (McNicol *et al.* 1990) and is a useful alternative to ilio-inguinal nerve block for inguinal herniotomy (Reid *et al.* 1987). The opportunity to use this valuable technique in neonatal surgery should never be neglected.

Peripheral nerve blocks

A wide range of peripheral nerve blocks are potentially applicable to neonatal surgery but in practice only a few are commonly utilised. They are commonly used in conjunction with general anaesthesia and can provide very effective, long-lasting postoperative analgesia. Dorsal penile nerve block is very useful for penile surgery, especially circumcision, and has in fact been used as the sole anaesthetic technique for this procedure (Stang *et al.* 1988). Blockade of the ilio-inguinal nerve provides very effective analgesia after groin surgery, particularly inguinal herniotomy (Reid *et al.* 1987).

Central (neuro-axial) blocks

Local anaesthetic drugs introduced into the epidural or subarachnoid space can produce anaesthesia and analgesia of the lower limbs, abdomen and even thorax depending on the technique and the dose and volume of local anaesthetic used.

Such techniques are becoming increasing popular in neonates as they allow major surgery to be undertaken under very light or even without general anaesthesia (Williams *et al.* 1997). In addition, they provide excellent post-operative analgesia, eliminating or reducing the need for opioid analgesia and mechanical ventilation after surgery (Bosenberg *et al.* 1992).

Caudal epidural anaesthesia

Single-shot caudal anaesthesia is suitable for all surgical procedures below the umbilicus, and is mainly indicated in neonates for inguinal herniotomy, lower limb surgery and genito-urinary surgery (Figure 14.2). It is a very simple technique and major complications are rare.

Continuous caudal epidural anaesthesia involves the insertion of a catheter into the caudal space. The catheter may be threaded in a cephalad direction to a lumbar or thoracic level. An infusion of local anaesthetic

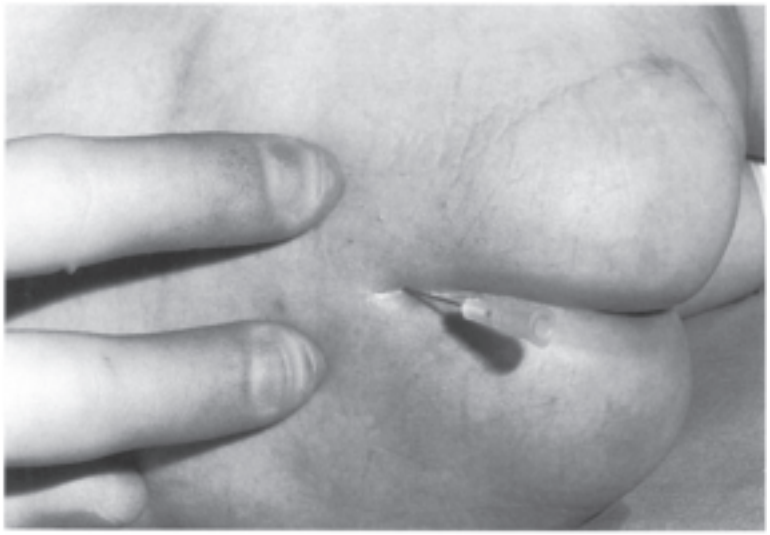

Figure 14.2 **Insertion of a caudal epidural block**

can then be used to provide analgesia following major abdominal and thoracic surgery (Bosenberg *et al.* 1988). The limitation of this technique is the proximity of the caudal catheter to the anus, which has raised concerns with regard to infection when this route is used for continuous postoperative analgesia.

Lumbar and thoracic epidural anaesthesia

Lumbar and thoracic epidural blockade is less popular than the caudal block because it is technically more difficult to perform, needs specially designed equipment and has the potential for more serious complications. These complications include inadvertent total spinal anaesthesia or intravenous injection of local anaesthetic with the potential for convulsions and profound cardiovascular collapse. Other rare complications include epidural haematoma or abscess. However, in a review of 240 lumbar or thoracic epidurals in neonates only three complications occurred, none of which was life-threatening (Bosenberg 1998). The haemodynamic instability commonly seen with epidural anaesthesia in adult practice (especially hypotension due to sympathetic nervous system blockade) is very unusual in the infant age group. This has been attributed to incomplete maturation of the sympathetic nervous system in the newborn resulting in very low levels of basal vascular tone. Epidural analgesia is now being increasingly used to provide excellent analgesia after abdominal and thoracic surgery. Though it is difficult to assess accurately the level of sensory block in a neonate, an adequate level can be assumed if the infant appears comfortable, while an excessively high block may be associated with an increased incidence of bradycardia or respiratory impairment.

Spinal anaesthesia

Spinal anaesthesia involves performing a lumbar puncture followed by injection of local anaesthetic into the subarachnoid space. It is technically easier than an epidural, has a rapid onset and provides complete motor and sensory blockade. The main disadvantages are a short and variable duration of action, which is usually less than one hour.

Spinal blockade is a useful technique for inguinal herniotomy in ex-preterm neonates where it is associated with a lower incidence of postoperative bradycardia and desaturation than following general anaesthesia (Krane *et al.* 1995). It may also be used in combination with epidural anaesthesia for major abdominal surgery (Williams *et al.* 1997).

Fluid therapy

Fluid administration during surgery must take into account the maintenance requirements of the infant, the loss of fluid from sequestration and evaporation, and blood loss. The goal of intraoperative fluid replacement is to maintain cardiovascular stability and organ perfusion, though caution should be taken to prevent fluid overload as this could potentially cause cardiac failure secondary to the reopening of a PDA.

Maintenance fluid

The fluid deficit generated during the fasting period should be replaced during surgery in addition to ongoing maintenance requirements. Opinions vary as to the best intravenous fluid to use. In the UK it is common practice to give glucose in combination with a low concentration of sodium chloride (e.g. 4% glucose + 0.18% sodium chloride). However, the hormonal stress response to surgery usually raises blood glucose concentrations in neonates (Sandstrom *et al.* 1995) and hyperglycaemia may occur with glucose-containing fluids. Their use should therefore be restricted to preterm infants or neonates less than 48 hours old. Compound sodium lactate (Hartmann's solution) can be used in all other cases, provided the blood glucose is carefully monitored, particularly during prolonged surgery.

Replacement of fluid lost by evaporation/sequestration

During surgery, water is lost by evaporation from the operative site and protein-rich fluid is sequestered in the surrounding tissues as a result of surgical trauma, depleting the intravascular fluid volume. In infants, these fluid losses during abdominal surgery are estimated to be between 6 and 10 ml/kg per hour, 4–7 ml/kg per hour in thoracic surgery and only 1–2 ml/kg per hour in superficial

procedures (including neurosurgery) (Bennett 1975). This should be replaced by Hartmann's solution or 0.9% sodium chloride.

Replacement of blood loss

The preoperative haemoglobin concentration (Hb) and blood loss perioperatively will determine whether blood loss is replaced by blood or non-sanguinous fluid. The estimated blood volume (EBV) should be calculated (90 ml/kg for a preterm baby, 85 ml/kg for a term baby). The maximal allowable blood loss (ABL) can then be calculated from the formula:

$$\text{ABL} = \frac{\text{EBV} \times \text{initial Hb} - \text{lowest acceptable Hb}}{\text{initial Hb}}$$

The lowest acceptable Hb will depend on clinical circumstances. Blood loss can be determined by accurate weighing of surgical swabs and noting suction losses and replaced with a colloid solution until the maximal ABL is reached. Thereafter blood should be used. The most commonly used colloid in neonates is 4.5% human albumin solution, although recent controversy regarding its safety has led to the increased use of other synthetic colloids, including Gelofusine and Haemaccel. Replacement of clotting factors by the use of fresh frozen plasma (FFP) should commence earlier rather than later due to the relatively deficient clotting systems in neonates. Platelet infusions may be required in cases of massive blood transfusion.

Assessment of the adequacy of volume replacement can be gauged clinically. Changes in pulse and blood pressure are both unreliable guides to hypovolaemia in neonates. Assessment of the peripheral circulation gives far more information: capillary refill time should be rapid (less than 2 seconds) and extremities should be pink and warm. Prolonged refill time and increased core-peripheral temperature gradient are important clinical signs, suggesting volume depletion (Lambert *et al.* 1998). Measurement of urine output and central venous pressure (CVP) may also provide useful information, particularly during prolonged surgery or where large blood and fluid losses are anticipated.

Anaesthetic equipment

Neonatal anaesthesia requires specialised equipment. All equipment should be thoroughly checked before commencing any anaesthetic.

Airway equipment

Many of the items of equipment required for airway maintenance are available in a variety of sizes to suit all infants. It is not always possible to predict

which will be appropriate, so a full range should be immediately available for every case.

Face masks

Face masks should have a low dead space to prevent rebreathing of expired gases. Several types are available: the Rendell-Baker mask is designed to fit anatomically but it can be difficult to obtain a good seal on the face in order to inflate the lungs without leakage of gases (Palme *et al.* 1985). Nowadays a circular cushioned rim type face mask is more commonly used which many anaesthetists (and neonatal intensive care staff) find easier to manipulate in small infants (Figure 14.3).

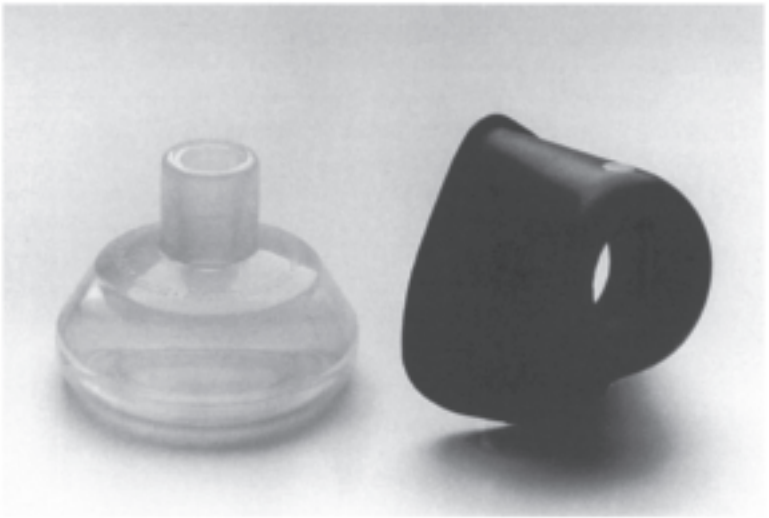

Figure 14.3 **Anaesthetic facemasks. The cushioned rim silicone mask *(left)* has now superseded the black rubber Rendell-Baker version *(right)* in neonatal practice**

Oral airways

Difficulties maintaining the airway in an anaesthetised neonate may be lessened if an oral airway is used, but will be increased if one of an incorrect size is chosen. Too small an airway may fail to overcome airway obstruction due to the tongue falling backwards whereas too large an airway may produce airway obstruction itself or induce laryngospasm or vomiting in a semi-conscious baby. The size is estimated by placing the airway against the cheek of the baby; with the flange at the level of the baby's lips the tip of the airway should reach the angle of the mandible.

Laryngoscopes

The infant larynx lies higher and more anteriorly than in older children or adults. In addition, the epiglottis is large and leaf-shaped and can obscure the laryngeal inlet. The best view of the larynx is obtained by using a laryngoscope with a short, straight blade, for example the Seward, Robertshaw and Wisconsin varieties.

Tracheal tubes

These should be parallel-sided, uncuffed, radio-opaque and marked at 1 cm intervals from the tip. Each tube is sized according to the internal diameter. The approximate sizes and lengths of tracheal tubes according to the weight of the infant are shown in Table 14.2. Plain tubes are suitable for most procedures and can be used via the oral or nasal route (see p. 289), though 'south facing' preformed RAE™ oral tubes are popular for orofacial and neurosurgery and 'north facing' preformed RAE™ oral tubes are becoming increasingly popular for other procedures because they are easy to secure in position and are less likely to kink (Figure 14.4).

Table 14.2 Size and length of tracheal tubes for neonates

Body weight (kg)	*Internal diameter (mm)*	*Length at lip (cm)*	*Length at nostril (cm)*
< 0.7	2.0	5.0	6.0
0.7–1	2.5	5.5	7.0
1.1–2	3.0	6.0	7.5
2.1–3	3.0	7.0	9.0
3.1–3.5	3.0	8.5	10.5
> 3.5	3.5	9.0	11.0

Source: Peutrell and Weir 1996: 180

Other equipment

In addition to the equipment mentioned above, small Magill forceps should be available for insertion of nasal tracheal tubes or throat packs and intubation stylets narrow enough to fit through the smallest tracheal tube should be available to aid a difficult intubation.

Breathing systems

The breathing system provides the means of delivering the anaesthetic gases and vapours to the patient. In a neonate it should be lightweight, easy to assemble,

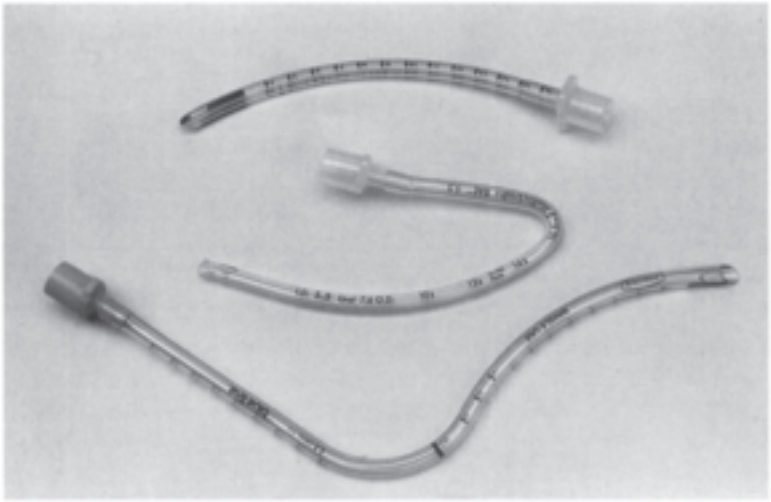

Figure 14.4 **Tracheal tubes. Uncuffed tubes are appropriate in neonates. Besides the familiar straight version (top), preformed tubes *(middle and bottom)* are useful during surgery, for example, head and neck procedures**

present minimal resistance to breathing and have as small a dead space as possible. The most popular system in the UK is the Jackson-Rees modification of the Ayre's T-piece (Figure 14.5). This can be used for spontaneous or positive-pressure ventilation. It can also be used to apply positive end-expiratory pressure (PEEP) or continuous positive airway pressure (CPAP), manouevres which are essential in neonates to prevent collapse of the small airways and improve oxygenation.

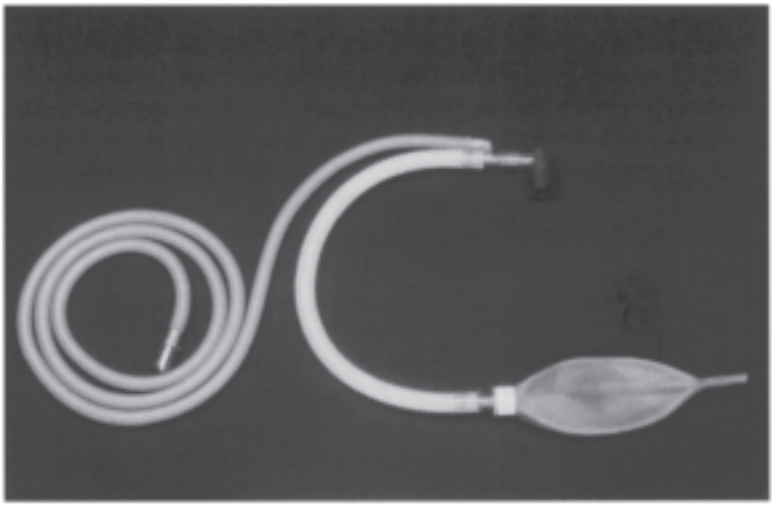

Figure 14.5 **T-piece breathing system**

Ventilators

Neonates are ventilated intraoperatively either by hand or by attachment to a mechanical ventilator. Manual ventilation is popular in neonates as it provides the anaesthetist with a 'feel' of the compliance of the lungs. There is a danger, however, of applying too much pressure and causing pulmonary barotrauma or even a pneumothorax. T-piece occluding ventilators which are pressure-limited (including the majority of those used on a neonatal intensive care unit) can be used in the very sick neonate with respiratory disease. More popular in the operating theatre setting are the 'bag squeezer' type ventilator which essentially replaces the anaesthetist's hand squeezing the bag. These produce a constant flow and tidal volume unless higher pressures are required, when most become pressure-limited. For reasons already mentioned, an additional feature required of all neonatal ventilators is the facility to apply PEEP.

Equipment for maintenance of body temperature

The importance of maintaining an infant's body temperature cannot be over-emphasised (see Chapter 4). In addition to a warm operating theatre, the following measures can be utilised.

Overhead radiant heater

This should be used during both induction of anaesthesia and application of the skin preparation. It can also be used during emergence from anaesthesia.

Warming mattresses

These are essential to reduce conductive heat loss through a cold operating table. The surface temperature should not exceed 39 °C to prevent skin burns. An alternative is the hot air mattress, which surrounds the infant in a microclimate of warm air.

Humidifiers

Humidification of inspired anaesthetic gases reduces evaporative heat loss from the respiratory tract and may be either active or passive. Active humidification requires a water bath with careful temperature control. This method is commonly used in the neonatal intensive care unit. Passive humidifiers are devices with a high surface area that allow an exchange of heat and moisture from exhalation to inhalation. They are included in the anaesthetic breathing system in close proximity to the tracheal tube. They have been shown to be as efficient as a heated humidifier when used in ventilated neonates over a 6-hour period (Schiffmann *et al.* 1997).

Other methods

All fluids administered should be warmed to 37 °C. In addition, the infant can be wrapped in cotton wool, aluminium foil or plastic sheets to prevent radiant and convective heat loss. Particular attention should be paid to the head, which is relatively larger in neonates and exposes a big surface area for heat loss.

Equipment for intravenous fluid administration

Intravenous cannulae

For reliable venous access an over-the-needle plastic cannula should be used. These are available in sizes as small as 25G. For all but the most minor surgical procedures most neonatal anaesthetists would ensure that there are two functioning intravenous cannulae in situ.

Fluid administration sets

The small volume of infusate required by neonates make syringe pumps and other sorts of infusion device preferable for the administration of maintenance fluids. They should have a volume limiter and a pressure alarm. Other devices include giving sets with a micro-drop outlet and a burette, which can be filled with a predetermined volume of fluid (for example one hour's requirements) to prevent inadvertent overhydration.

Administration of fluid boluses including colloid and blood product is most easily achieved manually by a syringe attached via a three-way tap.

Anaesthetic monitoring

Although there is no substitute for the continuous presence of a well-trained anaesthetist, there is evidence to suggest that intraoperative adverse events can be avoided by physiological monitoring of the neonate (Cote *et al.* 1988). Minimal monitoring in any baby undergoing surgery should include the following:

Cardiovascular monitoring

Precordial and oesophageal stethoscope

A precordial stethoscope is a device secured over the left sternal border. An oesophageal stethoscope is a soft catheter with holes in its distal end positioned in the mid-oesophagus. Both allow continuous monitoring of heart and breath sounds though they do not give accurate information about the adequacy of cardiac output or pulmonary ventilation.

Electrocardiography (ECG)

Cardiac output in neonates is rate-dependent. Bradycardia leads to a fall in cardiac output and hypotension, whereas tachycardia may be a sign of hypovolaemia, pain or inadequate anaesthesia. Although a very useful monitor, the ECG does not give any indication of the adequacy of vital organ perfusion.

Blood pressure

The oscillometric method and Doppler method of non-invasive blood pressure measurement are most commonly used in neonatal practice. The oscillometric method measures mean blood pressure accurately and derives systolic and diastolic pressure using a computer algorithm. It is a reliable method in neonates provided the correct cuff size is used (cuff width to arm circumference ratio should be approximately 0.5). The Doppler method uses an arm cuff and an ultrasonic transducer. This is placed over the radial or brachial artery and detects changes in vessel wall movement to record systolic and diastolic blood pressure.

Invasive arterial pressure should be measured via a catheter inserted into the umbilical artery or a peripheral artery when rapid fluid shifts, particularly large blood losses or sudden haemodynamic changes, are anticipated (for example during cardiac surgery). Indwelling arterial access also allows blood gases and other parameters such as blood glucose and haematocrit to be easily monitored during major and prolonged surgery.

Respiratory monitoring

Pulse oximetry

Pulse oximetry has proved a major advance in the monitoring of neonates. It provides continuous, non-invasive beat-to-beat monitoring of oxygen saturation and heart rate. Positioned in neonates over the lateral border of the foot or medial border of the hand, it also provides information about peripheral perfusion and therefore cardiac output and volume status. Limitations of pulse oximetry are interference from diathermy and motion. It does not accurately detect hyperoxia and is inaccurate at oxygen saturations below 75% (Cote *et al.* 1988).

Capnography

A capnograph measures the change in carbon dioxide (CO_2) with each breath by sampling expired gas. In healthy neonates the end-tidal CO_2 concentration closely approximates arterial PCO_2. The value of end-tidal CO_2 is influenced not only by arterial PCO_2 but also by cardiac output and ventilation. It is,

therefore, a useful monitor of cardiovascular status in addition to the adequacy of ventilation, including inadvertent disconnection from mechanical ventilation.

Temperature monitoring

Temperature should be monitored in all neonates undergoing surgery. Core temperature can be estimated at several sites, including oesophagus, rectum, tympanic membrane or nasopharynx. Oesophageal probes are the most convenient and can be incorporated into an oesophageal stethoscope. Skin temperature should also be measured because the gradient between core and peripheral temperature gives a useful indication of the adequacy of cardiac output (Lambert *et al.* 1998).

Postoperative care

All small infants require close observation and monitoring in the postoperative period. This may be in a high dependency area or in an intensive care unit, depending on the prematurity of the infant, the surgical condition and other coexisting medical problems. As with all postoperative patients, adequate analgesia must be provided; this may be achieved by several methods which have already been described. Fluid administration should be tailored to the needs of the infant and measures taken to maintain body and peripheral temperature (Beuton and Klein 1964).

There are certain postoperative problems that are encountered far more frequently in infants. These include apnoeic episodes, extubation stridor and respiratory insufficiency requiring ventilatory support.

Postoperative apnoeas

The neonate, particularly the preterm infant, breathes irregularly. Periodic respiration in which breathing and apnoea alternate is common; however, cessation of breathing for longer than 15 seconds (or less if associated with bradycardia or desaturation) is significant and abnormal. Neonates are prone to develop apnoeas following general anaesthesia, the risk rising with increasing prematurity (Stewart 1982) and with the use of intraoperative opioids. Regional anaesthesia without sedation reduces the risk significantly (Wellborn 1992). High dose caffeine (10 mg/kg IV) may also be protective (Wellborn *et al.* 1989). Those most at risk have a postconceptual age (PCA) of less than 45 weeks at the time of surgery (Wellborn *et al.* 1990), while apnoeas are rare beyond 48 weeks PCA unless neurological disease is present.

Infants less than 48 weeks PCA should be nursed in a high dependency area for at least 12 hours postoperatively with apnoea monitoring and pulse oximetry.

Most apnoeas can be treated successfully with stimulation, oxygen and occasionally intravenous caffeine or theophylline. Rarely, CPAP or mechanical ventilation are necessary if apnoeic episodes are recurrent in the postoperative period.

Post-extubation stridor

Stridor may occur either immediately or within a few hour of tracheal extubation. It is usually due to subglottic oedema caused by trauma exerted on the tracheal mucosa at the level of the cricoid cartilage by an oversized tracheal tube. The risk is greatest if there is no leak around the tracheal tube following insertion, if the tube moves within the trachea during surgery and if multiple intubation attempts were necessary.

Possible therapies include inhalation of nebulised adrenaline, intravenous dexamethasone or the provision of CPAP via a nasal prong. If reintubation is required a smaller tracheal tube should be used and an audible gas leak around the tube should be present before it is subsequently removed.

Respiratory insufficiency

A key issue in early postoperative management is the likely need for postoperative ventilatory support. It is important to understand the major factors affecting ventilatory function in order to minimise the risk of post-operative ventilatory insufficiency. These factors include the following:

Gestation at birth and postconceptional age

In addition to an increased risk of postoperative apnoeas, problems of prematurity such as respiratory distress syndrome and persisting patent ductus arteriosus affect the efficiency of pulmonary gas exchange and pulmonary mechanics and make the need for postoperative ventilatory support more likely (Stewart 1982).

Ventilatory drive

Ventilatory drive is immature in neonates and is more likely to fail postoperatively in the presence of hypoxaemia, hypothermia or hyperthermia, hypoglycaemia, sepsis and anaemia. All these factors must be avoided where possible. In addition, if the infant has been given opioid analgesics intraoperatively or is likely to require them postoperatively, hypoventilation is more likely to occur.

Assessing the need for postoperative ventilation

The requirement for postoperative ventilatory support will depend on a variety of perioperative factors.

Preoperative factors

Pre-existing pulmonary or cardiac disease (e.g. RDS, congenital heart disease) or upper airway problems such as subglottic stenosis or laryngomalacia make postoperative respiratory insufficiency more likely. The surgical condition should also be considered.

Postperative factors

Respiratory insufficiency is likely to occur following a prolonged anaesthetic due to the residual effects of anaesthetic and analgesic drugs (particularly opioids) on ventilatory drive. Abdominal distension following abdominal surgery may compromise diaphragmatic function and therefore effective spontaneous ventilation. Other factors, including hypothermia, acidosis and anaemia, may also be indications for a period of postoperative ventilation.

Signs of respiratory distress

The assessment of postoperative respiratory distress is largely clinical. A respiratory rate of greater than 60 breaths per minute should alert the anaesthetist to the possibility of respiratory insufficiency, particularly if associated with signs of increased work of breathing demonstrated by grunting respiration, nasal flaring and the presence of subcostal, intercostal and substernal recession. Restlessness, irritability, apnoeic episodes or failure to regain consciousness may also reflect hypoxaemia or hypercarbia.

Blood gas and acid base analysis should follow any suspicion of respiratory insufficiency. Other investigations, including chest radiography, may be indicated but if there is any suggestion that ventilatory support is required on clinical grounds this should be instituted without delay. This may initially be by bag and mask ventilation and subsequently nasal CPAP or reintubation and mechanical ventilation.

Conclusion

Many full-term neonates undergo anaesthesia and surgery uneventfully. The greatest problems are seen in sick and premature infants, particularly if cardiorespiratory insufficiency is present preoperatively. Care of these infants requires a multidisciplinary approach by skilled personnel. The importance of

communication between the anaesthetist and the neonatal nursing and medical team cannot be overemphasised and is essential if the best possible outcome is to be achieved.

Case study: anaesthetic considerations of a 10-week infant with bilateral inguinal herniae

George is a 10-week-old baby who was born at 28 weeks' gestation. He required ventilation for 3 weeks, nasal CPAP for a further week and is still oxygen-dependent. He now requires surgery for bilateral inguinal herniae.

Q.1. What are the main problems for the anaesthetist in managing this baby?

Q.2. What (if any) preoperative investigations does he require?

Q.3. For how long should George be starved prior to surgery?

Q.4. Does he require premedication?

He has both herniae repaired under general anaesthesia. At the end of the procedure he is extubated and returned to NICU.

Q.5. What potential problems should the nursing staff be looking out for?

Q.6. Is any special monitoring required in the postoperative period?

Q.7. What methods may be used to provide adequate pain relief?

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Chapter 15

Surgical Aspects of Neonatal Intensive Care Nursing

Chapter 15

Contents

Introduction

Neonatal surgery has evolved rapidly over the past 40 years, its success contributing to the fall in neonatal mortality. Sophisticated antenatal screening and fetal abnormality apperception has led to a changing pattern of operable malformations, and the delivery of a baby with an undiagnosed major structural abnormality is now rare.

Although fetal intervention is experimental and not of proven benefit, antenatal diagnosis of surgically correctable malformations will allow for in utero transfer and planned delivery in a specialist centre. Regionalisation secures exemplary utilisation of resources and the expertise of the multidisciplinary team (MDT). The surgeon apart, neonatologists, specialist anaesthetists, laboratory technicians, physiotherapists and pharmacists are all crucial to the perioperative care and recovery of these small, vulnerable, but surprisingly resilient patients.

Parents should meet with the surgeon as soon as the diagnosis is made. They need patient, expansive, reiterated information about the anomaly, its short-term management and the possible long-term sequelae. A team approach is recommended, with the neonatologist, geneticist, nursing staff and social services in addition to the surgeon providing their specific expertise to support the family through what might be a long-lasting adjustment to their child's condition. Meeting NICU staff, other parents and early receipt of specialist information booklets may help to avoid extended periods of uncertainty, especially when the diagnosis is made antenatally.

Multiple defects can be associated with chromosomal abnormalities: therefore it is important to differentiate, for example, between exomphalos and gastroschisis; and between duodenal and jejunal obstruction, as there may be associated cardiac anomalies and Down's syndrome. If a chromosome aberration is found in the fetus or neonate, the parents should be offered genetic counselling.

The timing and mode of delivery should be a joint decision between the obstetrician and the neonatal team. Although certain malformations require elective cesarean section, most problems are best managed by natural labour and delivery at term. Many babies will require resuscitation, and appropriately skilled personnel should be present in the delivery suite (see Chapter 3).

General principles of management

On the neonate's arrival in the NICU, the goal is expedient stabilisation. Skilful assessment and management preoperatively should determine how well the infant will cope with surgery. And, whilst the surgery itself may be relatively short, its success will depend on the calibre of the postoperative care.

Thermoregulation

Prevention of hypothermia is imperative. This is well recognised and compensated for in the NICU, but transportation vehicles and operating theatres may not be so well adapted, so the temperature should be adjusted accordingly. Prior to the surgery, the infant should be placed on a heated mattress and wrapped in warm gamgee with only the operation area exposed. Skin preparations should be aqueous rather than alcoholic, to reduce heat loss by evaporation. Core temperature should be maintained at 37 °C, minimising oxygen consumption and acidosis. Peripheral temperature should be maintained at above 36 °C, a fall below 34 °C indicating under-perfusion which may be due to hypovolaemia or infection. Infants undergoing laparotomy are at increased risk from heat loss from the exposed bowel.

Respiratory function

Assessment of respiratory function is a prerequisite for all 'surgical neonates', and urgent intervention may be required. Ventilatory insufficiency may present at delivery due to anatomical abnormalities of the respiratory tract, and increased intra-abdominal pressure can also lead to respiratory compromise; correct positioning to relieve the pressure is vital. Additionally, these neonates are not immune to medical problems such as respiratory distress syndrome (RDS) and aspiration pneumonia, and the degree of respiratory support they require will depend on clinical and radiological findings supported by blood gas estimation (see p. 108).

Gastric decompression

Gastric decompression is necessary to avoid aspiration and reduce splinting of the diaphragm; the need for augmented ventilatory support may indicate inadequate decompression. Additionally, a large volume of secretions may accumulate in a neonate's stomach, with a risk of aspiration pneumonia. Decompression and aspiration are achieved with a correctly positioned nasogastric tube large enough to prevent blockage (8fg or greater) (see p. 286). The tip of the tube should be in the stomach confirmed by the 'water recovery test' (Hassan and Hobsley 1970). The tube should be left on continuous drainage with gentle intermittent suction.

Fluid and electrolyte balance

Surgery can aggravate physiologic imbalances in the newborn, so continuous assessment of perfusion, parenteral fluid and electrolyte requirements, and metabolic responses to surgical trauma, is necessary for the restoration and

maintenance of homeostasis. Some infants will need fluid resuscitation preoperatively—hypovolaemia can result from continuous loss of saliva from the oesophageal pouch in oesophageal atresia, and large quantities of fluid from the exposed areas in gastroschisis and exomphalos. Estimation of insensible losses is crucial, and loss of fluid via the nasogastric tube may require replacement and supplementation with potassium.

Alterations in acid base homeostasis can be caused by several factors. Respiratory acidosis occurs with inadequate ventilation, in pulmonary hypoplasia secondary to congenital diaphragmatic hernia, and in any condition that causes decreased oxygenation or perfusion. Metabolic acidosis can occur when bicarbonate losses are increased, with poor tissue perfusion, tissue necrosis, infection, hypovolaemia and as a result of intestinal fistulas and necrotising enterocolitis (NEC). The most common cause in 'surgical neonates' is hypovolaemia and requires colloid. Correction with bicarbonate should be cautious, as it may cause hypocalcaemia (Lynch 1990).

Glycogen is a skeletal muscle and hepatic storage carbohydrate and is produced when blood glucose falls outside the homeostatic range (Clancy and McVicar 1995). Neonates have poor glycogen stores due to decreased availability of substrate in utero, and therefore need a constant glucose intake. This is especially important during surgery when blood transfusion may be required. It is important that dextrose should be administered concurrently and the blood glucose monitored frequently, maintaining a level of 2.6–5 mmol/l.

A neonate's blood volume is about 80 ml per kilogram body weight. A 2 kg infant therefore has a circulating volume equivalent to the average loss during minor adult surgery. It is vital that operations are performed by specialist neonatal surgeons, as their techniques have evolved to limit blood loss. Coagulation status should be assessed preoperatively and treated accordingly. The newborn is deficient in vitamin K, so this should be administered. Neonates with severe sepsis may develop disseminated intravascular coagulation (DIC) (see p. 180) with associated thrombocytopenia (Puri and Sweed 1996). Clotting factors should be replaced by transfusion with appropriate blood products.

During prolonged preoperative stabilisation, and following abdominal surgery causing paralytic ileus, total parenteral nutrition allows delivery of adequate nutritional substrates directly into the circulation. It promotes anabolism and provides for normal growth and development until gut function is restored. The stimulus of surgery and intermittent positive-pressure ventilation (IPPV) lead to increased aldosterone and antidiuretic hormone (ADH) secretion resulting in water and sodium retention. It is therefore pertinent to restrict fluid and sodium postoperatively (see p. 213).

A central line is highly recommended for prolonged venous access (see p. 301), an arterial line is necessary for monitoring haemodynamic, biochemical and respiratory status, and at least two peripheral cannulae are required for the administration of medications (see p. 298).

Pharmacological support

There is a risk of sepsis whenever surgery is performed, especially in intrauterine growth-retarded (IUGR) and preterm babies with immature immune systems. Untreated infection promotes deterioration of the respiratory and cardiovascular systems, and prophylactic antibiotic therapy can reduce this risk. Inotropes are often necessary to improve cardiac function, thus ameliorating organ perfusion. Pain relief is an important consideration, even preoperatively, as cellular damage, for example in NEC, releases pain-producing substances, augmenting the perception of pain (Clancy and McVicar 1995). Intubation and ventilation are usually necessary for the facilitation of adequate pain relief, as neonates have an enhanced sensitivity to the respiratory depressant effects of opiates. Effective analgesia via an epidural catheter can be provided without this risk providing that toxic doses of regional bupivacaine are avoided (see p. 326).

Transportation

The critically ill neonate can make several potentially hazardous journeys—from the delivery room to the NICU, possibly to a regional surgical centre, as well as to and from the operating theatre. Safe transportation demands collaboration between a doctor and nurse experienced in neonatal intensive care, and ideally, a specialist anaesthetist on the return journey from theatre. In the past, transport was often hastily arranged and was associated with significant morbidity. Disasters such as vomiting with aspiration, hypothermia and airway obstruction can be magnified during transportation, even on short journeys within the hospital, but can be averted with appropriate stabilisation (Puri 1996a) (see Chapter 16).

Postoperative considerations

Management in the postoperative period mirrors the preoperative care in the gaining and maintaining of physiological stability, but in addition the factors listed in Figure 15.1 need careful consideration:

Most commonly encountered congenital disorders

Oesophageal atresia (OA) and tracheo-oesophageal fistula (TOF)

The oesophagus and trachea have a common embryological origin. Initially they are fused, but a septal separation occurs by week 6 and failure of complete separation will result in **fistula** formation. The oesophagus should re-canalise and become patent by week 10 and failure of this results in atresia (Moore and Persaud 1993).

- Do not leave the operating theatre until the baby is stable. Ideally a doctor and anaesthetist should be in attendance on the return journey to the NICU
- Before returning the baby to the incubator and ventilator, check the settings, then re-connect fluids and monitor leads to static equipment
- Adjust maintenance and arterial line fluids. Commence NG replacement losses if necessary. Titrate sedation and epidural infusions as appropriate. Observe entry sites
- Attach nasogastric tube to drainage bag
- As soon as possible record:

core temperature	then 4-hourly until stable
blood sugar	then 1–2-hourly until stable
ventilator settings	then 1-hourly until stable
blood pressure heart/respiratory rate	continuous read-out, but record hourly

- Attach peripheral temperature probe and maintain temperature above 34 °C
- Organise a chest X-ray if the baby was intubated in theatre, there is a chest drain in situ and following diaphragmatic hernia repair
- Check blood gas and repeat as necessary
- Record urinary output—attach urine bag or weigh nappies—expect 1 ml/kg per hour after the first 24 hours
- Check biochemical and haematological status
- Maintain adequate pain relief
- Carefully observe wounds, stomas, etc., recording any losses
- Tailor endotracheal suction to each individual's needs—pre-oxygenating if necessary
- Encourage parental involvement in care as appropriate, remembering that minimal handling is essential to recovery. Apart from babies who are electively paralysed, who must have eye care, passive movements and a change of position every 4 hours—6- to 8-hourly care is adequate (see p. 337)

***Figure 15.1* General postoperative considerations following major surgery**

Thus TOF and atresia can occur as separate entities but more frequently occur concurrently.

The commonest form, constituting 85 per cent, is a blind proximal oesophageal pouch with a 1–2 cm gap, and a distal TOF (Ross 1992). Variations relate to the width of the gap, which may extend to a 'long gap' greater than 2 cm, or just consist of a membrane. There are four less common types of the condition: pure atresia with no fistula; a proximal and a distal fistula together; a fistula with no atresia (H-TOF); and a proximal fistula. Associated anatomical features include an hypertrophied proximal oesophageal pouch—a result of fetal ingestion

of amniotic fluid. If the pouch is not hypertrophied, a **fistula** should be suspected.

There is an association with other abnormalities, including VATER, VACTERL and CHARGE syndromes (see Glossary; Guiney 1996), and it is important to recognise the association with Edward's syndrome—in which the poor prognosis may not justify surgical repair (Spitz and Hancock 1994).

Maternal polyhydramnios is usually present because amniotic fluid is unable to pass into the gastrointestinal tract for absorption and subsequent transfer to the placenta. The anomaly may be diagnosed by ultrasound—the inability to demonstrate a fetal stomach in the presence of normal or increased amniotic fluid is highly suggestive of OA, allowing appropriate measures to be taken at birth.

However, there is often no antenatal diagnosis and symptoms present in the early postnatal hours. The infant will cough and choke on excessive saliva in the mouth and upper respiratory tract. If enteral feeds are offered, the oesophageal pouch will fill, followed by regurgitation. Gastric contents can also reflux from the stomach through the fistula into the respiratory tract, presenting the danger of aspiration and pneumonitis. The abdomen will rapidly distend as the intestines fill with air.

The accurate evaluation of a fistula requires special investigation, but the diagnosis of a proximal atresia is confirmed by passing a radio-opaque tube size 8–10fg through a nostril until resistance is felt and by X-ray of the neck, chest and abdomen. The tube will be seen coiled in the oesophageal pouch. Although infants with an H-type fistula may have early signs of respiratory distress, aggravated by feeding, the diagnosis (usually by endoscopy) is not often confirmed in the neonatal period.

At birth, these babies should be positioned with the head elevated at 45 degrees, or prone, to prevent aspiration pneumonia. A double-lumen Replogle tube (Replogle 1963) placed in the upper pouch and connected to low-pressure continuous suction will prevent this, and should be introduced as soon as possible. Instillation of saline (1–2 ml every 15 minutes should deliquesce the tenacious secretions, maintaining tube patency).

Endotracheal intubation is not usually necessary; indeed with a distal fistula it is possible to rupture the stomach with mechanical ventilation.

The timing of operative intervention, which involves the disconnection of any fistula and the establishment of oesophageal continuity, depends on the width of the oesophageal gap. Repair of the atresia is not an emergency, the aim being to optimise clinical status. Pulmonary function may be improved with physiotherapy and antibiotics, particularly if aspiration has occurred. Associated anomalies should be suspected and routine ultrasound scans of the abdomen, and cardiac echocardiography, should be performed preoperatively. It is essential to maintain the sucking reflex whilst awaiting surgery, especially in delayed primary anastomosis in long-gap atresia. A Teat-Replogle device (Boyd and Tsang 1996) provides oral comfort whilst encouraging sucking, is well tolerated for long periods, and should be introduced to the infant once the diagnosis is established.

Fistulae are usually repaired within hours of birth, together, if possible, with an end-to-end oesophageal anastomosis via a right posterolateral extrapleural thoracotomy. The distal fistula and oesophagus are usually hypoplastic due to the absence of functional challenge (Guiney 1996), and can make anastomosis difficult.

A 'long-gap' requires staged surgery. A cervical oesophagostomy to permit 'sham-feeding', repair of fistulae and a gastrostomy performed concurrently allows the infant to be managed at home, until there is hopefully sufficient distal oesophageal growth to perform a delayed anastomosis (Guiney 1996). Unfortunately, as the proximal end is unable to grow, anastomosis may not ultimately be possible.

More recently, an alternative has been to allow the oesophagus to grow in situ. Gastro-oesophageal reflux to distend the lower pouch is encouraged by spigotting the gastrostomy between feeds (Puri *et al.* 1992). Small oral feeds can be given cautiously as they will be retrieved by the continuous suction. This course of treatment involves hospitalisation for up to 6 months, or until there is sufficient growth—ascertained by contrast studies—for anastomosis to take place. The decision to proceed with this method needs to be taken after careful discussion with the parents. Even those who initially agree may then change their minds and request an oesophagostomy to enable them to take their child home.

Although the best oesophagus is the patient's own, and every effort should be made towards oesophageal preservation, sometimes the gap never shortens to an operable length and it is necessary to perform a colonic, jejunal or gastric interposition. However, this is a huge undertaking with potential serious complications, and should only be considered if the situation is irremediable.

The need for postoperative respiratory support not only depends on the gestation of the baby, but also upon the respiratory status and, more importantly, upon the tightness of the repair. If the anastomosis is under tension, the baby will need to be given muscle relaxants and nursed with the neck flexed for 5 days whilst healing takes place. A transanastomostic tube (TAT) will be in situ to keep the oesophagus from stenosing with scar tissue whilst healing occurs. It is essential this is secured as its replacement could seriously damage the suture line. A chest drain is usual, and antibiotics are continued until it is removed.

Nutrition is important to the recovering infant whether fed parenterally if ventilated, or via the gastrostomy. If a primary anastomosis has been performed, feeding may take place via the TAT; and following a barium swallow to check for the absence of an anastomotic leak, by breast or bottle. However, some centres consider routine contrast studies inappropriate as a minor leak is insignificant and will heal spontaneously, and a major leak will present with a tension pneumothorax, and can be an important cause of postoperative morbidity and mortality.

Gastro-oesophageal reflux is one of the most common long-term problems and is thought to be due to disordered oesophageal motility, or as a result of gastrostomy feeding (Spitz and Hancock 1994). Treatment is usually with

thickened feeds, but fundoplication may be required. Feeding difficulties and vomiting, which may last for years, especially following tight repairs, are relatively common and are related to oesophageal strictures. Frequent oesophageal dilatations may be necessary to encourage normal eating. Parents should be given detailed information and advice about this; it is useful to put them in touch with another TOF family, and the local support group.

Tracheomalacia can be serious enough to progress to respiratory distress with obstruction, cyanosis, bradycardia, respiratory and even cardiac arrest, and also causes the characteristic 'TOF cough' or 'seal bark' (Guiney 1996).

Fistular recividity may occur and should be suspected in the child who develops frequent respiratory infections, with gagging, cyanosis and even apnoea.

Earlier diagnosis, improved surgical techniques, and sophisticated intensive care have positively influenced survival. One of the most common major congenital abnormalities has progressed from being incompatible with life to one with a mortality limited to those cases associated with extreme prematurity, or other life-threatening anomalies (Spitz and Hancock 1994).

Case study: postoperative management of an infant with oesophageal atresia

Caroline is delivered at 36 weeks' gestation by ventouse extraction. She weighs 2.135kg. An ultrasound scan at 29 weeks had shown 'polyhydramnios, no stomach and paucity of bowel shadow suggestive of oesophageal atresia'.

Q.1. What further investigations and treatment should be undertaken both antenatally and before any surgery is attempted?

At 24 hours of age, operation revealed pure oesophageal long-gap atresia, with no upper or lower fistula demonstrated on bronchoscopy. A feeding gastrostomy was fashioned.

Q.2. How should Caroline be managed postoperatively?

At 7 weeks, it was decided to perform primary delayed oesophageal anastomosis. The distance between the two ends was 1.5 cm, with moderate tension

Q.3. What are the criteria to support the timing of this operation?

Q.4. What are Caroline's specific postoperative needs?

Following discharge, Caroline was having difficulty with feeding and distressing choking neccessitated frequent pharyngeal suction during the night, and particularly in the early morning.

cont. on p. 352

Q.5. What can be done to alleviate this situation?

Caroline's mother was taught to do bougie dilatations under supervision which became necessary at weekly intervals. Although Caroline is well, she only weighs 7.35 kg (below the 3rd centile).

Q.6. What other potential long-term sequelae may occur, and what further treatment might Caroline's condition necessitate?

Q.7. How may Caroline's parents be supported in the future?

Mechanical intestinal obstruction

Many babies vomit, but this is not necessarily serious and may simply be due to gastro-oesophageal reflux, especially if the vomitus is milk, with most of the feed being retained. Pyloric stenosis usually presents with projectile, non-bilious vomiting at around 2–4 weeks. Yellow vomit may not be serious as bile in the gallbladder and duodenum is yellow. However, if it becomes combined with digestive juices, indicating gastric stasis, it turns green, and green vomiting should always be taken seriously. Other danger signs of obstruction which may present insidiously are abdominal distension, poor feeding and failure to pass meconium.

Approximately one-third of cases of obstruction in the neonatal period are suspected antenatally with ultrasound demonstration of dilated bowel (Speidal *et al.* 1998); the remainder may be missed unless there is a high level of clinical awareness. All incidences may be associated with maternal polyhydramnios, fetal growth retardation and prematurity.

Obstruction can occur as a result of paralytic ileus in septicaemia, or in the extremely premature infant with an immature gut, but more importantly as a result of mechanical intestinal obstruction, of which there are numerous causes, necessitating urgent treatment. There are three common causes in the upper part of the gastrointestinal tract, and three in the lower part (Table 15.1); an abdominal X-ray will reveal the position of obstruction. Less common causes of bilious vomiting are incarcerated inguinal hernia, imperforate anus, necrorising enterocolitis and oesophageal atresia. In

***Table 15.1* Causes of intestinal obstruction**

Upper	*Lower*
Duodenal atresia	Low small bowel atresia
High small bowel atresia	Meconium ileus
Malrotation with volvulus	Hirschprung's disease

general, the lower down the small intestine the defect, the later the presentation after birth.

Irrespective of the cause, if obstruction is suspected, a size 8fg–10fg nasogastric tube should be passed, aspirated and left on free drainage. This will decompress the gut and prevent aspiration of gastrointestinal contents. Intravenous therapy should be commenced and fluid and electrolyte imbalances corrected.

Duodenal atresia

The duodenum begins its development in the fourth week of gestation. Epithelial proliferation is so abundant that at 5–6 weeks there is temporary duodenal occlusion, and its failure to become recanalised by the end of the embryonic period results in one of two main groups of atresia. In the first type, the proximal and distal segments of the duodenum end blindly and are separated, or joined by a fibrous cord. The second type consists of partial or complete duodenal webs. Infants with the latter anomaly may feed normally, until a milk curd gets stuck in a partial web and causes obstruction.

Vomiting is the most common symptom of complete atresia, and is usually present on the first day of life, and is usually bilious, as 80 per cent of the obstructions are in the post-ampullary region. Because of the high level, there is minimal abdominal distension. Meconium may be passed in the first 24 hours, followed by constipation. Fluid and electrolyte imbalance will follow if a diagnosis is not made. An X-ray shows high intestinal obstruction with the classic 'double-bubble' —fluid in the lower part of the stomach and in the duodenum proximal to the obstruction (Figure 15.2).

The principle of repair (duodenoduodenostomy) is the same for both anomalies, as it is not possible to remove the web, the bile duct often opening on to it. Parallel incisions are made above and below the web or atresia, and the two ends are anastomosed.

Approximately 30 per cent of neonates with duodenal obstruction also have Down's syndrome, and the incidence of prematurity is as high as 60 per cent (Menardi 1994). There is also an association with VATER and VACTERL syndromes.

Small bowel atresia

Jejuno-ileal atresia is a common cause of intestinal obstruction in the newborn, and is thought to be due to a localised intrauterine vascular accident with ischaemic necrosis of the bowel and subsequent resorption of the affected segment (Cywes *et al.* 1994).

At delivery, green liquor should alert suspicion of obstruction, and postnatally gastric aspirates exceeding 25 ml, persistent bilestained vomiting, progressive abdominal distension and delay in passing meconium are later presenting signs.

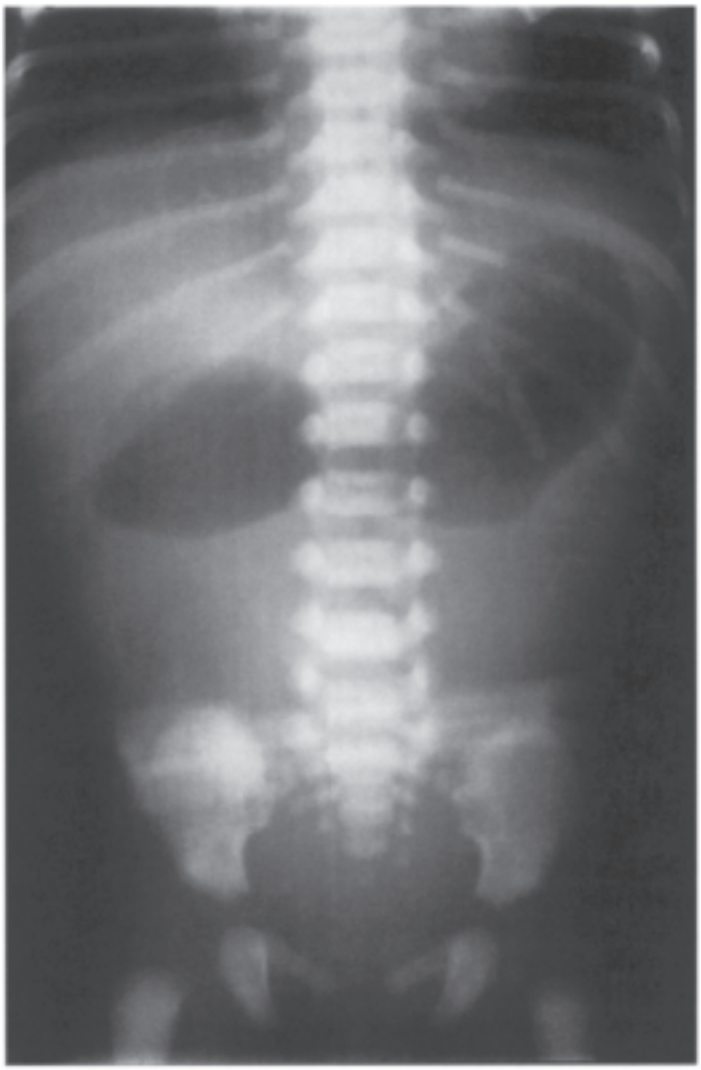

Figure 15.2 **Radiograph showing a classic 'double bubble'**

The X-ray appearance may fall into the high or low group depending on the level of the atresia. At operation the appearances are similar to duodenal atresia, with either a complete atresia or a web. Surgery entails resecting the atretic/ webbed area. Intestinal continuity is restored with an end-to-end anastomosis. If there is sufficient length of bowel, the adjacent distended and collapsed segments should also be resected. The proximal bowel has been dilated, hypertrophied and atonic for many weeks in utero. If it is not possible to remove it, there may be a long period before tone and peristalsis are restored—the higher the atresia, the longer the period of intestinal dysfunction. Once established, gradual weaning from parenteral to enteral

feeding can take place. Insufficient bowel length either as a result of the primary insult, excessive removal of residual bowel, or postoperative complications can lead to short gut syndrome necessitating long-term TPN (total parenteral nutrition).

As there is an association with cystic fibrosis, all infants with small bowel atresia should have a serum immunoreactive trypsin test, gene depletion assay or a sweat test to exclude it (Cywes *et al.* 1994).

Malrotation with volvulus

Development of the midgut results in rapid growth and physiological umbilical herniation during the sixth week of gestation. At 10–11 weeks, the gut begins a 270 degree counter-clockwise rotation around the superior mesenteric artery. The bowel destined to be the caecum re-enters the abdomen descending to the right iliac fossa, where fixation followed by closure of the abdomen occurs around the 12th week (Bass *et al.* 1998). The duodenum with the duodenojejunal flexure (DJF) on the midline or to its left, and the proximal colon, are attached to the posterior abdominal wall. The small bowel is suspended posteriorly by mesenteries which carry blood, lymphatic vessels and nerves, and extends from the DJF to the ileocaecal region.

Normal mesentery has a broad base and cannot undergo torsion. Malrotation occurs when development is arrested and the caecum ends up adjacent to the duodenum. The mesenteries fail to undergo normal fixation, so the small intestines are suspended by a narrow stalk, which is able to twist into a volvulus, causing an obstruction. All green bilious vomiting should arouse the suspicion of volvulus. The X-ray will look similar to the 'double bubble' of duodenal atresia. Unless surgery is carried out promptly, the volvulus continues to twist a few more degrees, the superior mesenteric artery becomes kinked, and the midgut becomes infarcted. Gangrene will then develop with loss of small bowel and part of the colon. The only chance of long-term survival, if this occurs, would be a small bowel transplant, which may work in a few select cases.

Surgery involves untwisting the volvulus as soon as possible. Frequently, Ladd's bands are found between the caecum and the peritoneum causing further obstruction by compressing the duodenum. These are divided and the gut is mobilised and returned to the abdomen with the caecum on the left and the duodenum on the right, broadening the base of the mesentery preventing the tendency to further twisting. Malrotation is thus changed into non-rotation (Ladd's procedure). If the infant's general condition is satisfactory and the caecal area shows no signs of inflammation or pathological changes, an appendicectomy is performed (Kluth and Lambrect 1994). With the ileocaecal valve in the left upper quadrant, appendicitis in later life could pose considerable diagnostic difficulty.

Meconium ileus

Meconium is a dark green mucilaginous material that is a mixture of secretions of the maturing intestinal glands, ingested amniotic fluid and the debris of proliferative epithelial cells. It begins to fill the lower ileum and colon late in the fourth month of gestation and continues until the time of birth (Polin and Fox 1992). Meconium ileus is caused by abnormal meconium blocking the terminal ileum.

Meconium-stained liquor is often reported which actually represents bilious vomiting in utero. At birth, the infant usually has abdominal distension, and continues to vomit. On examination loops of gut may be visible or palpable, and mucous plugs may be evacuated following rectal examination. The X-ray appearance is of a low obstruction.

Perforation and meconium peritonitis may occur, but providing there is no evidence of this and the infant is stable, this obstruction can often be relieved with a Gastrografin enema. Gastrografin is radio-opaque and will demonstrate microcolon with dilated small bowel proximal to the obstructing segment (Kiely 1996). However, it is a quiescently dangerous substance; its hygroscopic action draws large quantities of fluid out of a baby's circulation into the intestinal lumen, causing transient osmotic diarrhoea. Whilst having the advantage of lubricating the abnormal meconium which is then spontaneously evacuated, it can cause severe fluid depletion and cardiovascular collapse. It is therefore crucial that these babies have a good circulating blood volume and that they are carefully monitored during and following the procedure. There is the added risk of intestinal perforation if excessive pressure on the enema syringe is transferred to the bowel wall (Ein 1994).

If Gastrografin is contraindicated or several enemas fail to relieve the obstruction, surgery may be necessary. At laparotomy an incision is made into the bowel just above the obstruction and the abnormal meconium is washed out with Gastrografin and normal saline. The dilated bowel is resected if there is sufficient intestinal length, and continuity is restored with end-to-end anastomosis.

Some 90–95 per cent of babies with meconium ileus have cystic fibrosis, and 15 per cent of these present in the neonatal period with obstruction caused by the tenacious meconium (Ein 1994).

Hirschprung's disease

Hirschprung's disease is due to an absence of enteric ganglion cells in the submucosa of the distal bowel. They first appear in the developing oesophagus at 5 weeks and migrate down to the ano-rectal junction by 12 weeks. Their absence is attributed to the failure of migration, and the earlier the arrest of migration, the longer the affected segment of bowel. The aganglionic segment always includes the rectum, and the total colon is affected in 8 per cent of cases (Puri 1996b).

The aganglionic segment is non-peristaltic, causing functional intestinal obstruction. Abdominal distension, bilestained vomiting and failure to pass meconium are presenting signs. On X-ray, the normal bowel appears as megacolon with dilated small bowel proximal to the aganglionosis. When a gentle rectal examination is performed, the rectal wall always appears tight and resists further probing. This may cause the passage of meconium and flatus followed by normal bowel movements for a few days, before signs of obstruction recur. Enterocolitis—indicated by the presence of foul-smelling diarrhoea and potential perforation—may occur. Undiagnosed Hirschprung's disease can be fatal.

Diagnosis is confirmed with suction biopsies of the colorectal mucosa, which is a simple and apparently painless procedure. Surgery should be carried out with a pathologist available to examine frozen sections. A stoma is fashioned at the most distal part of normal innervation to decompress the bowel, whilst allowing the baby to feed and grow. Definitive surgery to remove the aganglionic segment and restore intestinal continuity usually takes place at 4–6 months of age.

Recently there has been a move towards performing definitive surgery without a stoma. Preoperative bowel decompression is carried out with daily rectal washouts using a Jacques catheter and saline.

Anorectal abnormalities

Anorectal abnormalities result from anomalous development of the urorectal septum, causing incomplete separation into urogenital and anorectal sections, and an aberrant anal orifice (Moore and Persaud 1993). They present with a spectrum of defects—from minor malformations requiring minimal treatment, to a very sick infant with intestinal obstruction and complex life-threatening defects (Pena 1996). Anomalies of the upper urinary tract, cardiovascular system and sacrum are associated, as are atresias of the gastrointestinal tract.

Lesions are classified depending on whether the rectum ends superior or inferior to the puborectalis sling. High lesions present as anorectal agenesis or rectal atresia, frequently associated with recto-urethral, -vestibular or -vaginal fistulae. Alternatively, low lesions are classified as anal agenesis, stenosis, or an imperforate anus (which may be just a thin membrane through which meconium can be seen, or excreted). Low lesions are associated with anocutaneous fistulae, and, in girls, an ectopic stenotic anus.

All newly born infants should have a rectal temperature taken, which will exclude an imperforate anus. A lateral X-ray with the infant prone and head down, with a radio-opaque marker over the skin dimple (at 24 hours of age to allow air to reach the lower bowel), will identify the level of obstruction, whilst revealing any associated sacral abnormalities.

Low lesions require division of fistula or anoplasty, and may need frequent subsequent anal dilatations, but the prospects of long-term faecal continence are excellent.

High lesions require formation of a defunctioning sigmoid colostomy, anorectoplasty at several months of age, followed by subsequent closure of colostomy. Some of these infants never achieve reasonable continence (Speidal *et al.* 1998), and long-term enema management may be neccessary. Urinary tract infections are common, especially with high lesions, and delayed treatment when there is a fistula can lead to progressive rectal distension and rectal inertia.

Congenital diaphragmatic hernia (CDH)

During embryological development the thoracic and abdominal portions of the body cavity move freely until the diaphragm develops to separate them (Moore and Persaud 1993). CDH results from defective fusion of the pleuro-peritoneal membrane when the intestines return to the abdomen from the umbilical cord, and abdominal viscera slip into the thorax. It usually occurs on the left side (8:1), through the posterior foramen of Bochdalek, but can also occur near the xiphisternum through the foramen of Morgagni, or on the right.

CDH is considered to be a syndrome, and it is speculated (Steinhorn *et al.* 1997; Thebaud *et al.* 1998) that an insult at a critical time of embryological development could lead to varying degrees of:

- Pulmonary hypoplasia, which is a result of impaired lung growth and bronchial division when the diaphragm fails to fuse. As terminal bronchi can only support a limited number of alveoli, there is a reduction in the gas exchange area. The compressed ipsilateral lung is obviously hypoplastic, although the contralateral lung may also show changes.
- Lung immaturity, which is further decreased by surfactant dysfunction. Prenatally diagnosed fetuses with CDH show amniotic lecithin/sphingomyelin ratios similar to surfactant-deficient premature neonates.
- Left-heart hypoplasia caused by mechanical compression by the herniated organs, or altered haemodynamics because development of the heart chambers depend on the blood flow they receive. At birth, hypoxia and hypercarbia trigger constriction of the abnormally thick-walled pulmonary arterioles, resulting in persistent pulmonary hypertension of the newborn (PPHN) (see pp. 106 and 136).

All these factors decrease gas exchange across the alveolar capillary membrane and lead to intrapulmonary shunting. If these processes are not reversed forthwith, decreased cardiac output and oxygen delivery become insufficient to sustain life. The mortality rate (50–60 per cent) is associated with the hypoplastic lungs, cardiac malformations and hypoxia-induced IVH.

CDH accounts for 8 per cent of all major congenital abnormalities. It can occur singly or as part of multiple malformation syndromes such as Fryn's (Steinhorn *et al.* 1997), and the pentralogy of Cantrell (Cantrell *et al.* 1958). The association with congenital heart and renal malformations is especially high.

Infants with a left-sided defect, especially if preterm, usually present with respiratory distress. This can range in severity from cyanosis totally unresponsive to intervention and incompatible with life, through tachypnoea, nasal flaring, recession and cyanosis not improved with supplemental oxygen. Some infants are asymptomatic or have mild symptoms not diagnosed for hours, days or even months.

Other clinical signs include a scaphoid abdomen with increased chest diameter, a shift in the trachea and cardiac impulse, and decreased breath sounds —but with bowel sounds in the chest on the ipsilateral side. Radiologically, there is bowel in the thorax and an absent diaphragm (Figure 15.3). Malrotation may coexist. The main differential diagnosis is congenital cystic adenomatous malformation of the lung (CCAM).

Management is aimed at correcting these abnormalities antenatally (rare) or ameliorating them by preventing PPHN postnatally. Little literature is available to support fetal intervention, although correction of CDH in fetal lambs has reversed associated problems (Steinhorn *et al.* 1997). Decreased surfactant activity in these infants suggests maternal antenatal steroids may be of value.

Some of these infants may be critically ill at birth and recovery can be a lengthy, vulnerable process. At delivery, it is important to avoid bag-and-mask ventilation; if air is forced into the gastrointestinal tract in the thorax, respiration will be further compromised. Early intubation to maintain adequate oxygenation is usually neccessary with IPPV or HFO ventilation. HFO maintains lung volume, permits adequate gas exchange with small tidal volumes, and has been shown to recruit collapsed alveoli. Exogenous surfactant may be of value (Steinhorn *et al.* 1997). Placement of an umbilical arterial catheter or a preductal peripheral artery cannula (more accurate when measuring PO_2 in PPHN) is crucial for determination of blood gas values. Gastrointestinal decompression with a nasogastric tube on continuous drainage with intermittent aspiration is of the utmost importance.

Intravenous access is a priority to provide adequate perfusion, although fluid restriction may be necessary. These infants do not tolerate positive fluid balance; increased circulatory volume could lead to pulmonary oedema worsening the respiratory status.

Support with inotropic agents may be necessary, and tolazoline may be required to improve oxygenation (see p. 122). Inhaled nitric oxide (NO) therapy may also be an effective vasodilator, but it is not universally effective in improving oxygenation in CDH infants, unless used in conjunction with extracorporeal membrane oxygenation (ECMO) (Steinhorn *et al.* 1997). ECMO (see p. 121) provides partial cardiopulmonary bypass in infants who have failed other modalities of respiratory and cardiac support, but have reversible cardiopulmonary failure. Although surfactant therapy and HFO ventilation *may* recruit collapsed alveoli, and inhaled NO *may* relax constricted pulmonary arteries, these treatments will obviously fail if there are insufficient alveoli and vessels in the hypoplastic lung to support life, and these innovations do not appear to have decreased the mortality in the last decade (Thebaud *et al.* 1998).

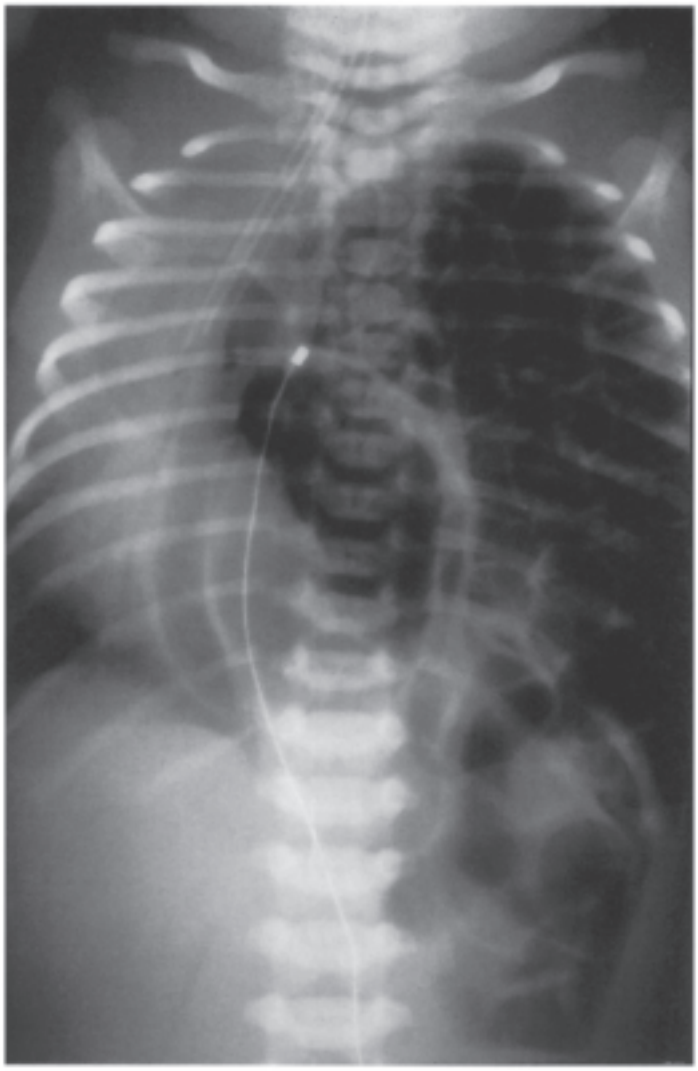

Figure 15.3 **Radiograph showing a left-sided diaphragmatic hernia shortly after birth**

Immediate management of CDH may be thus summarised:

A	Airway	intubate
B	Breathing	IPPV
C	Circulation	IV fluid bolus
D	Decompression	naso-gastric tube
E	Ensure good gases	

(Davenport 1999)

Until recently, repair of the diaphragm and reduction of herniated viscera were performed as an emergency, immediately following birth. Today, a delayed approach (up to 7–10 days) allows for adequate preoperative stabilisation, and probably improves survival. The optimal time for surgery is considered to be after 24 hours of stability with a PaO_2 of more than 8 kpa in less than 50% supplemental oxygen, with low rates and pressures (Davenport 1999). Surgery involves reduction of the viscera and repair of the defect through a subcostal incision (Figure 15.4). Rarely, the defect is repaired with a synthetic patch, or transposed latissimus dorsi or abdominal oblique muscle flaps. A chest drain with underwater seal is usually inserted on the ipsilateral side, to drain surgical losses, and should be managed accordingly.

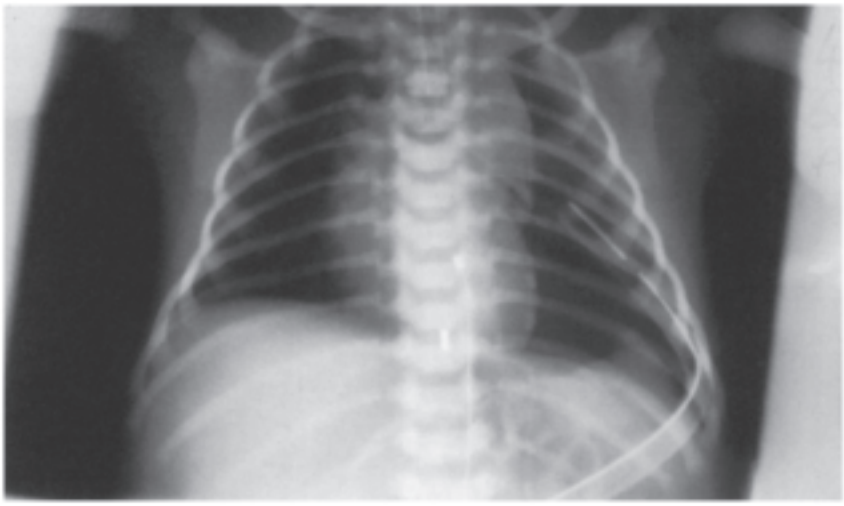

Figure 15.4 **Chest radiograph following surgical repair of diaphragmatic hernia**

Initial postoperative care is not significantly different from preoperative management: the goal is still to avoid PPHN and associated shunting; if there are still major problems, in most cases surgery was performed too early.

Ventilation may be difficult due to the tight abdominal closure, as well as the mediastinal shift splinting the contralateral lung in an overinflated, non-compliant state. High inspiratory pressures are necessary to maintain adequate oxygenation, and the FiO_2 should always be increased prior to endotracheal suction to prevent oxygen-deficit pulmonary vasoconstriction. Pneumothorax is common in the immediate postoperative period due to the high ventilatory pressures in spite of muscle relaxant use. Treatment is the immediate insertion of a chest drain. Ventilatory support can be weaned when the infant is stable, with normalised blood gases.

Pain control should be managed as per individual unit protocol—either by morphine (see pp. 204 and 325) or epidural infusion (see p. 328). Calorific requirements for healing, growth and development are maintained with TPN

until the gastrointestinal tract has recovered from the effects of surgery, sedation and paralysis.

Apart from postoperative complications of infection, fluid and electrolyte imbalance and haemorrhage, there may be long-term decreased lung function. Diaphragmatic eventration can occur as a separate entity, or occasionally after thoracic surgery, and requires plication. Chylothorax is a rare complication, and following thoracentesis and recovery, the infant will require semi-elemental milk (Greenough and Roberton 1999). Gastrointestinal reflux occurs in approximately 40 per cent of cases (10–20 per cent requiring fundoplication). Some 5–10 per cent will have a neurological handicap presumed to be due to the hypoxia and cardiovascular instability, or as a complication of ECMO (Davenport 1999).

Abdominal wall defects

Exomphalos and gastroschisis

The embryology of normal abdominal wall closure, and the sequence of events leading to umbilical and para-umbilical defects, is speculative. Some researchers feel that gastroschisis is the end result of a ruptured exomphalos, whilst others feel these two conditions have different pathology and embryology (Anveden-Hertzberg and Gauderer 1996).

The most popular description of exomphalos is a developmental arrest resulting in failure of the midgut to return to the abdominal cavity. The amount of herniated intestine varies from a small umbilical hernia-like lesion, to a huge anomaly, containing the entire midgut and liver. Unless ruptured, it is covered with a sac consisting of peritoneum and amnion, the umbilical cord emerging from the caudal part. Eviscerated bowel in exomphalos is usually normal in appearance.

Gastroschisis is due to incomplete closure of the lateral abdominal folds, leading to a defect in or near the median plane of the ventral wall leading to protrusion of the intra-abdominal viscera. There is no membrane covering the herniated bowel, and the cord is intact. The eviscerated bowel is often foreshortened, inflamed, thickened and matted with serosal peel. Clinical research suggests that this damage is caused by prolonged exposure to urine in the amniotic fluid, and/or progressive constriction on the intestine and its blood supply by the umbilical ring (Simmons and Georgeson 1996).

Accurate antenatal diagnosis can be made as early as 12 weeks of gestation, as an exomphalos in its sac is distinguishable from gastroschisis, the ultrasound appearance of which has been likened to a honeycomb due to the freely floating, thickened bowel loops in amniotic fluid (Molenaar 1996).

Although exomphalos and gastroschisis have similar incidences, malformations associated with gastroschisis are uncommon, apart from relatively few instances of coincidental intestinal atresia or stenosis. It can, however, be complicated by oligohydramnios severe enough to put the fetus at risk of

pulmonary hypoplasia, limb deformities and fetal distress, and is common in premature infants of young mothers. The affiliation of chromosomal and cardiac abnormalities with exomphalos is well recognised (Fisher *et al.* 1996).

There is much debate surrounding the issues of early versus late delivery, and cesarean section versus vaginal delivery for these infants (Cusick *et al.* 1997). Some authors feel there is no evidence that the mode of delivery improves the final outcome (Molenaar 1996), and infants with unsuspected exomphalos have been delivered vaginally with an intact sac.

Following delivery, the priority is protecting the sac from rupture and infection. Antibiotics should be commenced as soon as intravenous access is established. Large amounts of heat may be lost from the exposed bowel in gastroschisis, or through the amnion covering the exomphalos. The lesion should be immediately covered with cling-film to protect it from trauma and contamination, and to prevent traction on the mesentery. A nasogastric tube connected to low suction, or left on free drainage with intermittent suction, will decompress the gut and prevent aspiration. Respiratory distress should be managed appropriately, and all infants should have chest and abdominal X-rays to evaluate the lungs, heart, diaphragm and the air pattern in the bowel.

Significant protein and insensible fluid losses from the abnormal bowel in gastroschisis is unavoidable, and should be supplemented parenterally. Perfusion and blood pressure should be carefully monitored, as should blood sugar, as there is an association between exomphalos and Beckwith-Wiedemann syndrome (Speidal *et al.* 1998).

Surgery should take place as soon as the infant is haemodynamically stable, although in a non-ruptured and small exomphalos there is usually no urgency. Primary closure is preferred, and postoperatively artificial ventilation with initial muscle relaxation may be required. Recovery can be complicated by prolonged ileus and gastro-oesophageal reflux (Fasching *et al.* 1996). Early establishment of enteral nutrition can lead to necrotising enterocolitis requiring aggressive medical management, or even further surgery (Molenaar 1996), therefore parenteral feeding is advocated for some time. Sepsis is a continuing risk, and antibiotics should be continued for 7–10 days.

A large exomphalos containing centrally herniated liver poses challenges—the most significant being space limitations of the abdominal cavity, and hypotension as well as respiratory distress from diaphragmatic splinting. In addition, the liver may exert pressure on the vena cava creating acute hepatic vascular outflow obstruction and renal compromise (Skarsgard and Barth 1997). If primary closure is not feasible, a Silastic silo is an option. Prosthetic sheets are sutured to the medial edges of both rectus muscles, with the free edges sutured together in the midline. As more abdominal space becomes available, over 7–10 days, progressive plication of the sheets is executed, until the silo contents return to the abdomen and fascial closure is possible (Bax 1994).

Lee *et al.* (1997) have proposed the use of suture-less steridrape for a silo, whilst Gharib *et al.* (1996) suggest using a double-layer amniotic graft. Amnion is a freely available auto-plastic biological membrane; reefing and removal are unnecessary, and adhesion formation is unlikely.

The more recent conservative treatment of large exomphalos avoids the respiratory distress and caval vein obstruction that often follows primary closure, whilst permitting more time to assess associated abnormalities. Reconstruction of the abdominal wall is delayed for some months, allowing granulation, epithelisation and subsequent contraction to gradually reduce the sac size (Molenaar 1996). Various applications can be used to protect the sac, although infection may be an inherent problem. The infant, meanwhile, can safely be managed at home.

The prognosis of infants suffering from gastroschisis is infinitely better than those with exomphalos (Molenaar 1996). Ischaemic changes in the wall of the damaged intestine in gastroschisis may cause absorption and motility disturbances for some time after repair (Simmons and Georgeson 1996), and mortality is usually due to short-gut syndrome, or complications of long-term parenteral nutrition. Prognosis of exomphalos is dependent on the severity of associated abnormalities.

Umbilical hernia

This is due to a protrusion of a loop of bowel through the linea alba into a patent umbilicus. Unlike exomphalos, it is covered by subcutaneous tissue and skin. It ranges in size from 1 to 5 cm, reaching a maximum by the end of the first month, and distends during crying. It is relatively common, complications are rare and the deficit will often close by cicatrisation during the early years of life, umbilical herniorrhaphy being safely reserved for persistent lesions (Johnstone 1994).

Inguinal hernia

An inguinal hernia develops, often bilaterally, because an outpouching of the peritoneum through the inguinal canal fails to obliterate allowing abdominal viscera to prolapse (Puri and Surana 1996). It is one of the most common surgical conditions in infancy, the incidence increasing rapidly in preterm and small for gestational age (SGA) infants (Johnstone 1994), presumably precipitated by respiratory effort and artificial ventilation. It is more common in males, often associated with undescended testes (Moore and Persaud 1993), and can be secondary to increased abdominal pressure with necrotising enterocolitis and tight gastroschisis/exomphalos repairs.

Presentation is a swelling in the groin, extending to the scrotum or the labia. It is usually first noticed by the parents, and may only be visible whilst the infant is crying, feeding or straining to pass a stool.

The hernia can usually be reduced initially by gentle pressure, when it will return to the abdomen with a characteristic 'gurgle' (Johnstone 1994). Whilst it remains reducible and asymptomatic, most centres delay definitive surgery until the preterm infant is ready for discharge. Uemura *et al.* (1999),

however, recommend earlier repair to avoid operative difficulties. Although the premature infant may be at risk, warranting intensive care, the sac is small and easier to separate from the cord; and incarceration and gonadal ischaemia are prevented. Inguinal herniotomy consists of reduction of the contents into the abdomen, reconstruction of the posterior wall of the inguinal canal, ligation of the hernial sac and return of the testes to the scrotum (Wright 1994). Much controversy surrounds the question of surgical exploration contralaterally.

Repair of a simple hernia in a term infant is safe in an appropriate environment, with few complications. Wound infiltration with local anaesthetic is effective as postoperative pain relief, and feeding may be resumed as soon as the infant is awake.

Caffeine has been shown to reduce the incidence of postoperative apnoea and bradycardia, and herniotomy under local anaesthetic block is advocated for infants at particular risk.

If the hernia is not easily reducible it can become incarcerated or strangulated with vascular compromise of the sac contents. If this occurs, the baby will usually be in pain, and show signs of toxicity and shock, abdominal distension and constipation, and possibly bloody stools. Abdominal X-ray will show bowel, fluid levels and abdominal gas within the hernia. The infant should be nursed with the buttocks elevated until a surgical opinion can be sought. Ideally, if the infant is stable, non-operative reduction under sedation is preferred (Puri and Surana 1996), although repeated incarcerations require surgery.

If the hernia strangulates, haematological and biochemical assessment, and possible stabilisation, are necessary, prior to laparotomy and repair. Resection and anastomosis may be necessary if the affected bowel is non-viable. Post-operatively, intravenous fluids, nasogastric aspiration and antibiotics will be necessary until peristalsis is established.

Occasionally haematoma and wound infection develop. Late complications such as recurrence of the hernia 3 weeks to 4 years later, testicular infarction/atrophy following incarceration, and iatrogenic high testicle are rare (Johnstone 1994).

Hydrocoele

Occasionally the abdominal end of the processus vaginalis remains open but is too small to permit herniation of the intestine. Peritoneal fluid passes into it and forms a hydrocoele—a painless collection of fluid around the testicle, presenting as a soft, non-tender, translucent swelling. Hydrocoeles are very common, usually resolve spontaneously, and are of no significance unless they become very large and tense, when a surgical opinion should be sought, to avert torsion of the testes. Hydrocoele is sometimes difficult to differentiate from an incarcerated hernia; a rectal examination will exclude the latter (Puri and Surana 1996).

Most commonly encountered acquired disorders

Necrotising enterocolitis (NEC)

NEC is an inflammatory disease of the bowel, predominantly affecting premature infants, but sometimes occurring in 'epidemics' (Boston 1996). The specific cause remains enigmatic, but several putative risk factors have been identified which suggest the pathophysiology is multifactorial. Epidemiological studies reveal that most aetiological factors describe events in a population of physically stressed high-risk neonates. There appears to be a complex relationship between mucosal injury, infection and hyperosmolar enteral feeds.

Mucosal injury

Hypoxia and systemic hypotension lead to sparing of the vital organs at the expense of the gut, which is vulnerable to underperfusion and ischaemic damage. Factors associated with hypoxic stress include prolonged rupture of membranes, placental abruption, low Apgar scores, RDS and apnoea. Left-to-right shunting through a PDA compromises blood flow to the gut, and the presence of umbilical catheters is also implicated.

Microbial infection

There is little evidence that infection is the cause of NEC, but it is an important factor in the pathogenesis. Damaged or immature epithelium may lead to a leaky mucosal barrier allowing hydrogen- and toxin-producing micro-organisms to invade the gut wall, and decreased levels of IgG contribute to this mucosal damage (Hebra and Ross 1996). Although no single organism is consistently associated with NEC, blood cultures may be positive in 20–30 per cent of cases (Kliegman and Walsh 1992).

Enteral feeds

Hyperosmolar feeds, or a too rapid increase in volume, can damage the mucosa (Boston 1996), and intra-mural milk provides a substrate for bacteria. Ninety per cent of infants with NEC have received oral feeds. Breast milk, however, has been shown to have a protective effect (Hebra and Ross 1996) (see p. 249). Milk distending the stomach can lead to a reduction in lung volume with a consequent fall in PaO_2 and vascular compromise.

NEC can affect any part of the gastrointestinal tract, but the most frequently affected are the terminal ileum and the splenic flexure. It has a variable course, ranging from mild abdominal distension without systemic symptoms, to gradual clinical deterioration with lethargy, thermal instability and apnoeic episodes.

Severe signs of sepsis may follow, with bradycardia, pallor, skin mottling, jaundice and haemorrhaging secondary to DIC (see p. 180). There is usually an absence of bowel sounds, abdominal tenderness, a palpable mass with distended loops of bowel and a red indurated abdomen with periumbilical flaring. The classic 'triad of signs' is abdominal distension, bloody mucousy stools and bilious aspirate. Fulminant sepsis, multi-system failure and death may ensue.

Abdominal X-ray confirms the diagnosis, showing distended loops of oedematous bowel, pneumotosis intestinalis, pneumoperitoneum, gas in the portal vein and, on serial films, fixed distended loops of bowel and progressive ascites. Other investigations should include biochemical and haematological assays, blood gases and a septic screen.

Treatment for mild to moderate NEC, whilst monitoring for deterioration, is with antibiotics, intravenous feeding for up to 10 days and nasogastric decompression. More unwell infants may require platelet transfusion, supplemental oxygen, analgesia and, in severe cases, full respiratory support, inotropes, fluid resuscitation and blood transfusion, with operative intervention as a lifesaving measure.

Although it is not established practice, clinical trials have shown that intravenous IgG combats established infection, by exerting an immunoprotective effect on the gastrointestinal tract if given preoperatively to infants with NEC (Rowe *et al.* 1994).

Surgery is required when there is continued clinical deterioration, and drainage of the peritoneal cavity—although of debatable value—may be a first step for severely ill infants who would not survive anaesthesia and operative intervention. A Penrose drain is inserted percutaneously under local anaesthesia, for the evacuation of gas, exudate, pus and faeces (Koloske 1996).

Definitive surgery is usually necessary within five days of diagnosis, when all monitoring parameters would have improved had conservative treatment been successful (Hebra and Ross 1996), or if perforation occurs. Laparotomy and peritoneal toilet are performed, with further surgery aimed at preserving the maximum length of intestine whilst removing the source of sepsis. The affected gut may be just a punched-out perforation, or a much larger area which is dilated and purplish, with denuded areas of mucosa, but still viable. Alternatively there may be necrotic or totally disintegrated areas. If resection and anastomosis of the gut are not possible, formation of one or more stomas will be necessary. These can be closed at a later date providing there are no strictures, but a permanent proximal stoma may be the only treatment for NEC-totalis.

Post-operatively, the stoma should be checked regularly to make sure it is red and healthy, and that the skin around it shows no sign of irritation. Initially, a non-adherent dressing may be used, but when the stoma begins to function, a suitable-sized bag should be applied, and kept in situ for as long as there is no leakage, thus preserving skin integrity.

There is a risk of recurrence of NEC if enteral feeds are introduced too early. Strictures are the main concern, and rarely fistulae occur and are thought to follow sub-acute intestinal perforation. Malabsorption, failure-

to-thrive and weight loss may be related to the mucosal injury, and short gut syndrome may follow extensive resection (Hebra and Ross 1996). Despite improved prognosis, the aggressive form of the disease has not decreased and is still associated with significant rates of morbidity and mortality (Ade-Ajayi *et al.* 1996).

Case study: postoperative management of an infant with NEC

Maternal antepartum haemorrhage progressing to preterm labour at 28 weeks' gestation led to the delivery of a healthy male infant, weighing 1.24kg.

By 24 hours Tom had increasing respiratory distress, pyrexia, tender, abdominal distension with periumbilical erythema. Haematologically, there was an initial neutrophilia, which was rapidly reversed, accompanied by thrombocytopenia. No organisms were found on blood culture. Clinical deterioration continued, with tachycardia, hypertension and respiratory acidosis.

Q.1. What was the probable diagnosis?

Q.2. What treatment was instigated?

After confirmation of the diagnosis, Tom required urgent surgery. Perforation of the ileum and free gas, turbid yellow fluid and meconium were found in the peritoneum at laparotomy. The bowel was resected and a double-barrelled defunctioning ileostomy fashioned to allow the bowel to heal. Microscopic examination of the submucosa was entirely consistent with the diagnosis of NEC.

Q.3. What is Tom's postoperative management?

Q.4. What complications might he develop?

Q.5. What follow-up care is necessary for Tom and his family?

Spontaneous bowel perforation

Small infants continue to have a high mortality rate after spontaneous gastrointestinal perforation and subsequent development of peritonitis and sepsis, despite advances in perinatal care (Grosfeld *et al.* 1996).

There are mutifactorial associated factors. Perforation can occur following hypoxic, ischaemic challenges to the fetus, when blood is shunted to the heart and brain at the expense of the extremities and the mesentery (Mintz and Applebaum 1993). Fetal exposure to indomethacin via the placenta

can depress prostaglandin synthesis, causing premature closure of the ductus arteriosus with pulmonary hypertension, and associated hypoxic, ischaemic events (Vanhaesebrouck *et al.* 1988). High-pressure IPPV, congenital defects of the intestinal musculature, infection and dexamethasone therapy are common causes of spontaneous perforation; whilst nasal-prong and face-mask ventilation are implicated in a disproportionately high number of cases. Other causes are a result of intraventricular haemorrhage, hypotension and dehydration. Umbilical catheters have also been implicated, as they can develop adherent thrombi, and focal perforations could be a result of subsequent small septic emboli to the bowel. Other reported cases have occurred following inexpertly performed suprapubic aspiration and abdominal paracentesis, and the use of transpyloric feeding tubes (Duck and Bloom 1995). The ileum is the most commonly affected part of the bowel.

These infants usually present with a bluish/purple discoloration of the abdomen, and abdominal X-ray shows pneumoperitoneum, but no pneumatosis or portal vein gas as in NEC (Mintz and Applebaum 1993). Surgical reports typically describe normal-looking bowel, apart from localised focal perforation, which can be resected. Microscopic examination shows transparent serosal windows near the perforation, usually with no necrosis or inflammation (Duck and Bloom 1995). *Staphylococcus epidermis* or *Candida* are commonly grown on peritoneal fluid culture.

Miscellaneous disorders

Cystic hygroma (see p. 319)

The fetal lymphatic system develops around 5 weeks' gestation. Hygroma results from failure of the establishment of drainage into the venous system leading to pathological accumulations of fluid close to large veins and lymphatic ducts. A generalised lymphatic obstruction sequence may develop into progressive hydrops with a webbed neck, peripheral oedema and abnormal lymph flow; or even death (Goldstein *et al.* 1994).

Detection of fetal cystic hygroma in the second trimester of pregnancy is associated with an increased frequency of chromosomal abnormalities, so may warrant termination of the pregnancy (Trauffer *et al.* 1994). If it proceeds, vaginal delivery may be possible, but cesarean section should be considered for major lesions. Respiratory distress is the most significant complication, most commonly in infants with very large masses compromising the airway. Facilities for immediate intubation and ventilation (with the possibility of tracheostomy) are mandatory (Goldstein *et al.* 1994).

Some 50–60 per cent of cystic hygromas are identified at birth, with a discrete, soft, mobile, non-tender transilluminable mass. Their size varies

from a few millimetres to several centimetres, and they consist of fluid-filled cavities lined with endothelial cells, which continue to produce lymph (Moore and Persaud 1993). As they enlarge, adjacent structures are compressed or stretched. Lesions involving the tongue, or extending into the mediastinum, can result in progressive respiratory involvement, and failure to thrive.

Spontaneous regression is rare, and aspiration of the fluid is usually followed by re-occurrence, haemorrhage and infection; although it may prove necessary for emergency relief of acute respiratory distress. Neonates with large lesions causing tracheal compression require urgent resection. Surgery may best be deferred until 2–6 months of age, so long as the rate of growth is proportional to body growth (Brown and Azizkhan 1998). Hygromas are benign, so although every effort should be made to excise the cyst completely, no major nerves or vessels should be sacrificed.

Teratoma

Teratomas are neoplasms containing derivatives of one or more of the embryonic germ layers. Sacro-coccygeal teratomas are the commonest extra-gonadal tumour in neonates, especially in girls. They may be identified antenatally, and planned cesarean section will avoid dystocia, rupture or haemorrhage, as some are highly vascular. These infants should be protected following delivery, to prevent erosion of the surface; and blood for transfusion should be readily available.

On examination, there is a large skin-covered mass overlying the sacrum and coccyx, frequently displaying the anus anteriorly, palpable abdominally. Large lesions require early surgical referral, as untreated they can cause pain, constipation and urinary tract infections, and intra-spinal extension can cause lower motor neurone damage (Pollak *et al.* 1996). The important differential diagnosis is from a skin-covered spina bifida. Postoperatively the infant should be ventilated and nursed prone, with a urinary catheter in situ.

Teratoma of the cervical region is one of the rarest causes of neonatal respiratory distress, and requires urgent intervention. The lesion can be 8–12 cm in diameter and appear firm, tense, cystic and multi-nodular. Complete excision is required, and delayed surgery is only feasible in unstable patients.

The majority of teratomas occurring in neonates are benign, but histopathological studies are important to rule out malignancy and adjacent tissue invasion (Hany-Hassab *et al.* 1996).

Ovarian cyst

The typical neonatal and infant ovary is heterogeneous and cystic. Neonatal cysts are primarily of follicular origin and probably result from disordered folliculogenesis. The incidence is increased in neonates of mothers who have toxaemia. The larger placenta, with increased permeability to the hormones

associated with maternal diabetes and rhesus incompatability, is also thought to increase the risk of ovarian cyst formation (Aslam *et al.* 1995). With improved imaging techniques, the intrauterine diagnosis of ovarian cysts is encountered more frequently.

Postpartum management is contentious, because of potential malignancy. Cysts of less than 4 cm, that are uncomplicated (without solid components, septa, or debris) can be managed conservatively in the hope that they will resolve after cessation of hormonal stimulation. Surgical removal should be performed if the cyst increases in size, develops complications, or fails to resolve. Laparoscopy is well tolerated by newborns and will allow for diagnostic visualisation, biopsy and therapeutic intervention of the cyst if neccessary (Van der Zee *et al.* 1995).

Torsion is the most common complication; and can occur antenatally. Postnatally, torsion may be accompanied by pain, vomiting, fever, abdominal distension and peritonitis. Gastrointestinal obstruction, urinary tract obstruction and rupture can also occur (Aslam *et al.* 1995).

Testicular torsion

In neonates, extravaginal torsion of spermatic cord consists of multiple rotations proximal to the attachment of the tunica vaginalis; either in the inguinal canal or just below it. Torsion commonly occurs antenatally (Azmy 1994). The neonatal testis may be prone to torsion because of its extreme mobility within the scrotum. It is largely a condition of large term babies (Burge 1996).

On examination, the scrotum is swollen (two to three times larger than the other side), firm, discoloured and painless if antenatal torsion has already led to testicular necrosis. There is oedema, erythema and a distinct fixation to overlying skin. The swelling will not transilluminate.

Because the potential for testicular salvage is remote, early operative intervention will confirm the diagnosis (Stone *et al.* 1995). Following excision of the affected testis, the contralateral side is usually explored prior to fixation. Strangulation of the testicle may be secondary to an irreducible hernia (Johnstone 1994).

Biliary atresia

Biliary atresia occurs due to failure of recanalisation of biliary ducts, or liver infection in late fetal life.

The diagnosis is usually made from the recognition of prolonged jaundice in the first few weeks of life, followed by the development of pale stools and hepatomegaly. Haematological studies are mandatory for an overall evaluation. Although rarely significant in the the differential diagnosis from other non-surgical causes of jaundice, it is important to distinguish biliary atresia from neonatal hepatitis. If significant doubt remains, a liver biopsy should be performed, followed, if this is equivocal, by laparotomy and cholangiogram.

Accurate and rapid diagnosis is crucial if surgery is to be completed before 12 weeks of age (Kimura 1996). After this time, liver damage is progressive and the child may die if a liver transplant is not performed (Moore and Persaud 1993). Hepatic transplantation is also recommended for the management of patients who deteriorate after initial correction with portoenterostomy (Kimura 1996).

Conclusion

In today's climate, prenatal diagnosis of malformations requiring surgery is possible, and represents a major advance in their management. Neonatal teams are usually able to transform what was once an unplanned emergency service, with associated anxieties, into one that is essentially semi-elective with, hopefully, a consequent reduction in parental stress.

Neonatal surgery has reached a high degree of sophistication and demands centralisation in designated centres and the expertise of a full range of specialties. The fetus is usually in no danger until delivery, so negotiating intrauterine transfer is probably safer than the resuscitation and transport of a decompensating neonate (Theorell 1990). Family counselling, the timing and mode of delivery, and the availability of services can be optimised at the tertiary centre, and subsequent maternal-infant bonding can take place without the disruption of an emergency transfer.

However, neonates, particularly preterm infants, have little tolerence to changes in normal physiological parameters, especially when in a compromised state. Accordingly, they endure surgery much better after punctilious preoperative stabilisation. Compounded complications can be abated with a more delicate surgical technique, effective and safe pain-relief, improved antibiotics, together with implicit management of respiratory status using sophisticated ventilatory techniques, thermoregulation, and fluid, electrolyte and acid base homeostasis.

Surgery is traditionally carried out in the operating theatre. Associated with this is transportation, potentially hazardous manipulation of ventilation and interruption of the continuity of care. Gavilanes *et al.* (1997) found that the NICU could be a suitable place for major surgery, with no increase in infection or perioperative mortality rates. However, a footnote to this study delineates the difficulties of performing intricate surgery with inadequate lighting. Improved outcomes for these very sick infants, therefore, can be achieved with superior transport facilities, centralisation of specialist surgical units and the provision of dedicated neonatal theatres close to the NICU.

Neonatal surgery presents a multidimensional challenge, and unequivocal outcome exacts the coordinated, cooperative and complementary skills of the surgeon, the specialist anaesthetist and the neonatologist. Together with the nursing staff, many of whom are competent in extended roles and find the care of the surgical neonate to be stimulating, they can improve both the survival and the quality of life of these tiny patients.

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Chapter 16

Neonatal Transport

chapter 16

Contents

Introduction

Transferring babies within or between hospitals is a significant element of neonatal intensive care nursing. All critically ill neonates should have access to a NICU, and a service trained to move them swiftly but safely to these facilities. Transportation of sick infants is an elaborate enterprise, challenging the integrated expertise of diverse personnel. Communication, teamwork and attitude are the essence of an efficient transport system (Bloodworth 1995). The aim is to stabilise the neonate prior to transport, and to deliver the patient in optimal condition, whilst providing a level of care indistinguishable from that available in the NICU (BPA 1993).

Until comparatively recently in the UK at least, transfer was largely the responsibility of a locally organised *ad hoc* team who commonly had inadequate training, resources and equipment (Krug 1995). Staff were not in an optimal position to manage the infant en route, nor to deal with such emergencies as physiological deterioration, loss of intravenous access or endotracheal tube misplacement (Kelly *et al.* 1996).

More sick babies are transferred for specialist care than any other group of intensive care patients (Major 1996), and their delivery and retrieval fits into several categories:

- Transferring patients to the more appropriate facilities of another hospital for specialist management.
- Retrieving patients from peripheral hospitals for intensive care. Previous research indicated that babies in district hospitals were twice as likely to die as those in teaching hospitals; transferring them to a specialist unit led to lower mortality and improved outcome (Lyall 1993). However, studies by CRIB investigators have not confirmed this (International Neonatal Network 1993).
- Intra-hospital transport—transferring patients from the delivery ward to the NICU, and to and from the operating theatre.
- Back transport to the referring hospital for convalescence—by which time intensive care is not required.

The transport team

Although there may be risks for both mother and baby, there is no doubt that it is easier, and generally safer for the infant, to be moved in utero. Careful discussion between the obstetric and neonatal teams should precede any decision to transfer, however, as unforeseen problems can still complicate the journey. If there is a history of antepartum haemorrhage, severe pre-eclampsia or the risk of impending delivery, in utero transfer should not take place (Field 1999).

The majority of neonatal transfers are attended by a doctor and a nurse, with perhaps the addition of a neonatal intensivist, a paramedic, or a technician, but

Leslie (1997a) suggests there are wide local variations in the composition of the team, and the grade, experience and training of these personnel.

The Transport of Neonates in Ambulances (TINA) report (Medical Devices Agency 1995) makes recommendations regarding the organisation and conduct of neonatal transfers. The document makes it clear that personnel should have adequate training and experience, with frequent exposure to the transportation of critically ill infants to maintain their expertise. It is not acceptable to send staff on transfers so infrequently that skills are lost. If the number of transfers is erratic, it may not be cost-effective for each area to have a central interhospital retrieval team. This raises the question, should transport teams be based geographically rather than each hospital having specifically trained staff? ECMO centres have their own highly skilled retrieval teams (Field *et al.* 1996), as do a few areas in the United Kingdom. Neonatal nurses need wide-ranging skills, and a transport nurse unable to use those skills full time is probably not cost-effective. A team from rostered staff should, however, be trained with specific transport skills, rather than training 'on the job', as part of their extended role. Eligible staff should be available 24 hours a day and able to mobilise immediately.

Training should include an action plan of what to do in specific emergencies, as this may differ from similar situations in the NICU. Staff should be capable of providing advanced life support—including airway and ventilator management, vascular access and the emergency relief of pneumothorax at the referring hospital, and be able to maintain that level of support during transportation back to the receiving hospital. The level of intervention depends on the nature and the severity of the problem, the facilities available, as well as the distance from the NICU (Leslie 1997a). All personnel should also be well versed in the use of the equipment, the commonest causes of its malfunction (Bourchier 1994), and able to 'troubleshoot'.

Intra-hospital transport from the delivery suite to the NICU, and to and from the operating theatre, presents comparable risks and equally demands the attendance of suitably experienced personnel. A study by Wallen *et al.* (1995) of 180 intra-hospital transfers found that there was a significant change in at least one physiological variable in 71.7 per cent patients transferred to and from the intensive care unit. There was one equipment-related mishap in 10 per cent of transfers, and at least one major intervention was performed in 13.9 per cent of transfers in response to physiological deterioration (34.4 per cent were ventilated), or equipment-related mishap. Although a paediatric study, these findings could be extrapolated to the advantage of the neonatal population.

Occasionally an infant will be well enough to be transported by a lone nurse. However, this will only be possible if she has the competence, confidence and the ability to problem-solve even if the infant is deemed 'stable'. These circumstances include a positively diagnosed, neurologically intact infant, with essentially normal laboratory indices and vital signs, a patent airway and minimal oxygen requirement; and with intravenous maintenance fluids and medication (e.g. antibiotics) only.

Transport vehicles

Infant transportation existed in the 1950s but it bore little resemblance to contemporary highly organised technological methods. Travel is usually by road. Conventional transport incubators are heavy, contravening manual lifting regulations, and until recently they were inadequately secured. This was highlighted by the media following a road traffic accident (Madar and Milligan 1994), in which the ambulance rolled over and the incubator and fixings tore away from the wall-mounted anchoring. The infant fortunately survived, but serious injury was caused to one of the medical team. The TINA report recommends that 'incubators should be secured effectively within the ambulance'. A traditional incubator on a trolley weighs 130–200 kg, but newer, lighter (100 kg) incubators with collapsible trolleys are being developed (Leslie 1997b). Specially designed ambulances for the transfer of neonatal intensive care patients are now available, with purpose-built anchor points and flexible fixing methods, as well as a simple two-person loading system (EAA NHS Trust 1996).

An ambulance has the advantages of rapid mobilisation, the ability to travel in most weather conditions, it can be easily diverted, or can stop if procedures need to be carried out within a relatively large working area. It has an urban speed of approximately 15 mph, and a rural speed of 60 mph. Disadvantages of ground transport are the longer transit times and traffic-related delays.

In most areas, air transport is used on special occasions only. A fixed-wing aircraft has the advantages of rapid transport over long distances (300 mph), and can fly over or round bad weather. However, flying is expensive, there is a lack of appropriate landing sites near most hospitals, so an ambulance must be arranged at both ends to move the baby between the airport and the hospital. Helicopter transfer has the advantages of rapid mobilisation and transport time (150 mph), and can reach otherwise inaccessible areas. Its disadvantages are the need for an unobstructed landing site (unless the hospital has a helipad), the small, noisy, vibrating cabin space, where it is difficult to perform procedures, and weight limitation. Flying can also be limited by inclement weather.

Hazards of transportation

The infant's life is at risk during the journey, but with care, sophisticated monitoring equipment and the skills of the team, the risk can be minimised (Cooke 1992).

Caring for a critically ill infant during a journey is very different from caring for a baby in an intensive care unit where more equipment and staff are available; it is daunting and fraught with environmental hindrances. A doctor and a nurse are often the only personnel present, and the transport environment is even more challenging than the NICU, with excessive noise, mechanical vibration and instability of equipment, restricted lighting, limited work space and support services. The back of an ambulance or an aircraft are highly unsuitable places in

which to perform resuscitative procedures, with small infants becoming cold very quickly. Although team members must be trained to respond appropriately to such situations, pre-empting potential problems, prevention in the first instance is preferable.

Meticulous attention to detail in the preparation of the patient and equipment should minimise the possibility of equipment problems and clinical instability during transport. Outcome depends on how well the transport team prepares before leaving the referring hospital. The incidence of iatrogenic and secondary insults, occurring during transport, is related to the level of training of the staff (Macrae 1994). Categories of avoidable insult include failure to resuscitate and stabilise shocked infants adequately, failure to assess and manage the airway, which Macrae suggests is probably the most common cause of morbidity associated with transport, and failure to treat discrete changes in vital signs. Clinical monitoring during transport is clearly important and must be comprehensive with reliable electronic machines. Auscultation is difficult in moving vehicles and audible alarms may be undetected against background noise.

Equipment can fail and drugs and oxygen supplies can become exhausted. It is recommended to carry twice as much oxygen as the estimated journey time requires, to cover delays and possible breakdown. Most transport cylinders contain 600 litres/2000 PSI and the quantity required can be calculated by a simple formula (taken from ALSG 1997):

$$\text{minutes of oxygen available} = \frac{\text{PSI} \times 0.3}{\text{flow in litres per minute}}$$

Altitude also has an effect on the patient's gas exchange. Air is largely composed of oxygen and nitrogen. Dalton's law declares that the total pressure of a gas is the sum of all the individual (partial) pressures of all the gases in a gas mixture. Barometric pressure is the sum of the partial pressures of individual gases in the atmosphere. The partial pressure of oxygen in the atmosphere (PO_2) is equal to the concentration of O_2 in the atmosphere (21%) multiplied by the barometric pressure. At sea level, the barometric pressure is 760 mmHg, and at 10000 feet it is 523 mmHg. At sea level, the PO_2 in the air is 160 mmHg (0.21×760) and at 10000 feet it is 110 mmHg (0.21×523) (Aoki and McCloskey 1992).

With increasing altitude, there is a fall in the ambient oxygen tension and an inverse relationship between altitude and barometric pressure (Bourchier 1994). If an infant requires an FiO_2 of 40% (0.40) at ground level to maintain an adequate physiological PO_2, a simple equation can determine the required increased FiO_2 at 10000 feet (taken from Aoki and McCloskey 1992):

$$\frac{\text{oxygen requirement at sea level} \times \text{barometric pressure at sea level}}{\text{barometric pressure at actual altitude}}$$

$$\frac{0.4 \times 760}{523} = 0.58\ (58\%)$$

As air pressure falls, there is an associated expansion of gas within body compartments. Hence all infants undergoing air transport must have a widebore nasogastric tube on free drainage, which is aspirated frequently. At particular risk of deterioration are those infants with a diaphragmatic hernia (see p. 358), as bowel gas within the thorax expands. Patients with a pneumothorax should have a chest drain inserted with a Heimlich valve attached before departure (Bourchier 1994).

Whilst the infant's well-being is paramount, the transport team need consideration too. Altitude can have an adverse effect on them, and the ability to stay alert, concentrate and make rapid decisions can be impaired by vibration and noise. Cabin pressurisation reduces these effects by maintaining a pre-set barometric pressure regardless of the actual altitude (Aoki and McCloskey 1992). Motion sickness may also be a particular problem for personnel, and appropriate medication should be taken by those affected prior to departure.

Equipment required for transporting a sick neonate

Reliable equipment to initiate and subsequently manage intensive care must be designed to work in difficult environments, and be commensurate with the severity of the neonate's illness and the anticipated duration of transport (Wallen *et al.* 1995). It should be adaptable for babies with a variety of problems and gestations.

The dual power-sourced transport incubator should provide an enclosed, warm, illuminated environment for the infant and a ventilator suitable for use in a bumpy vehicle. There should be easy access for observation, monitoring and fluids. Associated instrumentation to provide invasive and non-invasive parameters must be anchored to the incubator. Monitors with long-life internal batteries that are rugged, compact, lightweight and operable in all types of vehicle are required. Appropriate electrodes, probes and leads should be available to give readings of heart and respiratory rates, temperature, oxygen saturation and invasive and non-invasive blood pressure. Battery-powered syringe drivers capable of operating syringes of up to 60ml capacity and performing accurately at all infusion rates, apparatus for bag-and-mask-ventilation, and a stethoscope should be readily accessible.

Other equipment should be packed into a robust bag that is also anchored firmly to the incubator. Figure 16.1 shows the suggested contents of a transport bag.

Recording charts and maps should also be available. A mobile phone will provide immediate contact between both hospitals and the transport team. Relying on a third party's communication network (i.e. the ambulance service) to pass on messages about clinical status is not satisfactory (Macrae 1994).

It is essential that all apparatus is checked by the team before and after each journey, and is regularly maintained and serviced by the hospital's medical engineering department.

Intubation equipment	**IV access equipment**	**Arterial access equipment**	**Drugs**
laryngoscopes ET tubes *introducers* ETT fixatives/hats airways batteries/bulbs	cannulae taps, T-pieces, bungs *syringes, needles* splints, cotton wool lancets intraosseous needles sodium chloride	umbilical catheters transducer set *umbilical tape* blades, scalpels sutures Kaltostat	adrenaline atropine *caffeine* diazemuls frusemide heparin hydrocortisone Konakion midazolam naloxone pancuronium sodium bicarbonate tolazoline vecuronium colloid normal saline water for injection
Relief-of-pneumothorax equipment	**Fluids**	**Miscellaneous**	
butterfly needles sterile water syringes/needles trocars antiseptic gauze, Bioclusive universal container Heimlich valves	heparinised saline 10% dextrose 0.9% saline 50% glucose giving sets lectro-spiral tubing ramps spikes labels syringes, needles	sterets, steristrips gloves feeding tubes mucous extractor cord clamps identity bands thermometer elastoplast, etc. paperwork BM stix	

Figure 16.1 **Suggested contents of a transport bag**

At the referral unit

Joint management collaboration by the referring hospital and the transport team should commence immediately since successful resuscitation and stabilisation is crucial to ultimate outcome.

In most areas the transport of newborn infants is co-ordinated through the referral centre, some having a telephone hotline, and at this time the degree of urgency required must be ascertained together with clinical details—name, age, gestation, birthweight, provisional diagnosis, clinical status and current management. Based on this information, which is ideally recorded on a proforma unique to the unit, interim management can be advised until the transport team arrives. This advice should include ventilatory and fluid management, initiation of medications and the obtaining of maternal blood to assist with cross-matching at the referral centre. Before leaving, the infant's specific needs should be considered. Any additional equipment required can be added to the transport bag, and medication doses can be calculated in advance. The ambulance should be available to the team as soon as they are ready to leave.

At the referring unit

The transport team should ensure that they introduce themselves to the staff and parents on arrival. A résumé of change in clinical status subsequent to the initial call should be obtained prior to assessment of the infant. Failure to address the way in which the handover is paced and integrated with the resident team's care is likely to cause hostility (Leslie and Middleton 1995), and derogate their clinical effectiveness. It is, after all, currently 'their baby'.

The infant must be stabilised prior to returning to base, and this may take several hours if the infant is particularly unstable. It is not usually necessary to leave the unit before vital signs are optimised. Research has shown that the presence of hypothermia, hypotension and acidosis before transport has significant negative outcomes and should be prevented at all costs (Major 1996). As these cannot be provided en route, it is essential that all necessary investigations are completed before transfer (Macrae 1994). It is therefore necessary to complete a full set of observations and to normalise blood gases, temperature, blood pressure and blood sugar. Vibration and noise stressors experienced during transport can exacerbate hypoglycaemia (Aoki and McCloskey 1992). Diagnostic X-rays and haematological and biochemical results carried out by the referring hospital should be checked, documented and if necessary repeated.

If the infant is not ventilated on the team's arrival, it is probably prudent to secure an airway prior to the journey if there is any suggestion of respiratory difficulty. Hand-bagging, in any form of moving transport, is inconsistent, erratic and potentially ineffective, whilst increasing the risk of dislodging the tube. It also unnecessarily restricts the activities of one of the team, whilst potentially generating thermal instability. The position of the endotracheal tube must be checked radiologically before departure. Appropriate sedatives and muscle relaxants may be necessary, for comfort and safety, whilst reducing the chance of accidental extubation. The patency and security of lines should be checked, as should current drugs and dosages. At least two secure venous access lines are essential for maintenance fluids, resuscitation and drug administration during the journey. Haemodynamically unstable neonates should have invasive blood pressure monitoring.

The infant should be transferred to the transport incubator only when stabilisation is complete and the move should be punctilious. Three people are required for the move: one for the baby, one to manipulate ventilator tubing, chest drains, fluid lines, etc., and one to open and close the incubator (Leslie 1997b). Once the infant is in the transport incubator, air entry and other lines should be rechecked, and when the team are satisfied that the infant is stable in the vehicle, with monitors and equipment working and visible, the team will be ready to leave.

The receiving unit should be telephoned prior to departure with an update of clinical status and the estimated time of arrival.

The parents

One of the transport team should allow time to talk to the parents, collecting relevant information about the infant and his or her family, and maternal blood for cross-matching if surgery is anticipated. Current guidelines indicate that only the surgeon performing the operation should obtain consent, and this can create a problem unless the parents are able to accompany their neonate. Consent by telephone, although not ideal, may be the only option.

Good communication skills are vital in a successful transport team, especially where the parents are concerned. Dodds-Azzopardi and Chapman (1995) found that parental stress was related to lack of communication between parents and health professionals regarding the infant's transfer, as well as to differences in care practice. Parents are already suffering acute emotional problems associated with the birth of their ill baby, and the fact that the baby has to be moved to a specialist unit is an indication of the severity of the condition. They are often present on the unit at the time that the team is stabilising the baby for transfer (Leslie and Middleton 1995), and staff should appreciate the extra anxiety brought about by the transfer (Alfonso *et al.* 1992). According to the ALSG (1997), if clear explanations are given, their anxiety will be lessened and their cooperation will be increased.

If the mother is newly delivered in one hospital and the baby is critically ill in another, the father faces visiting dilemmas. When the mother recovers, she may not be able to spend much time with her ill baby, especially if she has other children some distance away (Wilman 1997). Redshaw *et al.* (1996) found that the NICU experience represents a psychosocial crisis for parents, and support services are often inadequate. Social support is widely documented as an effective coping strategy, especially in relation to parents in the NICU (McHaffie 1992; Prudhoe and Peters 1995). Stressors are magnified when the baby is transferred out and the family is disunited. If the mother is moved near the baby she may well be estranged from insular support by 'select close persons' (Bose 1989; Coffman *et al.* 1993).

It is essential that parents have a telephone number for the base unit as well as directions for getting there. They might conceivably insist on accompanying the team, but the needs of the patient take precedence. There is limited space available in any vehicle, and the team would be unable to offer them adequate support, should any emergency intervention be necessary to the infant during the transfer to the receiving hospital (Melville and Print 1996). If they decide to follow the ambulance by car, they should be reminded that, unlike emergency vehicle drivers, they are not permitted to disregard certain traffic regulations.

The return journey

The optimum way for the vehicle to proceed needs to be determined, according to the infant's condition. A 'blue light' return might be justified in some cases, when speed limits may be exceeded if it is safe to do so, and red traffic lights

may be regarded as 'Give Way' signs. In exceptional circumstances a police escort might be requested to clear a path through heavy urban traffic. Slow and steady may be better, but even stable ventilated patients should not be stuck in traffic.

During the transport vital signs and oxygenation, fluid rate, gas consumption and incubator temperature should be monitored regularly. If the patient has been properly prepared for transfer, few procedures other than monitoring will be required, even over long distances.

All procedures should be documented. While no definitive documentation is legally obligatory, the *Standards for Records and Record Keeping* (UKCC 1993) expects accurate records of all intervention and management strategies, together with the names of relevant personnel.

Back at the referral unit

The infant should be transferred immediately to the NICU and reassessed. The work of the transport team is not complete until the patient has been safely transferred to static equipment, with a comprehensive handover being mandatory (Leslie 1997b). A chest X-ray to determine tube displacement, and repeat blood gases, biochemical and haematological assays are also mandatory following transportation of an intensive care infant.

The referring unit and parents should be contacted, and all equipment should be cleaned, checked and replenished ready for the next emergency.

Legalities involved with transporting a sick neonate

Transport organisation and management must be in the best interest of the patient, but can give rise to legal issues concerning the referring hospital, the transport team and the receiving hospital. Legal responsibility for the transported patient therefore represents a continuum (Melville and Print 1996). The receiving hospital's responsibility begins when the referring hospital makes the phone call and the patient is accepted. Their liability increases substantially once the patient is accepted for admission. The referring hospital's liability diminishes in proportion to the involvement of the receiving hospital, once that initial phone call has been made and the team issues management instructions (Aoki and McCloskey 1992). The unique variables of emergency transport demand that the team consider their complex legal accountability in the context of transport. Although no regulations apply directly to intensive care transport in the United Kingdom, there is no substitute for professional accountability and responsibility. The UKCC (1992) has delineated maxims for accommodating the scope of professional practice, which includes 'acknowledging limitations of knowledge, skills and competence'. If harm occurs to the patient due to lack of skill, knowledge or competence of a transport nurse, that nurse will be liable, and the nurse's employer vicariously liable (Melville and Print 1996).

Conclusion

The goal of an effective transport team is to provide timely and safe transport, so that definitive care can be provided for the neonate at the receiving hospital. The basic principles of good transport should be applied to all sick patients moved within or between hospitals, whether or not a specialist team is involved. Conscientious initial assessment, resuscitation and felicitous emergency management should reduce the chance of transport-related morbidity and mortality (ALSG 1997). Effective communication between the referring hospital, the NICU and the team has also long been identified as an essential element to providing optimal patient care during neonatal transport (Finterswald 1998), and is a medicolegal necessity (Melville and Print 1996). As Leslie and Middleton (1995) conclude, 'transfer is more than the movement of the baby and associated information, it involves the meshing together of two networks of care'.

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Chapter 17

Family Support

Contents

Introduction

Budin established the importance of parents in relation to the sick or premature newborn infant in 1907. Almost a century later, supporting parents in their essential role remains a complex issue for nurses and is becoming increasingly so as parents and their expectations are rapidly changing. This issue, for the purposes of this chapter, is referred to as 'family support' whereas others have called it 'family-centred care'. The difference is one of terminology alone.

This chapter will:

- Define family support
- Explore the concept of family support/family-centred care in the light of current literature
- Discuss the main components of family support
- Consider strategies for providing family support within the neonatal setting
- Suggest some basic principles of family support

Family support—a definition

Forming emotion bonds and attachments with a premature or sick infant can be extremely difficult for some parents. It is for the nurse caring for that infant to offer family support that will assist this important process. Enabling parents to care for, take responsibility for, and gain knowledge about their sick and preterm infant is the very essence of family support. No one can deny, however, that the work of supporting families is demanding and can leave nurses exhausted and dispirited. How often has it been said, 'If only the parents would leave us to get on with caring for the infants'? Sometimes it seems like family support is one of the lowest priorities in the neonatal setting. Nevertheless, family support is intrinsic to the nursing of infants. Nurses need to find a way to make it their personal priority if they are to deliver a quality service to every family.

Twenty-five years ago neonatal nurses cared for what we would now call special care infants and excluded their parents. The situation today is one of highly technical intensive care performed in the presence of unlimited visiting by parents and anyone who is important to them. This situation calls for a new model that 'refines the existing model by attending to the psychosocial needs of infants and their parents' (Wyly and Allen 1991).

Family-centred care

The concept of family-centred care has been extensively covered in neonatal and paediatric nursing literature (Nethercott 1993; Darbyshire 1995; Taylor 1996). However, it has been suggested that 'many people from management to bedside staff, are still not sure what it means' (Gregory 1992). Most

authors agree that family-centred care concerns parents' relationship with their infant, their participation in their infant's care and any decisions that are made about their infant, and parents' relationship with the health care provider, that relationship being more equal than previously seen. Family-centred care emphasises a shift from the family as passive recipient to the family as active participant in their infant's care. It represents a holistic approach to care. Enabling and empowering families, it elevates parents' position on the neonatal unit to that of equal partners with the health care professional.

Providing family care is not about whether the appropriate resources and facilities for parents are available, neither is it about whether they are allowed to perform parenting tasks on their infants or not. It is about the ability of nurses to provide family nursing, simply to care and support the family of a sick or preterm infant.

The Welfare of Sick Children and Young People in Hospital guidance in 1991 divided family-centred care into three main concepts:

- an approach to nursing which regards the unit of care as being the family and not just the patient;
- an acceptance of the family's own definition of what constitutes 'family', when nurses may not have concrete or appropriate definitions of the nature of the family;
- when the nursing care and environment help to promote the strengths and individuality of the family in order to enable them greater scope for caring for their relative.

Doubts have been raised as to whether family-centred care in the neonatal unit is achievable (Beresford 1997). Others have considered that 'practice does not always meet the ideal and there is little guidance on how nurses should shift their focus from the child to the family' (Whyte 1996).

The components of family support

There are three main components in the provision of family support within the neonatal setting:

- The caregiver group—neonatal nurses.
- The recipient group—the infants, the parents and the siblings.
- The neonatal environment.

The caregiver group

Few nurses come to neonatology to care for parents; their reasons for working in the area are usually about caring for sick infants and working with intensive technology. Griffin (1998) describes nurses as directing their energies towards their patients' physical needs. She notes that

physical care may temporarily take priority over the family's needs. Other authors comment that neonatal nurses are 'generally focused on the technical components of clinical care' (Haut *et al.* 1994). The driving force within many neonatal units is one of physiological survival. Family concerns and parent-infant bonding are usually low in priority in comparison.

Study days and post-basic training are tailored towards providing the skills that are needed to work on a busy neonatal unit. Education that provides the skills that neonatal nurses need to support families is more difficult to find, yet Haut *et al.* point out that 'neonatal nurses are often the front line managers and co-ordinators of family care in the NICU'. They further argue that neonatal nurses 'are charged with the challenge of understanding and providing "state of the art" technological care in an environment that must also adapt to the ever changing needs of parents and families who cannot be considered visitors, but an integral part of their infant's care and survival'. Unless there is commitment to family support from the highest level, nurses may be set up to fail as they battle to fulfil everything that is asked of them.

The recipient group—the infants and their families

One in every ten infants born in the UK each year is admitted to a neonatal unit. Over half of these infants require admission because they have been born prematurely (Moore 1995). When infants are born early or sick the whole family is thrown into crisis. The parents may need to go through a grieving process of giving up the blueprint infant they had imagined in order to accept the infant in front of them. Parents may take personally the infant's prematurity or sickness, blaming themselves and believing that the difficulties reflect on their abilities to care for their infant (Perehudoff 1990; Miles *et al.* 1991; Affonso *et al.* 1992).

Each neonatal unit will have its own particular mix of families. Nurses need to understand the cultural differences, lifestyle and background of the parents (Dawson 1994). Finding out the differences between those families and targeting those parents with the most acute needs will help to overcome barriers to successful family support. The most vulnerable families may not have the sickest infants; members of staff may have to become advocates for these parents as their infant's condition may not make them a priority within the neonatal service. Parents are individuals and for each family the experience of having an infant on the neonatal unit or special care infant unit is different (Redshaw *et al.* 1993).

Parents are not a homogenous group. In the past, groups of parents may have been categorised under their infants' gestation or diagnosis, considering that all parents of the 25-week gestation infant would have similar feelings and needs. This is not necessarily the best way to predict parental needs. Nurses need to look closely at the cultural and social backgrounds of the parents to provide adequate support for the whole family.

Family support must encompass the whole family, including siblings. The birth of a preterm or sick infant is a time of crisis for all members of a family and the needs of the siblings may be overlooked, yet it is vital to the child's future health and well-being that the family continue to function as a cohesive unit (Smith 1997).

The sibling relationship is perhaps the most long-lasting and most influential relationship of a person's life; it begins with the birth of a brother or sister and continues throughout one's lifetime. Encouraging parents to include the siblings in activities surrounding the ill newborn sibling can help them come to terms with this new situation. Resources such as age-appropriate stories books, colouring books and play activities can assist this process. Siblings need to get to know the new family member. This is a normal and necessary step in becoming a family. A survey undertaken in 1997 shows the vast majority (92 per cent) of neonatal units in the UK allow open visiting for siblings (Smith 1997). Parents should feel welcome to bring their other children to the neonatal unit when they wish but bringing small children to a busy high-dependency unit is not without its problems. It may be necessary in the early days for them to arrange for a relative or friend to visit with the family; this person can concentrate on caring for the siblings, thus allowing the parents to give their full attention to their sick infant. Arrangements could be made for siblings to stay with relatives or friends for short periods while the parents visit their infant. If there is no one to care for the other children, the neonatal unit staff should make a case to the local Social Services department that the sibling is a 'Child in Need'. In these cases temporary, short-term placements at local family centres or day care schemes may be available. Nurses know that it is not easy for parents to cope with the needs of their new baby and those of their other children.

As the infant moves through the unit, visits from brothers and sisters should become more frequent. Neonatal units can be trying places for young children, and nurses should advise parents to bring favourite toys and snacks as boredom and hunger can make the situation more difficult for the siblings. Almost all units have some play facilities but these vary greatly from a small box of toys within the nursery to a separate playroom with play leader supervision.

The neonatal environment

The neonatal environment can be a stressful and frequently frightening experience for parents (Benfield *et al.* 1976; Miles *et al.* 1991; Affonso *et al.* 1992; Miles *et al.* 1992; Gennaro *et al.* 1993). Dawson (1994) cites Bender and Swann, whose research indicated that neonatal units are perceived by parents as technologically formidable and threatening places that are pervaded by an atmosphere of urgency and crisis—literally one of life and death. Bender reminds us that it is in this strange, noisy, anxiety-provoking atmosphere, usually devoid of privacy, where parents must struggle to establish and maintain the bonds of affection with their vulnerable infants.

Traditionally, parents have not been the driving force in the development and acceptance of neonatal intensive care. The present system of neonatal care is conducted in an environment established on the expectation that high-technology care can save and cure even the tiniest and sickest patients (Raines 1996).

Strategies for providing family support in the neonatal setting

Supporting the family in the antenatal period

In recent years, it has become apparent that families with high-risk pregnancies need support in the antenatal period. Advances in ultrasound scanning have redefined early identification of the high-risk pregnancy. Antenatal diagnosis has assisted in the early preparation of some parents for the neonatal environment. Parents of infants identified in the antenatal period as potential neonatal admissions should be offered a preparatory visit to the neonatal service. Good communication between paediatrician and obstetrician, neonatal nurse and midwife is essential if parents are to be given adequate antenatal preparation.

However, the work undertaken by Redshaw *et al.* in 1995 indicated that antenatal visits to the neonatal unit are not routine. Although 58 per cent of units had a policy in operation, only 20 per cent of mothers in the study had seen the unit before their infant was born.

Nurses need to work closely with midwives to identify the physical health status of the mother. Many mothers have had a complicated obstetric course and may be feeling far from well on their first visit to the neonatal unit. Both mother and father may feel a lack of emotional support; they are in an emotionally labile state—one minute in tears, one minute ecstatically happy. They may appear to lack motivation, while in reality they may be stunned into inactivity by the enormity of the task (Miles 1989; Perehudoff 1990; Affonso *et al.* 1992).

Supporting the family during the admission period

Even the prepared parent will experience shock and grief during the admission of their infant. Parents may be so overwhelmed by events that initially they may adopt a passive role and concentrate on familiar routines that help them accommodate this new dramatic situation in their lives (Redshaw *et al.* 1993).

Parents may experience a range of negative emotions, including grief, anxiety, fear, helplessness, denial, shame, shock and denial. Fear for the future and for the infant's future and long-term outcomes is common. Parents will look to the admitting nurse to recognise and acknowledge how they are feeling and will need to be offered strategies to enable them to get through this stressful period (Lindsay *et al.* 1993). It is at this time of crisis, when the parents have

greatest need for information, that there is the greatest chance of them not being able to absorb the volume of information that they are given (Gennaro *et al.* 1990). However, 'being emotionally troubled does not make parents unintelligent, nor prevent them from using the intelligence they have' (Taylor 1996).

Our communication with parents at this time of crisis must be basic and as reassuring as possible, with clear written information in a language that they can understand. If information is repeated and reinforced on a regular basis, the opportunity for effective communication, where the parent retains and understands the information, is maximised (Taylor 1996). Where English is not the first language of either parent, an interpreter must be used.

Many parents feel frightened and anxious during this period (Affonso *et al.* 1992). It is in this tense atmosphere, with people who are stressed and exhausted, often at the beginning of an emotional roller-coaster, that neonatal unit staff have to begin the crucial process of care and support (Redshaw *et al.* 1993).

During admission, the medical and nursing needs of the infant often outweigh the psychosocial needs of the parents. It is equally important to find time for the parents. Nurses often decide when they want to speak to parents, but this does not necessarily fit in with the parents' needs for information. Where verbal information is limited, it is important to offer alternatives, such as parent information booklets or videos.

Most neonatal units record the infant's arrival on the neonatal unit with Polaroid photographs, which are given to the father to share with the mother if she is not able to come to see her infant immediately. A warm friendly approach to parents will help allay some fears by making them feel welcome. Staff should introduce themselves and colleagues, giving parents time to ask questions and adjust to their altered role.

There is usually a short period of time when one or more parent is resident on the maternity ward and the infant is in the neonatal unit. This period can often be fraught with communication problems and misunderstandings. It is not good practice to use the parent as a messenger between neonatal nurse and midwife. The nurse caring for the infant can achieve proactive management of the potential problems by communicating with the midwife caring for the parent(s) daily.

While parents experiencing the emotional turmoil associated with a preterm birth must try to some extent to understand the clinical approach and the high-tech environment of the NICU (Wyly and Allen 1991), nurses must acknowledge and address their psychosocial needs.

Processes or phases that typify a parent's journey though the neonatal unit have been identified (Darbyshire 1994; Beresford 1998). Darbyshire described them as:

- Naive trusting: in this phase the parents' needs are for information, reassurance, security and support from isolation.
- Disenchantment: in this phase the parents voice concern about the care their infant is getting.

- Guarded alliance: parents work with nurses to achieve a given goal. The phase usually coincides with discharge.

Beresford (1998) describes a definite, psychological process which helps parents adapt to having an infant on the neonatal unit. She also identifies three distinct phases as the crisis phase, notably the impact of the technology and how it affects professional-parental relationships, followed by the transitional period, focusing on the parental determination to return to normality, and finally the necessary adjustment to normal life.

Once the question of the infant's survival appears resolved, nurses may see a change in the attitudes of the parents. Conflict may arise when parents challenge nursing actions and unit policies and procedures. The parents may be trying to regain ownership and control of their infant and their own lives. Nowadays, infants move quickly through the neonatal unit; there is less time than ever for parents to go through these psychological processes. Nurses must learn the skills to identify the parents' position in the psychological continuum.

Being aware of the profound effects of separation

Separation is undoubtedly the worst feature of having an infant in the neonatal unit. It prevents a mother from getting to know her baby and often threatens her perceptions of her ability to become a 'good mother' to her child (Affonso *et al.* 1992).

Separation has been shown to result in emotional, psychological and developmental delays in the infant (Elsas 1981). Infants should not be separated from their parents unless their survival depends upon it. Neonatal units must adopt strategies that reduce separation:

- By ensuring that the infant is in the right place at all times, nurses can hasten the time that the infant can be reunited with the parent. The right place may be in a tertiary neonatal unit with the parents in unit accommodation, or with unlimited visiting and travel cost reimbursement; a special care unit near to home; or a postnatal ward receiving transitional care from parent and midwife.
- Daily assessment of how parent and infant can be reunited is essential to the long-term development of their relationship.
- Visitation policies that reflect the need of the parents to have unlimited access to their infant are the norm in most neonatal units.
- Dedicated phone lines for the parents' use only allow them to speak directly to the nurse caring for their infant.

Parents of ill neonates have the right to be close to their infants at critical periods in the course of their treatment (Bowlby and Robertson 1955; NAWCH 1985). Neonatal units should provide or arrange accommodation for all parents. However, that provision remains a challenge for many neonatal units. Even

where funding has been made available and several rooms are on offer there are times when demand outstrips supply and difficult decisions need to be made. It is virtually impossible to prioritise the needs of parents or to identify whose needs are the greatest.

Separation may not be merely geographical; it may be physical or emotional. As soon as the infant's condition allows, every effort must be made for infant and parent to be physically and emotionally reunited. Facilitating contact and involving parents in care is an important method of overcoming the emotional problems that can arise following separation (Redshaw *et al.* 1993). Nurses need to be aware, however, of the stress that can be generated from their attempts to promote contact between mother and infant.

Many new parents are not prepared for the conflict between their own needs and their infant's needs. There may be siblings or other relatives who need care and new parents are often faced with enormous practical problems with travel and child care. Travel costs may become a huge problem for some parents.

Supporting the family during the discharge preparation period

Most nurses are familiar with the slogan 'discharge planning begins on admission'. It is a difficult concept to accept when the infant's survival is in question. Yet, increasingly, on neonatal units the time between admission and discharge is becoming shorter, and often before nurse or parent anticipated, the discharge date is fixed. Once survival is guaranteed the parents' one objective is to take their baby out of the neonatal unit. Everything they learn is in order to achieve that one objective.

Discharge planning or preparing parents to take their infant out of the neonatal unit should begin as soon as survival is guaranteed. It is much better to prepare gradually over the whole length of time rather than there be a flurry of information and activity in the 24 hours prior to discharge. However, if adequate preparation for discharge is to occur, then nurses must become experts at predicting lengths of stay in the neonatal unit.

Traditionally parents were counselled at the time of admission to the NICU that their baby's discharge would probably occur around the original due date. This estimate was usually too long by several weeks (Vecchi *et al.* 1996, citing Rawlings *et al.*). Most preterm infants are being discharged at around 35 weeks' corrected age, five weeks before the EDD. Parents have less time to prepare than they anticipated; for some this is seen as a good sign that their baby has 'done better' than the neonatal team thought he or she would, while others are thrown into a panic.

One study of parents in a neonatal unit showed that despite efforts to address the concerns of parents taking their children home, there was still a large group of parents (24 per cent) who did not feel properly prepared for discharge (Curran *et al.* 1997).

Supporting the family of the dying infant

All parents expect their pregnancy to end in the birth of a live, healthy infant. Going home without an infant from whatever cause is almost unbearable. The sudden unexpected death of an infant is one of the most distressing events that can happen to any family (FSIDS 1997). From the moment the death of the infant is known, the parents are thrown into shock and turmoil. They look to their caregivers for help and advice (Cooper 1996). If an infant's health begins to worsen and death appears imminent, the consultant and the nurse caring for the infant should speak to the parents. Parents need information about their infant's condition and what will happen in the next few hours; they should be encouraged to ask as many questions as they want at this time.

Parents may feel very alone at this time, but they may be surrounded by others who feel as they do. Grandparents, older children and close friends and relatives will grieve with them.

If the parents have not yet done so, it may help if the nurse suggests that they give the baby a name. Naming the infant will help them and others know that the infant was born and died, even though its life was very short. Most hospitals have a chapel and a chaplaincy team. Many have multi-faith centres. Chaplaincy teams are available to help parents through this difficult time and to have their infant blessed or baptised if they wish. Sometimes it is possible for parents to spend time alone with their baby before he or she dies. If this is the case, the nurse should facilitate the provision of appropriate accommodation where the parents can have privacy and time to be with their child. This may be in the parents' home.

The nurse caring for the infant should remove all tubes (according to each neonatal unit's bereavement policy), dress the infant and offer the infant to the parents to hold. Parents can use cameras or a video camera to record these moments.

Parents should be given written information and guidance on how to arrange their infant's funeral.

Nurses have a special role in helping the parents to collect memories of their infant. Locks of hair, Polaroid photographs, footprints and other things that belonged to the infant should be offered to the parent. If they do not want them immediately the items should be stored with the medical notes until they do. As Polaroid photographs fade, the hospital photographer should be asked to take photographs of the infant, which can be given to the parents at the bereavement appointment. This is when the neonatal consultant sees bereaved parents again to answer any questions that they may have and to discuss the infant's death.

If the infant's death was due to a possible genetic or inherited factor, the parents should be offered an appointment at the genetic counselling clinic. Nurses should be able to talk to bereaved parents about their infant's short life.

Many hospitals now have a Babies' Book of Remembrance, which is kept in the hospital chapel. Each page is dedicated to a infant who has died. Parents can write their own special message for their infant to go in this book. Each year hospital chapels hold services in memory of the infants who have died. The neonatal unit invites parents to attend for as long as they wish. Working with bereaved parents is an emotionally demanding and stressful task. All staff involved in their care should be linked into adequate training and personal support systems. Nurses should know where bereaved families could get further help and support within their communities

Basic principles of family support

Each neonatal nurse must develop personal strategies that enable him or her to support parents. Each neonatal unit will have differing service provision, some with large specialist teams that offer programmes of family support, others where the nurse caring for the infant has responsibility for ensuring that the family receives appropriate care. Whatever the situation, whoever provides the care, there are some basic principles that will always apply.

Working in partnership with parents

Enabling and empowering families is at the root of family support (Shelton *et al.* 1987). Acceptance of and respect for the family are key elements of the empowerment model.

It is important for professionals to recognise parents, particularly mothers, as partners in child health care. When professionals are inflexible, judgemental and see their perspectives as invariably superior to those held by mothers, it is impossible to 'work in partnership'. Nurses must have an understanding of the competing demands and the conflicts faced by mothers, and recognising that they do their best and are very responsible people would be a beginning towards real partnership (Wyke and Hewison 1991). Implicit in the word partner is the concept of equality, but there are many types of partners: senior partners, junior partners and indeed sleeping partners. 'Working in partnership with parents' has not been clearly illustrated to nurses. Working as partners means sharing tasks, responsibilities and power.

Good and effective communication is essential for a successful partnership relationship. Effective communication skills are not easily acquired; many nurses find difficulty in talking with parents. Wyly and Allen (1991) explained these difficulties by suggesting that parents and professionals see the world from different perspectives. Professionals are often in a clinical/medical mode *vis-à-vis* the infant, while parents view their infant from a social/emotional state. Despite the difficulties, nurses must continue to communicate and interact with parents.

Parental involvement in the decision-making process

The pace of decision-making at the infant's admission can be frenetic and initially it can be extremely difficult to involve parents. Parents, however, will have to contend with the long-term consequences of any decision made and therefore should have a real voice in making those decisions (Raines 1996 citing Gardner).

If parents are to participate in the decision-making process nurses need to give them the knowledge they need and the range of choices available to them. Nurses need to adopt new skills, which will allow them to establish trust, mutual regard and respect with two-way communication. By the nurses listening to what the parents have to say about their infant, parents can be drawn into the decision-making process.

Nurses should respect parents' ability to make decisions that will affect themselves and their infants based on sound, up-to-date facts. Nurses should be knowledgeable enough to offer facts that will form the basis of parental decision-making. Nurses should give information and not advice and use praise and positive language and actions, to build parents' self-esteem and confidence.

Making contracts, parenting care plans or agreements of cooperation

If nurses are to share care with parents, then each participant must be clear about what is required of them. By sharing knowledge and skills with the parents and demonstrating positive interventions with their infants, nurses can encourage parents to join in the care of their infant. It is not always necessary to teach parents—some will already have the necessary expertise. It is the nurse's role in these cases to recognise and build on this expertise. Darbyshire (1995) found that parental participation was more a set of unexpressed expectations than any form of discussed or articulated agreement. Participation, he found, was limited to what he called 'basic mothering'. For both parents and nurses, this was everyday child care practice. There was a clear distinction in both parents' and nurses' minds between basic mothering and nursing or medical work, although this distinction dissolved somewhat as the child's length of stay increased.

The contract or parenting plan should be clearly documented and kept where all parties can have access to it. It should be revised daily and communicated to all staff caring for the infant. It is essential that the nurse and the parent know their roles and what tasks they are responsible for on a daily basis.

Parents and their situations vary. Each family should be treated individually and the assumption not made that they are willing to give the infant care that could be described as over and above 'basic mothering'.

Walters in 1987 noted that 'There is concern about the tendency to assume that relatives or carers are willing to give increased support after a patient's discharge from hospital. It is imperative that information about carers should

be an intrinsic part of the admission assessment and the process of discharge planning and that plans for carers' assistance are documented.'

Individual family assessment

It is important to have information about families. In recent years, nurses have collected less data about parents, preferring to concentrate on details that relate to the infant and to medical audit. However, many nurses make these assessments in an informal manner, accumulating information over a period of weeks. Formalising this process allows for more accurate predictions of family need and therefore assists discharge planning. The assessment needs to be accurate, thorough and complete and should include a synopsis of the family's social circumstances. Rather than focusing on diagnosis and treatment, nursing knowledge should be used to facilitate the family's ability to understand their health experience and identify their own health concerns (Hartrick *et al.* 1994).

A family assessment should consist of the following:

PHYSICAL ASSESSMENT Establishing the health status of relevant members of the family is important. Maternal ill-health, post-natal depression in particular, will have a considerable effect on the infant and his or her neonatal stay. In families where there are siblings with handicap or illness, considerable stress is placed on the family dynamics.

INTELLECTUAL ASSESSMENT If parents are to become active participants in family-centred care, they must be able to understand the verbal and written information that they receive. All parents come with differing levels of knowledge and it is most helpful to ascertain what previous knowledge the parents have. It is not sufficient to make a judgement from their stated occupation, as the terms 'housewife' or 'unemployed' give little indication of the intellectual abilities of the person. Literacy skills are essential in a neonatal unit, and although it may be difficult to ask if the parent can read, nurses must be aware of this situation and adapt their communication skills to reflect parental ability.

In families where English is not the first language, a careful assessment must be made. Where there is any doubt about the parents' level of understanding of the spoken or written word, an interpreter must be used. It is not acceptable to use another family member.

EMOTIONAL ASSESSMENT Parents will experience a wide range of different emotions—shock, fear, denial and anger to name a few. Nurses should 'understand how these emotional states affect communication and parents' level of functioning in the NICU' (Wyly and Allen 1991).

Neonatal nurses, who are skilled in assessing how the parent is feeling, can acknowledge that feeling, and empathise with the difficulties they might be having. Strong emotions can lead parents to exhibit challenging behaviour.

Showing sensitivity to the emotion while managing challenging behaviour produces a stronger nurse-parent relationship.

SOCIAL ASSESSMENT It may be necessary to speak to the primary health care team in order to make a full assessment of the families' social circumstances. Some issues may have been raised in the antenatal period and social workers may already be involved. In many cases, however, problems only surface during the course of the neonatal stay. Every neonatal nurse will be well aware of the myriad of social problems that they meet on a daily basis: poverty, homelessness, alcohol and drug misuse, single unsupported parents and multiple birth—the list is endless. When families present with very complex psychosocial needs the course of the neonatal stay is often affected. It is important to identify these psychosocial needs as soon as possible so that the relevant agencies can be involved.

Nurses need to be sure that they have good grounds for believing they know the needs of the family (Shelton *et al.* 1987). If nurses are to work with parents, they must develop the skill to accept them as they are. This depends on an understanding of differing cultural differences, lifestyles and background. It is easy to make assumptions about parents, but they may not be as ill informed about caring for their infant as nurses think. They need to be reassured and encouraged. It is important to respect all parents—and to hold as a fundamental belief that the vast majority of parents have the desire to be good parents.

Daily contacts with parents

Family-centred neonatal care should be based on open and honest communication between parents and professionals on medical and technical issues (Harrison 1993). Each nurse must establish a communication process with the parents that includes listening, and providing empathy and support. The nurse should always learn and use the parent's chosen name. When taking over the care of their infant it is important to acknowledge the parent, and to ask what they think has happened to their infant that day and listen actively to what the parent says. These contacts are invaluable in increasing parental self-esteem and creating a strong nurse-parent relationship. They allow the nurse to find opportunities to praise parents and reinforce the feelings of self-worth. Daily contacts are chances to identify parental concerns and negotiate solutions together. Listening to and acknowledging parents' feelings will help them to work through their emotions so that they can better cope with their situation.

Listening is a way of being with clients in a therapeutic way, accepting them (but not colluding with them) and caring for them in a way that fosters independence rather than dependence, thus allowing them to grow and develop (Reed-Purvis and Dakin 1993, citing Carl Rogers). Listening is fundamental to all 'helping'. The foundations of effective listening are the listener's respect for and empathy with the parent, and genuineness based on self-awareness.

Facilitating parents' attachment with their infant

The rhythmic, synchronised and empathic interaction between a mother and baby is often described as a 'dance' (Israel and Dolby 1997, citing Stern 1977 and Schaffer 1980). The dance is an individual one for each mother-infant dyad. Interventions that attempt to prescribe for a parent how and when to interact with their baby should be avoided. When infants are admitted to the neonatal unit the dance is interrupted. Israel and Dolby have developed a Parent-Baby Intereraction Programme which facilitates parent-baby interactions by teaching parents to recognise and interpret their infant's signals and behaviours during everyday caring activites.

Nurses should encourage parents and families to touch, talk to and stroke their baby, as he or she has a need for comfort, and to bring in small items of clothing for the infant as well as photographs, small toys and, if appropriate, some representation of their religious and cultural beliefs. The species-specific behaviour of touch is not completely satisfied until the parent is able to 'hold' the infant.

Developing key parenting skills

For a family to complete the bonding and attachment process they must be involved in providing care for the infant. Nurses need to work very closely with the parents, sharing their knowledge, demonstrating the necessary skills and providing parents with relevant information so that they can care for their infant. A skilled nurse can do this without appearing 'expert' and reinforcing the parents' feelings of loss of control. A mother's sense of self-esteem is threatened when she is taught about the special needs of a preterm infant by expert 'caregiving professionals' such as nurses, who typically are viewed as the 'good and skilled' mothers during the infant's hospitalisation (Affonso *et al.* 1992).

Involving the parents in procedures such as nappy changing, cord care, mouth care and changing the infant's sheets and position can help them to feel they have gained some degree of control.

Attachment and parenthood are complex, interactional developmental processes that must evolve over time. In the neonatal environment, however, it may seem that the role of the parent is condensed into a list of child care tasks that each parent must accomplish before hospital discharge. In reality, the parents willingly undertake these tasks as a means to eventually taking their infant home.

There are, of course, some key parenting skills in which parents of preterm or sick infants should be competent before leaving the neonatal unit.

- Parents should be instructed on how to keep their baby at the right temperature. Temperature control is usually dependent on two factors: developmental age (around 35 weeks) and parental skill. It is important to commence teaching temperature control to parents from admission. It is a

complicated issue, with the neonatal service staff urging parents to keep their infants warm and the Health Education Council advising 'Don't let your infant get too hot'. Parents need information that is tailored to their infant, the chance to practise in safety, i.e. in hospital, and support and reassurance once at home.

- Parents should be taught every aspect of how to feed their infant. If tube and cup feeding procedures are taught at an early stage they will be accepted as the norm. It is only when things are introduced as 'ways to take your infant home earlier' that some parents feel uncomfortable taking on tasks that they see as nursing duties.
- Parental training on 'what to do when an infant stops breathing' is increasing in popularity. It is important that parents receive this training before they have sole care of their infant.

Communication of quality information to parents

The Patient's Charter (1992) states that the clear explanation of any treatment, the access to health service data and the availability of information to relatives are the prerogative of all patients. This right to knowledge has long been recognised by parents and professionals (NAWCH 1985).

Facilities for parents

In this age of resource management, cost limitations and budgets, it is more and more difficult to argue for the 'soft issues' on the neonatal unit. High-tech issues always appear to get priority. Nurses need to have clear objectives when putting forward cases of need to neonatal or paediatric directorates. It could be argued that facilities for parents have significant influence on length of stay in the neonatal unit.

Planning programmes of family support

If any neonatal unit is to provide family support it must have trained and committed staff. In these days of staff shortages, busy units and more and more complex care it is often difficult for nurses to lift their heads above the parapet of the daily list of tasks to consider the long-term plan for the family. The nurse and the parent are the key people in this type of in-depth planning, which involves making decisions every day. Family support is a dynamic concept that is constantly changing and cannot be set in stone, but nurses and families need to be aware of the policies and guidelines that govern it. No one should feel unsure or compromised by unit policies that seem to offer everything to parents when the reality is something less.

Senior management must be committed to the principle of family support. It is their responsibility to raise awareness and engage the commitment of the nursing and medical staff. There are several ways in which staff and families can be encouraged to discuss family support on their unit. One suggestion is to look at the 'hot' issues around families and their care by using peer review meetings where nurses can debrief and share concerns. If family support is to be accepted at unit level, everyone should be entitled to express his or her views within a safe environment.

Finding out parents' perceptions of the care they receive can be more difficult. Continuous assessment can be carried out in a very informal way by the nurse talking to each parent on a one-to-one basis, finding out what is happening to him or her and what the parents perceive as their needs. However, if such information is to be valued then there must be a formal way of feeding it back to someone who can act on it. Parents support groups and networks can be a reliable source of information to help build family support policies. Group discussions, using buzz groups or focus groups, working with a dynamic group of parents as they pass through the neonatal unit, are extremely helpful but need a skilled facilitator. Staff can often feel threatened by such activity, but regular feedback sessions of parental viewpoints can be a great catalyst for change. Only by regular audit of the family support provision through parent questionnaires or interviews will neonatal units be able to maintain effective and relevant standards and policies.

Nurses need to act as advocate for the parent as well as advocate for the infant, speaking out on their behalf and using advocacy as a process where parents and nurses can be empowered. Family support only really works if all parties are willing participants. Parents may need to be empowered to take up their role of routine involvement.

It is important for managers to translate philosophies into action or nurses may feel overwhelmed by the enormity of the task. There is a need to demonstrate a change in practice, to allow staff to gain understanding and awareness, and for management to take into account the constraints on nurses' roles.

Conclusion

Caring for families takes time and effort. Parents need constant reassurance. Allowing parents to be involved in their infant's care by giving information from which they can make realistic choices is perceived to be more time-consuming than working in a directive manner. Telling parents what to do may appear less trouble for the nurse on that shift but it is a short-sighted strategy, as every nurse will have to adopt the same policy or be perceived by the parent as not helpful. The nurse who constantly tells, does for and colludes with parents creates a dependency culture within those parents. When this occurs, the question needs to be asked whose needs are being met, those of the parents or those of the nurse?

The challenge of providing family support is in developing the skills and expertise of working in partnership with parents. If neonatal nurses choose to work in a manner that acknowledges a family's right to participate in decision-making and respects their knowledge about their infant, they will enable the family to make health choices and by those choices take ownership of their infant's health. Teaching parents new knowledge and skills allows them to make independent decisions that influence health in the long term and assists them to retain control. Sick and preterm infants stay on neonatal units for such a short period of their lives but their parents will care for them until they reach adulthood. Supporting parents in ways that allow them to assume this responsibility must be one of modern neonatal nursing's objectives. Sherr and Rosenblatt (1994) capture the essence of this challenge:

> The future of the child lies in the capacity of the parents to nurture him or her throughout childhood. Any system, which allows parents greater opportunities to make contact, to learn their infant's needs, and promote parental feelings of importance, confidence and well being, will do much to optimise competency.

Case studies

The following case studies show how negotiation and formation of contracts with parents can be used to diffuse volatile situations and educate parents with the NICU environment.

Case study 1: discussion of family considerations for a preterm infant with chronic lung disease

Tracy is the 24-year-old mother of a 580 g, 23-week gestation infant, Michael, who is now 15 weeks old with chronic lung disease and requires low levels of oxygen via nasal cannulae. Tracy has been a resident in the neonatal unit accommodation since Michael's birth. Her husband, Richard, has returned home, some distance away, as he has a very demanding job and visits briefly in the evenings. Tracy spends all her time at Michael's cotside and performs all his cares independently.

Today something is wrong. Tracy is angry and tearful; she is short-tempered and abrupt with the nurse caring for Michael. The nurse seeks the advice of a more experienced member of staff. The two nurses decide to discuss the situation with Tracy away from the nursery in the less threatening environment of the unit 'Quiet Room'.

Tracy is very tearful and angry in the meeting. She refuses to look at the nurse caring for Michael and the atmosphere is difficult. The more

experienced nurse asks Tracy to share the story of Michael's birth and his stay in the neonatal unit.

Tracy's story is a common one and includes the following elements:

- A very precious but precarious pregnancy, with frequent admissions to hospital.
- An antenatal stay of 4 weeks where she was given antibiotics and uterine muscle relaxants to 'hang on to the baby'.
- A traumatic birth by cesarean section 'to give the baby the best chance'.
- Michael's slow and complicated progress through the neonatal unit.
- The feeling of isolation when Richard went home after visiting each evening.
- The lack of privacy in the busy low-dependency nursery.
- The feeling of being undervalued by the nurses when she did all Michael's cares but was not allowed to do more complicated tasks. Tracy had been reprimanded by the nurse for increasing Michael's oxygen level earlier in the day when his saturation monitor showed a fall in PO_2.
- No definite plans for Michael's discharge.

A contract was made that addressed Tracy's concerns and those of the nurses.

- Weekly 'Quiet Room' meetings were arranged between the nurse and Tracy to discuss any issues that had arisen in the week.
- Tracy would spend some time on her own, leaving the unit with Richard for short periods together; her neonatal accommodation would remain open to her.
- Michael would be moved to a side ward for privacy for the family.
- A contract between Tracy and the nurse caring for Michael would identify the boundaries of care for all parties.
- All nurses caring for Michael would be aware of the contract and of Tracy's involvement.
- Tracy agreed to work with the nursing staff in these matters.
- A discharge plan to include caring for the oxygen-dependent infant in the community would be started.

This plan of family support worked well. Tracy, Michael and Richard stayed for several more weeks in the neonatal unit side ward, and although Michael was eventually discharged into the community just after his due date he remained in oxygen and supported by the community paediatric team for two years.

Case study 2: discussion of family considerations for two teenage parents with limited reading and writing skills visiting the neonatal unit

Donna and Sean are the teenage parents of a 1200 g, 30-week gestation infant, Angela, who is now 3 weeks old. Donna and Sean live separately with their mothers but spend a lot of time in the neonatal unit visiting Angela. The nursing staff have expressed concerns that Donna and Sean do not seem to appreciate Angela's needs and often act inappropriately, i.e. handling her excessively. Donna and Sean spend a lot of time squabbling with each other about who should feed and change Angela. The nurses seek the advice of a more experienced member of staff.

Family support is offered in the following way. An assessment was made of Donna and Sean's educational status. Both parents disclosed that they had difficulty reading and writing. This had made it impossible for them to understand neonatal parental information. Once this problem was identified the nursing staff tailored the way they communicated with Donna and Sean to maximise their limited abilities. Breaking down information into small chunks and using it with a practical task (e.g. making up a feed) and constantly reviewing the parents' knowledge of a situation, and encouraging them by building on the gaps in their knowledge.

Donna and Sean were asked what was their level of understanding of Angela's condition and development status. They did not appear to be aware of the special feeding and handling needs of a premature infant. The nurse caring for Angela encouraged them to look carefully at her when they cared for her and to note the differences in her appearance and behaviour.

Donna and Sean were asked about the situation at home. Both were unemployed and living with their mothers and a wider family group, including step-siblings. Donna planned to take Angela home to her mother's house until a flat became available for them all. The Health Visitor was invited to visit the unit to meet Donna in preparation for eventual discharge. Donna was asked to think about the things she would need at home in order for Angela to be discharged. A referral to the Hospital Social Work Team enabled Donna to be more independent by acquiring equipment and organising benefits.

A contract was made, that addressed Donna, Sean and Angela's needs and the concerns of the nurses.

- Daily discussions with Donna and Sean and the nurse caring for Angela to plan her care for the day.
- Donna and Sean would agree to the plan and not squabble over who did what.
- The nurse would go through the parent information with Donna and Sean to ensure that they understood it and could benefit from it.

- All nurses caring for Angela would be aware of the contract and Donna and Sean's involvement.
- Donna and Sean agreed to work with the nursing staff in these matters.
- A discharge plan to include caring for the preterm infant in the community would be started.

This plan of family support worked well. Donna, Sean and Angela's stay on the neonatal unit lasted a further two weeks. They continued to squabble over Angela's cares and had to be reminded of the contract by the nursing staff. However, they were very keen to learn the new skills that the nurses taught and soon were caring for Angela in a manner that was more responsive to her needs.

Angela was discharged home to Donna and her mother's home and was followed up by the family care team and the primary health care team. She continues to do well.

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Chapter 18

Chapter 18

Ethics and Neonatal Nursing

Contents

Introduction

Neonatal nursing has witnessed a level of technological change and development in recent years which has resulted in the survival of extremely premature infants. This has called for a simultaneous development in the theoretical and practical skills of neonatal nurses. It has also increasingly exposed them to the intriguing world of ethics and moral decision-making, involving fragile human beings, often at the edge of viability.

Allmark (1992) examines the ethical component of the nature of nursing, suggesting that nurses make many ethical decisions in the course of their daily work. Not all of these decisions result from ethical or moral dilemmas: the decision may result from the nurse being faced with more than one option in a given situation and having to decide what is the most appropriate course of action to take. A moral or ethical dilemma arises when two or more of these courses of action could be appropriate and yet conflict with each other. Which ought the nurse to take? Beauchamp and Childress (1994) state that often these dilemmas can be resolved through reasoning and reflection. Some, however, may not. Jones (1997) provides further insight into the neonatal nurse as an autonomous practitioner who has to make choices.

The aim of this chapter is not necessarily to provide answers to specific situations but rather to stimulate thought and awareness about some of the ethical issues within neonatal care. It also seeks to explore briefly the issue of fetal rights and the role of neonatal staff in this arena.

Ethical theories: an overview

According to Beauchamp and Childress (1994), there are several types of ethical theory. It is not possible within the confines of one chapter to provide an extensive exploration of these. For more detailed information, reference should be made to the work of Gillon (1986), Rumbold (1993) and Beauchamp and Childress (1994). A brief overview follows of four of the ethical theories.

Utilitarianism or consequentialism

This theory adopts the belief that the act that leads to the best outcome for all is the right one to implement. If this were to be rigidly applied within neonatal nursing, then every nursing action would seek to result in the maximum benefit for every infant and family within the unit, all the staff and for society as a whole. There are, of course, occasions when a utilitarian-based decision may be the appropriate one to implement. An example of this would be where the maximum safety of all persons within the neonatal unit had to be ensured. A major criticism of utilitarianism is that it tends to be a more impersonal approach. It may even cause an immoral act to be committed against an individual whilst pursuing the maximum benefit for the majority of individuals (Beauchamp and Childress 1994).

Deontological or Kantian theory

This theory results from the work of Immanuel Kant (1724–1804) and is obligation- or duty-based. Rumbold (1993) provides an example of this, which could be applied to neonatal care, by citing the duty to preserve life. If this duty were to be rigidly adhered to in the purest deontological sense, decisions such as the withdrawal of treatment in the best interests of the infant would not be ethically possible. The duty is performed, irrespective of the consequences (Seedhouse 1998).

Virtue-based theory

Virtue ethics are concerned with the person who is required to make a choice and initiate the ensuing action. The morality of the action would be judged on the basis of whether or not this person is considered to be a good or virtuous person—that is, they demonstrate characteristics which are socially and morally valued. Although virtue ethics may influence one's perception of another, even virtuous people can make errors of judgement resulting in inappropriate action being taken. This creates a weakness in the sole use of this theory to try to determine the morally right course of action to take.

Rights-based theory

Rights, or justified claims, predominantly arise from legal and moral sources. They may also arise from documents such as the Patient's Charter (Department of Health 1995), or from individual institutions. Some rights are absolute but generally they are *prima facie* (Beauchamp and Childress 1994). In other words, most rights are only obligatory if not overridden by competing rights. Infringement of a right occurs when another has justifiably overridden it; violation occurs when it has been unjustifiably overridden.

A neonatal example of this can be found in the case of baby C, born in Ayrshire in 1996. The baby was born at 23 weeks' gestation and deemed, following clinical assessment by the paediatrician attending the delivery, to be non-viable. The parents challenged this decision, believing that the baby had been 25 weeks' gestation. It would appear from the Sheriff Principal's determination that they considered it to be their parental right to insist that resuscitative treatment should have been initiated. A competent paediatrician, considered to be acting in the best interests of the child, justifiably overrode this exercising of parental right, an action which was upheld by the subsequent Scottish law-based Fatal Accident Inquiry (Hay 1997). Montgomery (1997) discusses the principle of acting in the best interests of a patient incompetent of making a decision. *F* v. *Berkshire HA* ([1989] 2 All ER 545) and *Re C* (medical treatment) ([1998] 1 FLR 384) provide legal examples of how doctors may, at times, have a common law duty to act on behalf of patients for their best interests.

Although virtue ethics may influence the decision-making process for neonatal nurses, it is more likely that ethical decision-making will be founded on a combination of the other three theories. For ease of use, these can be succinctly summarised as being goal, duty or rights-based. Each has strengths as well as weaknesses but an awareness of the concepts should help neonatal nurses to be better advocates for the babies and families within their care.

Ethical principles

As with the preceding topic, the intention of the author is only to provide an outline of the four ethical principles. Further information can be found within the cited texts.

Respect for autonomy

Autonomy suggests the ability of an individual to think and act freely, to be able to make decisions and to be responsible for the results of those decisions (Jones 1997). This is reflected in the principles of responsibility and accountability, as laid out in the *Code of Professional Conduct* (UKCC 1992a). Respect for autonomy places a requirement on individuals to acknowledge the rights of others to act autonomously. Beauchamp and Childress (1994) develop this further and state that to do this requires more than just help. It also involves respectful attitude and action by one person or group of people to another. Obviously this does not necessarily mean promoting or respecting action that is illegal.

The neonate cannot exercise autonomy but parents can. If this is to be respected, it is important that neonatal staff ensure that appropriate information is given and understood. This should include the consequences of any decisions if parents are to be enabled to make truly informed choices. It will not always be the duty of the neonatal nurse to give this information but, as a partner in care, there is a responsibility to ensure that parents have opportunity to obtain it.

The principle of respect for autonomy can also be applied to the role of the neonatal nurse, particularly as role expansion and development increases. Hunt (1994) states that nursing autonomy is far more than simply the gaining of further biomedical knowledge. Whilst maintaining the close and cooperative inter-professional working relationships required when caring for neonates, neonatal nurses can exercise their authority to influence changes in policy and practice. This should be possible whether they have clinical, managerial or educational responsibilities. In order to do this it is necessary that nursing knowledge must also increase. Using the four patterns of knowledge identified by Carper (1978), it can be seen that development would be required in the areas of aesthetics or the art of nursing, personal knowledge and ethics as well as empirically.

The Scope of Professional Practice (UKCC 1992b) has enabled nurses to develop more autonomous practice. The introduction of the advanced neonatal nurse practitioner (ANNP) has also contributed to this, an issue which is discussed in more detail in Chapter 1. There is a risk, perhaps, that increased nursing autonomy may be viewed as being a power struggle between medicine and nursing. The challenge to neonatal nurses is to develop their autonomy as suggested by Hunt (1994) in order to be respected by others as professional practitioners.

Non-maleficence

In essence, this principle is that of *primum non nocere,* or the duty to do no harm. Therefore, any known side-effects of treatment, for example, must be weighed against the anticipated benefits (Gillon 1986). It would be morally reprehensible to pursue a line of care where side-effects outweighed any benefits, particularly in the name of research. Clause 2 of the *Code of Professional Conduct* (UKCC 1992a) reflects this responsibility for nurses. Inexperience may be excusable, as all neonatal nurses have had to learn their skills. Ignorance, however, is not an excuse when minimising or doing no harm to a vulnerable neonate is under consideration. Legally, the principle of a minimum level of competence is upheld in the findings of the case *Nettleship* v. *Weston* ([1971] 2 QB 691 (CA)) (Kennedy and Grubb 1994). All staff involved in patient care are held to a same minimum level of competence necessary for the safety of the patient. This highlights the importance of ensuring that staff are not placed in the position of having to take responsibility beyond their level of competence. It is equally important that each individual can also recognise the limits of their competence. Lack of competence may arise from lack of confidence. Therefore, there is an element of responsibility placed upon more experienced neonatal staff to support and encourage staff development through appropriate exposure to learning opportunities.

To seek advice, when uncertain, from a more experienced colleague is good practice. It may also, as demonstrated by *Wilsher* v. *Essex AHA* ([1986] 3 All ER 801), prevent individual staff being held liable in the event of a parental claim of negligence. In the event of medical/nursing staff facing litigation for negligence, standards of professional practice must be legally determined. This is done by the application of the *Bolam* principle in which staff are measured by the standard of practice deemed acceptable and reasonable by a responsible group of practitioners from the same specialty (Tingle 1998).

In an ideal world, accidents would not happen. Unfortunately, this is not the case and even the most meticulous care cannot always prevent incidents such as extravasation injuries from occurring. Nursing responsibility is to ensure that any necessary help and advice is sought and care initiated to reduce any further risks to the infant. Evidence-based care is influencing nursing practice, as can be witnessed from many clinical guidelines. Berragan (1998), in an

exploration of how different ways of knowing influence nursing practice, appears to voice some caution about a total reliance upon evidence-based guidelines. The reason given is that it could exclude the use of areas of aesthetic or personal knowledge such as intuition. Professional accountability and the principle of non-maleficence bind all medical, nursing and paramedical staff. Knowledge and information, however gained, are not under the exclusive ownership of a minority of people but need to be shared and discussed for the benefit of all.

Beneficence

Essentially, the ethical principle of beneficence is about doing good and actively seeking to promote the patient's welfare (Gillon 1986; Rumbold 1993; Beauchamp and Childress 1994). It is clearly the underlying principle of Clause 1 of the *Code of Professional Conduct* (UKCC 1992a). Beauchamp and Childress (1994) divide beneficence into two further principles, those being positive beneficence and utility. The former is concerned with the provision of benefits whilst the latter asks that those benefits be weighed and balanced against any drawbacks. Provision of neonatal care facilities is obviously a benefit to many families, that is positive beneficence. Utility, on the other hand, requires that the provision of such facilities to every infant, irrespective of their condition at birth, be balanced against what is in the best interests of the infant. This is probably particularly pertinent with the birth of an extremely preterm infant.

Beneficence is inextricably linked with the other ethical principles of autonomy, non-maleficence and justice (Gillon 1986; Beauchamp and Childress 1994). The following example aims to apply this link to practice. Neonatal nurses seek to promote the welfare of the babies in their care and to take reasonable precautions to minimise any harm to those babies. Yet they must also respect individual parental autonomy whilst balancing the needs of all the families in the unit. Policies and protocols offer some degree of fairness and equality for all, although this may not always be readily understood by relatives and visitors, who can then start to place additional demands upon staff. The process of acting beneficently can become a juggling act for staff, but there can be little argument that the welfare of all the babies will inevitably be the greatest influence upon the nurse.

Justice

It is very clear from the cited texts that any attempt to define what justice means is far more complex than may at first be anticipated. A common consensus within the literature is that Aristotle's principle of justice as being that which is fair and lawful is widely accepted within other theories (Gillon 1986; Beauchamp and Childress 1994). Fairness and legality appear to be straight-forward terms to understand, but they are, in fact, influenced by political, sociological and

religious beliefs. This is apparent from Gillon's description of libertarian, utilitarian and Marxist theories of justice (Gillon 1986).

In order to focus on what justice is, it may help to consider what constitutes an injustice. It is also important to remember that rights not only bring benefits, but equally place responsibilities upon individuals. According to Beauchamp and Childress (1994), injustice occurs when failure to act or a misdoing by one person or a group of people prohibits others from experiencing the benefits of their rights. That same failure to act or misdoing could result in an unequal or unfair distribution of responsibilities, something which could also create an injustice.

Justice may be distributive, criminal or rectificatory (Beauchamp and Childress 1994). Distributive justice essentially refers to the appropriately fair and equal distribution of resources within society. It is influenced by the infrastructure of that society and involves areas such as civil and political rights as well as the more obvious ones of health care and education. Criminal justice is concerned with the maintenance of law and order and the punishment of those who break the criminal law. Rectificatory justice is generally applied via the civil law and can be seen to be effected through fair compensation for medical malpractice, for example.

For neonatal nurses, it is probably the principle of distributive justice that is of most concern. This can be viewed from different perspectives. *Re B* ([1981] 1 WLR 1421) demonstrates how the judicial system may have to be involved in order to ensure fair access to health care if this is deemed to be in the interests of the child. The case centred on a baby with Down's syndrome who also had a duodenal atresia, the question of whether or not surgery should be performed being the cause of referral to the courts (McHale 1998a). The decision of the court was that surgery should be performed, Down's syndrome not being a prohibitive factor. With increasing demands being placed upon the National Health Service to provide a vast range of general and specialist services, it is difficult to see how finite resources can be equally, fairly and appropriately distributed throughout the country. Many neonatal staff will be familiar with the scenario of a pregnant woman in premature labour having to be transferred to another hospital due to lack of resources, for whatever reason, at the local hospital. Although that may be considered by some as being unfair to that individual woman, those making the decisions must also take the needs of others into consideration. Decisions involving the issue of resource allocation are reflected in *R* v. *Secretary of State ex p Hinks* ([1980] 1 BMLR 93 (CA)) and *Re J (a minor) (wardship: medical treatment)* ([1993] Fam 15, [1992] 4 All ER 614 (CA)). In the latter case, Lord Donaldson MR identified that it is a reality of life that health authorities may, due to insufficient resources, have to make choices regarding treatment. In the former case, Lord Denning MR stated that consideration must be given as to how a service can be provided to the whole country, not just to a particular hospital (Kennedy and Grubb 1994). As with beneficence, justice is closely linked with the other three ethical principles and it is clear that there are no easy answers as to how it can be truly implemented within an imperfect society (Gillon 1986; Beauchamp and Childress 1994).

Despite this having only been a brief overview of ethical theories and principles, it can be seen how they can influence even the simplest day-to-day decisions within a neonatal intensive care unit. When allocating staff to infants at the beginning of a shift, consideration is given to the needs of the infants as matched against the skills of the staff and the overall needs of the unit. Surely this is an unconscious balancing of the needs of individuals against the whole and an effort to maximise benefits and minimise risk of harm within the given resources. The application of ethics and moral decision-making to practice is not just about life and death decisions or the provision of resources on a national scale. It is present in everyday life and neonatal nurses, as autonomous practitioners, should be encouraged to develop their awareness of the subject.

Fetal rights

The issue of fetal rights or maternal-fetal conflict appears to be gaining some momentum, judging by the increased amount of literature published on the subject in recent years (Carter 1990; Draper 1996; Flagler *et al.* 1997; Reid and Gillett 1997; Rhodes 1997; Mohaupt and Sharma 1998). Although this trend has mainly emanated from the United States of America, there have been cases involving the issue in Great Britain. Most notable was that of *Re S* ([1992] 4 All ER 671) (Kennedy and Grub 1994; Draper 1996). Mrs S was expecting her third child and was six days overdue. She was admitted to hospital in spontaneous labour, with ruptured membranes and the fetus lying in a transverse position. Despite the gravity of the situation and being given relevant information, Mrs S refused consent to delivery by cesarean section on the grounds of religious belief. Application by the Health Authority to the courts for permission to perform the operation in order to save the life of Mrs S and the unborn baby was granted.

Whilst this may be a fairly extreme case, the underlying issues are important to the subject of fetal rights. The concept of fetal rights has already become an issue within legal cases such as *Re F (in utero)* ([1988] 2 All ER 193) and *Re MB* ([1997] 2 FLR 426). *Re F (in utero)* questioned whether or not F could be made a ward of court whilst in utero. F's mother led a nomadic lifestyle around Europe and there was concern about the adverse effects that this might have upon the fetus if medical attention was not received. Wardship was denied (McHale 1998b). *Re MB* [1997] was a case where a pregnant woman, overwhelmed by needle phobia, initially refused consent to delivery by cesarean section despite the fact that this could have an adverse effect upon the fetus. The courts could not declare such delivery as lawful if refused by a competent woman. In this case, however, surgical delivery was judged lawful, as the woman was deemed incompetent due to her needle phobia. Mason *et al.* (1999) provide valuable legal insight into this issue of fetal rights within the United Kingdom.

Fetal rights should also be important to paediatric and neonatal staff, as they are concerned with the welfare of the infant once born. An inappropriate choice of delivery could result in hypoxic damage to the infant, necessitating admission

to the neonatal unit. Neonatal staff have to provide care and support to parents who may be experiencing a high level of grief and anxiety about the infant. Therefore they cannot be distanced from the effects of decisions taken which try to balance the interests of mother and fetus. They may even have a vital role in the debate. It will not be difficult to see how some of the issues raised are equally valid within the environment of the neonatal unit.

Respecting maternal autonomy: the giving of informed consent

As has already been indicated, respect for autonomy is one of the four ethical principles. Linked with the right to be an autonomous person, according to Carter (1990), is the giving or withholding of consent to treatment. The Changing Childbirth document (Department of Health 1993) aims to enable pregnant women to exercise their autonomy and choice. This may occur through the woman being able to express preference over proposed methods of delivery. As in the case of Mrs S, however, there may be very good medical reasons to advocate one method of delivery over another even although this may clash with the woman's desire. Shortly before *Re S* [1992], *Re T* ([1992] 4 All ER 649) had led to Lord Donaldson advising health professionals to seek legal guidance if a person's mental capacity to refuse treatment gave cause for concern (McHale 1998c). This raises the issue of informed consent.

The principle of 'informed consent' does not exist within English law (McHale 1998c). Legally, it may be permissible not to inform a patient of the risks associated with a treatment providing that this is what a responsible body of professional opinion would expect. Morally, it is more questionable if an individual's autonomy is to be respected. How can someone make the right choice if they do not have the information necessary to help them understand the implications of that choice?

This raises the question of just how much information should be given and who should give it. Too much information may cause undue anxiety and distress whilst too little may give rise to an accusation of negligence. Paragraph 29 of *Guidelines for Professional Practice* (UKCC 1996) provides guidance on this. Although presented within the terms of gaining consent, the same principles can be applied to many situations where information is being given.

Clearly, the nurse is responsible for giving information when that is what would be reasonably expected under the terms of the *Bolam* principle, but it is not always nursing responsibility to give the information. If, however, the nurse is concerned about the level of information given or the recipient's comprehension of it, personal accountability is primarily to the patient or client (UKCC 1996). This does not mean that the nurse should immediately provide further information; rather, the advice and help of an appropriate doctor and/or line manager should be sought in order to reduce the risk of legal proceedings being initiated against the nurse (McHale 1998c).

The importance of careful documentation following the giving of information cannot be over-stressed. In years to come, this is what will provide evidence to

support or refute personal recollection of events. This is particularly important where practice in the neonatal unit may vary from information given to the public. One example could be the recommendation to place babies on their backs to sleep as a means of reducing cot death (Department of Health 1996). In neonatal units, it is not unusual to observe preterm babies being nursed prone as this contributes towards an improved respiratory status (Kurlak *et al.* 1994). This apparent conflict of practice may cause confusion for parents and it is therefore important that staff clearly explain the rationale for their actions and the parameters within which they work. Prior to the discharge of the baby, professional practice should be seen to be consistent with information given to parents. Within the arena of fetal rights, the appropriate people must give the pregnant woman information if autonomous decision-making is to be empowered. This may well involve members of the neonatal and paediatric teams who are best equipped to provide information relevant to the future of the baby, should admission to the neonatal unit be anticipated.

It is possible, despite attempts to persuade otherwise, that the woman may choose not to consent to or follow the professional advice given. This is her right as an autonomous person but she must then also be willing to accept the responsibility for the consequences of that choice. *Re T (a minor) (wardship: medical treatment)* ([1997] 1 All ER 906) confirms that it may be legally permissible for a parent to make an autonomous decision not to consent to professional advice being carried out. This does not absolve staff from their duty of care. They may have to seek more experienced or specialist help and will have to continue to provide care within the parameters placed upon them. There must be documentation regarding the information given, which includes the consequences of a refusal and the reasons for the refusal (Brahams 1995; Montgomery 1997).

Should fetal rights exist?

There seems a certain irony in the fact that, with one exception, the fetus is not a legal person before birth and therefore has no rights. Yet immediately after birth, the infant has legal and moral rights as a living person. The need for safeguard and care as well as legal protection for children before and after birth is acknowledged within the Convention on the Rights of the Child (General Assembly of the United Nations 1992).

The only legal rights afforded to the fetus under English law are contained within section 59 of the Offences Against the Person Act 1861 and the Infant Life Preservation Act 1929 (Davies 1994). The former protects the fetus from death resulting from the unlawful causation of miscarriage. The latter makes it an offence to intentionally kill a fetus that is capable of being born alive, this being defined as a fetus with a gestational age of 28 weeks or more. Obviously the Abortion Act 1967, amended by the Human Fertilization and Embryology Act 1990, permits the legal termination of pregnancy. It was as a result of the last Act that the age of viability was reduced to 24 weeks. Interestingly, the gestational age of 28 weeks presumed by the Infant Life Preservation Act 1929

has not yet been amended. It may be that this has been considered unnecessary because of the amendment to the Abortion Act 1967.

The reason why there are no other fetal rights is because, legally, the fetus is not a person (Montgomery 1997). This is clearly demonstrated in several of the cases already cited. What constitutes being a person? Why should the fetus prior to birth not be regarded as a person and yet afterwards is? The arguments about what is a person and what is personhood are complex and contentious. They also appear to be strongly influenced by views on when life begins and when a human being is capable of self-consciousness (Brykcznska 1994; Strong and Anderson 1994; Reid and Gillett 1997).

Gillon's definition of 'person' questions the requirement for self-consciousness in order to be a person with moral rights by suggesting that newborn babies would not be entitled to be regarded as persons with the right to life (Boyd *et al.* 1997). It is doubtful that this would be an acceptable situation to society as a whole and demonstrates some of the difficulties involved in philosophical debates of this nature. The legal perspective simplifies the situation a little. According to Ivamy (1993), any human being who is capable of having rights is a person. This stand is supported by legislation such as the Children Act 1989, which offers legal protection to children from the time of birth, irrespective of gestational age.

Neonatal nurses are well aware of the effect that technological advance has had within obstetric and neonatal care. The lives of infants of 24 weeks' gestation, occasionally less, can now be saved and it is possible for some forms of fetal surgery to be performed. This may cause some people to question why the fetus should not have rights. Equally, the distress of caring for a baby suffering from severe drug withdrawal secondary to maternal addiction may prompt the same question. In order to try to find an answer, the wider issues involving the ethical principles must be considered.

If, for example, the fetus had the legal right to be protected from harmful substances what effect would this have upon the mother's moral right to act as an autonomous individual? Draper (1996) offers the example of women who smoke during pregnancy. The suggestion is made that if the fetus was given legal protection against exposure to passive smoking whilst in utero, then the resultant obligation placed upon the mother would also have to be placed upon society to protect all children from the effects of passive smoking. Such fetal rights could put society on a road towards prohibition of certain lifestyles. Conversely, to argue against action taken in the interests of the fetus because it is not a person could ultimately place society on a road towards infanticide (Draper 1996).

It is evident from the literature that there are no easy or quick answers to the controversies contained within the subject of fetal rights (Carter 1990; Draper 1996; Reid and Gillett 1997). Perhaps the legal system is correct in adopting the stance it does. On the other hand, technology enables infants to survive who may not have done so when the laws came into being. For parents who suffer the loss of a fetus of a viable age through no fault of their own, possibly as the result of accident or injury, there can be little comfort to realise that the law

does not regard their 'baby' as a person. The time may be coming for the law to recognise 'potential personhood'. Will neonatal nurses be ready to advocate on behalf of the unborn child in a manner that supports and complements maternal rights?

Ethics in practice

Throughout this chapter, it has been demonstrated that the use of ethical theories and principles has a very real place within clinical practice for neonatal nurses. Unfortunately, the application of ethics to practice does not always remove the interpersonal conflicts that can occur when staff hold different views that may all be equally justifiable. If professional ethics conflict with the law, clearly the legal guidance must be adhered to if there is not to be an accusation of breach of civil or criminal law levied. The blind following of a doctor's order will not be sufficient justification for the nurse who recognises an error but fails to exercise professional accountability and autonomy in seeking to correct it. The need for nurses to be guided by medical instruction, especially in areas such as neonatal intensive care, creates the paradoxical situation of the nurse being, yet not being, an autonomous professional. How can this be overcome and will it ever be overcome? Perhaps it requires nurses to reflect upon what is happening in both their practice and that of others. If the consequences of decisions made are considered using some form of ethical framework, this may aid nurses to feel sufficiently empowered to exercise their professional autonomy.

When reflecting upon situations and trying to find what the right answer may be, there is a very real risk that personal values and feelings may inhibit the adoption of an unbiased perspective (Johnstone 1988). Research by Hammerman *et al.* (1997) suggests that families, when involved in medical decision-making, are more strongly influenced by philosophical, moral and religious beliefs rather than specific life experiences. If such research were to be repeated with neonatal staff, it would be interesting to observe whether the same phenomenon appears. It is difficult not to allow a blurring of the edges between professional and personal relationships with babies and their families. This may be particularly true with the families of longer-stay babies. As more extremely premature babies survive, it is quite possible that there will be an increasing number of 'geriatric neonates' in units. This highlights the importance of staff being able to resolve conflict between personal values and ethical decision-making through an enhanced understanding of ethics.

An ethical grid, such as that shown in Figure 18.1, has the issues of autonomy and respect for autonomy at the centre. As the grid radiates out, all the ethical principles are reflected. However, by asking a series of questions, the neonatal nurse may be able to create a framework appropriate to each individual situation. These questions can begin from any layer of the grid, although some may consider the centre elements to be the basis from which to start. Not every facet need be included but consideration must be given as to which are the most appropriate ones at the time of discussion.

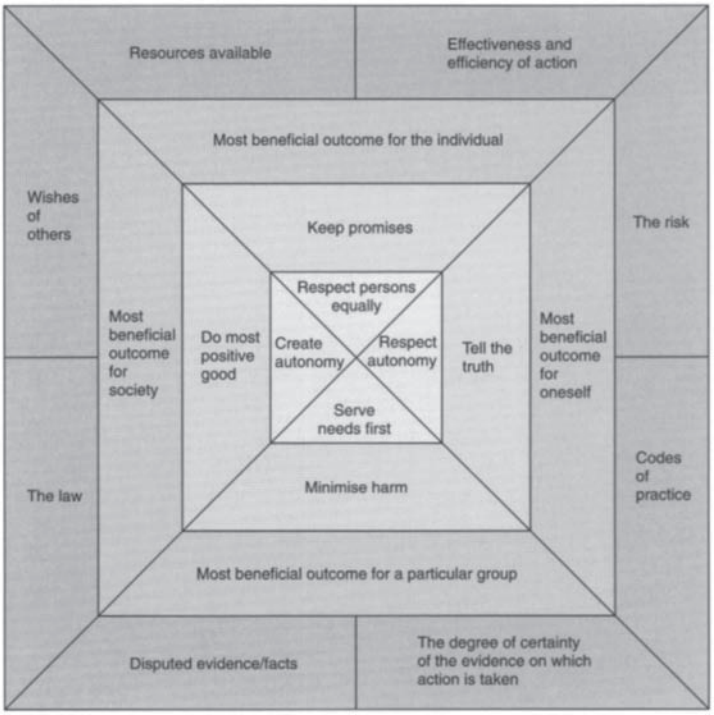

Figure 18.1 **The ethical grid**

Source: Seedhouse 1998:209, with permission of John Wiley & Sons

Case study: consideration of the ethics of pursuing an aggressive management policy in a situation involving a preterm infant with possible long-term neurological damage born to a 39-year-old mother with no other children

The following case study offers an opportunity to explore a possible ethical framework for use within a neonatal unit.

Baby Peter is 10 days old. He was born at 26 weeks' gestation after his 39-year-old mother went into spontaneous labour. His birthweight was 950 g. Peter's parents have no other children and have been anxious to have a baby for several years. His mother did not realise that she was in labour. On arrival at the hospital, she was in advanced labour and there was insufficient time to administer any steroids prior to delivery. Peter was given surfactant shortly after birth. Despite this, ventilatory management

has been difficult and he has been very unstable. Peter has required inotropic support since 3 hours of age. A head ultrasound scan has revealed a left-sided grade three intraventricular haemorrhage. Concern has been expressed regarding the possibility of long-term neurological damage as periventricular flaring was also noted. Within the past 48 hours, Peter has become increasingly unstable and has a severe mixed acidosis. Infection screening has been carried out; initial reports are suggestive of overwhelming sepsis. Given the difficulties in managing this baby, Peter's consultant is considering whether it is appropriate to pursue an aggressive line of management.

Before discussing the situation with Peter's parents, it has become obvious that there are very strong and differing views about the situation amongst the nursing staff on duty. The senior nurse for the unit has realised that this dissension and inharmonious atmosphere could be detrimental to parents and staff. The decision has been made to try to help the staff analyse the situation objectively in order that they continue to provide professional standards of care to Peter and his parents.

Q.1. What personal values may be influencing the staff?

Q.2. Compare these values against each of the ethical theories. Why has conflict arisen?

Once the bias that arises from personal values in situations such as this has been identified and acknowledged, it is important that those involved can try to place it to one side as the wider issues are discussed.

Q.3. How may each of the ethical principles be related to this situation?

Q.4. It is important that staff consider long-term consequences of any decisions as well as the immediate ones. The clinical evidence presented in Peter's case would suggest that, if he were to survive, he would have on-going health and educational needs. With consideration to this, apply the ethical theories and principles as well as the concept of informed consent to the four options now presented:

- Option 1: Present treatment is withdrawn.
- Option 2: Present treatment is continued but no new treatment initiated in response to further deterioration.
- Option 3: The situation is controlled and continued responses made to alterations in Peter's condition on an individual basis. Does this allow a controlled pushing of the boundaries?

- Option 4: Treatment is continued at all costs either because Peter is presenting a challenge to be overcome, boundaries have to be pushed in order to further skill and knowledge, or parental wishes are being respected.

Q.5. How may neonatal nurses exercise their professional accountability, responsibility and autonomy to Peter, his parents and other members of staff?

The difficulties encountered in reaching life and death decisions within neonatal units have been researched by McHaffie and Fowlie (1996). It is clear that, when trying to find answers to situations such as this, it is vital that staff utilise the appropriate legal and professional guidelines, for example, those produced by the Royal College of Paediatrics and Child Health (1997). It is possible, with the use of an ethical framework, to reach apparently opposite decisions. Using the case study given, neonatal staff may decide that the primary considerations are to minimise harm to Peter, to consider the efficacy of any future treatment as balanced against the likely outcomes and the future impact upon his family and his life. This could lead them to advocate for Option 2 to be followed. On the other hand, Peter's parents may be prepared to adapt their lifestyle to accommodate his needs and wish to give him as much care at home as they can. By respecting their autonomy as parents, to follow Option 3 may enable the most positive good to be done for that family in a manner that respects their wishes.

Conclusion

Ethical decision-making occurs on many occasions within neonatal units. It is not solely about dealing with difficult situations or dilemmas such as those presented within the case study. If the question 'what is the right thing to do in this situation?' is asked, then an ethical approach is required to answer it. In order to do this, neonatal nurses must have some understanding of the principles and theories that are involved.

Consideration should be given as to why a particular course of action is believed to be correct. An overview of four theories demonstrates the diverse approaches that can be taken and the fact that no one theory will always be the right approach for every situation. On some occasions it will be necessary to achieve the best outcome for all concerned. Other occasions will require predominant consideration by staff of duty or the rights of an individual. It is probable that the majority of situations will require a combination of approaches.

Whichever theoretical stance is adopted for the problem-solving or decision-making process, the four ethical principles of respect for autonomy, non-

maleficence, beneficence and justice must be adhered to. As neonatal nurses expand and develop their roles, so professional accountability and responsibility increases. An enhanced understanding of the ethical and legal framework within which nursing and medicine function will enable nurses to increase their professional autonomy.

Neonatal nurses should be aware of external factors that may impact upon their practice and be able and willing to respond to the opportunities and challenges that can result. Such an issue has been presented within the concept of fetal rights and how neonatal nurses may support midwifery colleagues to enable pregnant women to make informed choices.

The use of ethics within the daily life of neonatal nurse is valid and necessary. Although this is a complex subject which can raise contentious issues, it does not have to be beyond the reach of every member of staff. Indeed, the use of ethical decision-making processes and an enhanced awareness of ethical principles should be encouraged.

Legal references

All ER	All England Law Reports
BMLR	Butterworths Medico-Legal Reports
Fam	Family Division Law Reports
FLR	Family Law Reports
QB	Law Reports, Queen's Bench Division
WLR	Weekly Law Reports

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Re J (a minor) (wardship: medical treatment) [1993] Fam 15, [1992] 4 All ER 614
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Chapter 19

Chapter 19

Medication in the Newborn

Contents

Introduction

The interest in the use of medicines in children is growing, with neonates being a unique group within this population. They have impaired renal and hepatic function in comparison with adults and also have limited ability to absorb medicines orally, particularly when they are sick. It is not usually appropriate to prescribe medicines to neonates purely as a proportion of the adult dose. Medicines tend not to be manufactured with the neonate in mind and frequently are supplied in a form that makes administration difficult, both in calculation and delivery of the dose. This creates problems for the staff on the neonatal unit in terms of the administration and economic use of drugs. Many medicines used in neonates are used outside the terms of the product licence. This may be a matter of great concern to staff working in a neonatal intensive care unit as information is often collected in infants after the drug has been used in adults. It is the responsibility of health professionals to ensure that toxicity of medicines is kept to a minimum but that children are not denied appropriate medicines (Choonara *et al.* 1996). In order for nurses to both safely administer drugs to neonates and monitor their effects, it is important that they have an understanding of the way that the body deals with drugs.

This chapter will aim to provide information on the way in which neonates handle drugs in the body (**pharmacokinetics**) and the importance of this information in both the calculation of drug doses and drug monitoring. The use of unlicensed medicines will also be considered.

This chapter will not attempt to provide comprehensive guidance on dosage and specific details on drug administration. Most units will have guidelines and policies which should be followed. It is the responsibility and also a professional requirement of nurses to have an understanding of drugs they administer (UKCC 1992), and if necessary, additional information, beyond the scope of this chapter, should be sought. There are many texts, including *The Neonatal Formulary* (Northern Neonatal Network 1998) and *Medicines for Children* (RCPCH 1999), which contain more detailed drug information. *The British National Formulary* (Joint Formulary Committee 2000) also contains very useful material.

Pharmacokinetics

Pharmacokinetics is a mathematical way of describing the way in which the body handles a drug. The most important processes involved are *bioavailability, distribution* and *clearance,* as defined below. These factors vary between individuals and need to be considered when determining a dosage regimen. Neonates are a diverse group in terms of their renal and hepatic maturity, making it extremely difficult to predict how an individual infant will respond to a drug.

Bioavailability describes the extent of the administered dose which is available in the body to exert a pharmacological action. If a drug is given

by the intravenous route, the entire dose is available, in other words it is 100 per cent bioavailable or has a bioavailabilty of 1.0. Medicines given by the oral route may be incompletely absorbed or be partially metabolised by the gut or liver before entering the systemic circulation—hence they have a bioavailability of less than 100 per cent; a drug that is only 80 per cent absorbed has a bioavailabilty of 0.8. Drugs given by other routes, including rectal, percutaneous, intramuscular and subcutaneous, usually have reduced bioavailability compared with the intravenous route. Chemical properties of the drug will also determine the rate and extent of absorption.

Several factors affect the absorption from the gastrointestinal tract. The most relevant are *gastric emptying* and *gastric pH*—both of which continue to change with maturation of the infant. In the term infant, gastric pH is neutral at birth, drops to 1.5–3.0 during the first few hours, returning to neutral over the next 24 hours. For the following two weeks of life there is relative achlorhydria. In the preterm infant there is no initial fall in pH because of immature secretory mechanisms (Morselli 1989). The gastric emptying rate is prolonged in neonates compared with older infants; it is further delayed in premature neonates compared with term infants. Gastrointestinal transit time and peristalsis are increased in infants; these are also influenced by the type of feed the infant receives. For example, prolonged gastric emptying times were demonstrated with feeds of increasing calorie density (Siegal *et al.* 1984). The delay in gastric emptying may decrease or delay the peak concentration of a drug which is given by the oral route, although the clinical significance of this is not known. A combination of these factors contributes to the erratic and unpredictable gastrointestinal absorption. In infants who are shocked, acidotic or obviously unwell, there is a risk of paralytic ileus and delayed absorption with oral medication.

After the drug has entered the systemic circulation by whatever route, it has to be distributed throughout the body. *Distribution* is influenced by chemical properties of the drug, route of administration and patient variability. Many of the factors that influence drug distribution change markedly as the neonate matures. For the purposes of simplification, the body can be described in terms of an extracellular and an intracellular compartment, the extracellular compartment being the greater. Water-soluble drugs are distributed mainly in this compartment. This process is described by the *one-compartment model*. It is assumed that after the drug appears in the blood it quickly distributes within the body tissues so that the rate of change of the concentration of the drug in the blood is equivalent to the rate of change of the concentration in all body tissues. It is important to remember, however, that many drugs distribute into more than one compartment—for example, two compartments, where one is a small compartment made up of the plasma and well-perfused organs (brain, liver, gastrointestinal system, heart and kidneys) and a second compartment made up of the rest of the body. The drug concentration in each compartment will vary at any given time (Roberts 1984). The apparent space into which the drug distributes is described by the *volume of distribution*. It does not actually refer to a

physiological volume but does indicate the total amount of drug in the body relative to the concentration in the blood. In neonates, total body water makes up to 75 per cent of the total body weight and it may be as high as 85 per cent in preterm infants. Water-soluble drugs that distribute into body water, such as frusemide, therefore, have a greater volume of distribution in neonates compared with adults. This means that for a given dose of drug the total concentration in neonates is lower.

Fat only contributes about 20 per cent of body weight in children, and is further reduced in the neonate compared to older children.

After a drug has been absorbed into the systemic circulation it is either free drug or bound to plasma proteins. Only free drugs are active and can exert a therapeutic effect. Plasma proteins are relatively large molecules with binding sites on the surface. Circulating free drug attaches to the binding site, becomes bound and therefore inactive. Drug-protein binding is a reversible process. Drugs have differing affinities for plasma proteins. Any factor that affects the extent of binding to plasma proteins will affect the amount of active drug in the body. These factors include administration of other plasma proteins (such as albumin), presence of other drugs which have a stronger affinity for plasma proteins, and other compounds, such as bilirubin, which bind to plasma proteins. For example, indomethacin is 95 per cent bound to plasma proteins and only 5 per cent is free and capable of exerting a pharmacological effect (Ohning 1995a). If the degree of protein binding of indomethacin changes from 95 per cent to 90 per cent this will result in an increase in the free drug from 5 per cent to 10 per cent, a doubling of the active indomethacin concentration in the blood.

Acidic drugs tend to bind primarily to albumin compared with basic drugs, which have a greater affinity for other proteins—alpha-1 acid glycoprotein and lipoprotein. These proteins are different in infants from adults, both in the concentration in the body and in their affinity for drugs. Neonatal albumin has a reduced binding capacity for drugs compared with adult albumin. For example, greater concentrations of free drug were found in cord blood (which contains fetal albumin) compared with concentrations found in adult plasma for several drugs including phenobarbitone, penicillins and morphine (Reed and Besunder 1989). There is a theoretical risk that there will be competition for albumin binding sites between drugs and bilirubin. Displacement of bilirubin from albumin by drugs could result in kernicterus. This is historically reported with sulphonamides which resulted in many babies suffering brain damage as a result of their antibiotic medication (Silverman *et al.* 1956). There is only likely to be a significant problem with displacement of bilirubin with drugs which are more than 90 per cent protein bound (Rylance 1991). The affinity for binding to albumin by bilirubin is considerably greater than most drugs and therefore the clinical significance of this is still not known.

Clearance describes the rate of drug removal and can be described in terms of plasma clearance, organ clearance or total body clearance. There are two main processes involved in the clearance of drugs from the body— metabolism, primarily in the liver, and excretion by the kidney. Lipid-soluble drugs need to

be converted into more water-soluble compounds in the liver before being removed by the kidney. Total body clearance is the sum of these processes.

The processes by which drugs are modified in the liver are known as *biotransformation reactions,* which can be subdivided into phase I (non-synthetic) and phase II (synthetic) reactions. Phase I reactions include oxidation, reduction and hydrolysis, which result in the formation of inactive compounds or alternatively active metabolites. These compounds are usually more water-soluble than the original drug and hence can be removed more easily by the kidney. This group of reactions depend on enzyme systems in the membranes of the hepatic microsomal endoplasmic reticulum or enzymes present in other parts of the liver (deaminases) or the blood (esterases). Phase II reactions involve conjugation with glucuronic acid or glycine and sulphate production. These reactions involve liver microsomal pathways (e.g. cytochrome P_{450}) and take place in the mitochondria and in solution in the cell cytoplasm. The drug molecule is rendered inactive as a result of the attached group (e.g. glucuronide) preventing it from crossing biological membranes (Ohning 1995b).

In neonates these metabolic processes are immature, the extent of which depends on the gestational and postnatal age of the infant. The different metabolic reactions mature at different rates. Cytochrome P_{450} activity is about half the adult capacity in term infants. Sulphation and glycination occur at similar rates to adults but glucuronidation is reduced (Rylance 1991).

Removal of drugs and their metabolites may also be performed by the kidney. The factors which effect elimination are blood flow to the kidneys, glomerular filtration and tubular secretion and reabsorption.

Premature infants have a reduced degree of glomerular filtration compared with babies born at term and the rate of maturation is correspondingly slower. It reaches similar rates to adults at around 5 months of age. Glomeruli continue to be produced up to 35 weeks' gestation so it follows that a baby born preterm will have a reduced number of glomeruli and therefore a lower glomerular filtration rate (GFR).

Glomerular filtration in the kidney allows removal of small molecules, which include most drugs, but not large plasma proteins. The free drug molecules are transferred into the renal tubules by glomerular filtration. This process cannot remove protein-bound drug molecules. If the infant becomes hypotensive, the GFR will fall and the rate of removal of drugs by the kidney will be reduced. If this is not taken into account when calculating a suitable dose of a drug, it may result in drug accumulation and toxicity. Any process which affects renal blood flow will influence drug clearance by the kidney.

Most drugs are removed by the liver, the kidney or a combination of the two. Gentamicin is excreted purely by the kidney and because of its potential toxicity should be closely monitored, particularly in babies with impaired renal function. Paracetamol and morphine are examples of drugs that are mainly metabolised by the liver. In adults and children paracetamol is metabolised by glucuronidation. In preterm infants, however, this pathway is immature, so the sulphation pathway is utilised. This mechanism is only used in preterm infants. Phenobarbitone is removed from the body by a mixture of renal and hepatic mechanisms. This is a

much slower process in a neonate compared with an older child and explains the prolonged neonatal half-life of phenobarbitone, which may be up to 200 hours compared with around 70 hours in children.

It is clear that it is important to know how the body deals with drug elimination when prescribing for neonates. The rate at which a drug is removed from the body is described by the pharmacokinetic term *elimination half-life*, which can be defined as the time it takes for the concentration of drug in the blood to fall by a half. The maturity of the infant will affect the pathways available for drug metabolism and excretion. As such, dosage regimens should be adjusted to take into account the gestational and postnatal age of the infant. As the baby matures, the metabolic processes used may change, as with paracetamol. If a drug is given an infant where there is not a mature metabolic pathway capable of handling the drug, accumulation and toxicity may develop.

The different pharmacokinetic parameters illustrate the different clinical effects of drugs. Morphine has a prolonged half-life in preterm infants of 6–14 hours compared with 2–11 hours in term babies. Morphine is metabolised by glucuronidation in the liver to form two metabolites—morphine-6-glucuronide (M6G) and morphine-3-glucuronide (M3G). M6G is a potent analgesic itself and also is a respiratory depressant but M3G antagonises the analgesic effects of both morphine and M6G and stimulates respiration. M3G has been found to be the predominant metabolite in sick preterm infants with M6G being present to a variable extent because of the reduced ability by premature neonates to convert morphine to M6G (Hartley *et al.* 1993). The variability in clinical response to morphine may be explained by the variability in morphine metabolism in sick neonates. Infants who produce significant amounts of M6G experience a greater analgesic and sedative effect than those who are able to conjugate M6G to a lesser extent (Bhat *et al.* 1992).

Diamorphine (di-acetylmorphine) is a semi-synthetic derivative of morphine which is metabolised to morphine. It, like morphine, is given as a loading dose followed by a continuous infusion (Barker *et al.* 1995). It has little to offer over morphine with the exception that it causes less histamine release and may cause less hypotension. The hypotension which occurs may be due to a reduction in stress in the infant as a result of the analgesic and sedative effect (Elias-Jones *et al.* 1991). Diamorphine has similar effects to morphine on respiration and the gut. It is more lipid-soluble than morphine, which may cause sedation more quickly than morphine (Wood *et al.* 1998).

Fentanyl is also used as an analgesic and sedative in neonatal intensive care. It has a shorter duration of action than morphine and is 50–100 times more potent, primarily as a result of its greater lipid solubility. It has a half-life of 6–32 hours in neonates (Koehntop *et al.* 1986) which is prolonged in babies undergoing ligation of a patent ductus arteriosus (Olkkola *et al.* 1995). It is eliminated almost entirely by metabolism in the liver. Fentanyl is given as a loading dose followed by a continuous infusion. The main advantage of fentanyl over other opiates is that it rarely causes haemodynamic instability since it is unlikely to cause histamine release. One study in neonates has also indicated

that fentanyl may cause fewer problems with gastrointestinal motility and therefore enteral feeding (Saarenmaa *et al.* 1999).

Chloral hydrate is a sedative with no analgesic properties. It is usually given orally, although it can also be given rectally. Chloral hydrate has a long but variable half-life, particularly in preterm infants and those with impaired renal and hepatic function. It may also displace bilirubin from its binding sites and long-term use may cause hyperbilirubinaemia. Chloral hydrate is metabolised in the liver to trichloroacetic acid and trichloroethanol, which also has sedative properties (Alexander and Todres 1998). The main side-effect is gastric irritation and it has been associated with paradoxical agitation. There is a risk of accumulation of chloral hydrate because of its long half-life and this has been associated with toxicity; long-term use should be avoided.

Therapeutic drug monitoring (TDM)

TDM is necessary in neonates because doses required are often very different from those used in children and adults. It is usually used when the therapeutic concentration of a drug and the toxic concentration are close together. This type of drug is said to have a *narrow therapeutic index*. The drugs which are most commonly monitored in neonates include the aminoglycosides (gentamicin, netilmicin, amikacin, tobramycin), vancomycin, phenobarbitone, phenytoin and theophylline (Patrick 1995). This subject is still relatively new in neonatology and the drug levels required in neonates may be different from those in older children or adults. A good example of this is theophylline (Table 19.1).

***Table* 19.1 A comparison of serum theophylline levels in infants, children and adults**

	Desired therapeutic range (mg/l)	
Infants	*Children*	*Adults*
5–12	5–15	10–20

Source: Walson 1997

The main problem with TDM in neonates is that it usually requires blood samples. Not only is this painful for the infant but it can also contribute to iatrogenic anaemia as a result of the low circulating blood volume necessitating blood transfusions (Gilman 1990). With a blood volume of 80–100 ml/kg, a premature neonate weighing 500g will only have a total blood volume of 40–50 ml. Laboratories that have microassay techniques available should be used for TDM in infants (Koren 1997). Some drugs will require both trough levels (blood taken immediately before a dose when the concentration is at its lowest) and peak levels (blood taken at a specified time after a dose when the concentration

is thought to be at its highest) and some may only require trough levels to be measured. It is important that levels are measured at the correct time, and this varies from drug to drug, otherwise the information could at best be meaningless and at worst be dangerous. Serum concentrations have been determined for the narrow therapeutic index drugs which relate concentration to therapeutic effect and toxicity (Sagraves 1995).

***Table 19.2* Plasma elimination half-lives (in hours) of some drugs given to neonates**

Drug	*Preterm infants*	*Term infants*
Caffeine	31–132	26–231
Gentamicin	3.5–16.1	2.3–5.9
Phenobarbitone	60–200	41–120
Phenytoin	60–130	10–100

Source: Rylance 1991

Drug levels are usually taken when it is expected that the drug will have reached a steady state within the body, that is, when the rate of absorption is equal to the rate of elimination. This is usually taken as four to five times the half-life of the drug. The half-life will vary between individuals and is also influenced by the age of the patient. Some examples are shown in Table 19.2. For drugs that have a long half-life, such as phenobarbitone, it can take many hours or even days to reach the desired therapeutic blood level. It is for this reason that loading doses are given with these drugs to enable therapeutic drug levels to be reached more quickly.

Unlicensed medicines

In 1968 the Medicines Act was introduced to provide legislation covering licensing of medicines to try to ensure safety, quality and efficacy. The Act was in response to drug-related toxicities which occurred with thalidomide (phocomelia) and chloramphenicol ('grey baby syndrome') (Mulhall *et al.* 1983) in the developing fetus and neonate. Despite this legislation, infants and children continue to receive medicines that have not been subject to the licensing system (Choonara and Dunne 1998). In the United Kingdom, most drugs have a product licence issued by the Medicines Control Agency (MCA) which delineates the indications for which a drug can be prescribed and the recommended dose. Currently, many drugs used in children and neonates are not covered by the product licence—that is, their safety and efficacy have not been endorsed by the manufacturers. Drugs that are used which do have a product licence but do not have an indication for use in children are said to be used 'off label'.

Similarly the formulations available of many medicines are not suitable for administration to children. It may be necessary for the pharmacist to manipulate the medicine in some way to enable it to be administered to a child (Nahata 1999). These medicines are unlicensed. Tablets that may need to be crushed to form a suspension, injections or parenteral nutrition which are provided in a ready-to-administer form by a pharmacy aseptic unit, and medicines that are imported from another country are all examples of unlicensed use of medicines.

It is not illegal for a doctor to prescribe, a pharmacist to dispense and a nurse to administer an off-label or unlicensed medicine, the Medicines Act 1968 makes provision for this to take place. The licence prevents pharmaceutical companies from promoting an unlicensed product or an unlicensed use of a product. The doctor carries responsibility for the prescribing of the medication. The standard reference texts which provide drug information are often of little use in neonatal prescribing and it is necessary to refer to more specialist paediatric or neonatal formularies—for example *The Neonatal Formulary* (Northern Neonatal Network 1998).

A study on a neonatal intensive care unit by Conroy *et al.* (1999) showed that 90 per cent of patients were given a drug that was either unlicensed or used off-label. Nearly 55 per cent of prescriptions were off-label and 10 per cent were unlicensed.

In May 1996 a joint working party between the British Paediatric Association (BPA) and the Association of the British Pharmaceutical Industry (ABPI) published a report on the licensing of medicines in children (Working Party of the BPA and ABPI 1996). In 1997 European guidelines were published on the clinical investigation of medicines in children. This document (European Agency for the Evaluation of Medicinal Products 1997) states:

there is a responsibility shared by applicants and the competent authorities to ensure that children have timely access to safe and effective medicines which have accurate, scientifically justified, prescribing information.

The problem has been addressed in the United States of America by establishing several paediatric research units to set up clinical trials to investigate drug use in children. It is anticipated that similar units may be developed throughout Europe.

Drug administration

In many situations in neonatal practice the choice of formulations available may be as important as drug selection. A dearth of suitable preparations available for administration to neonates can cause problems for the neonatal nurse. As a result of this there is an increased risk of medication errors taking place (Koren *et al.* 1986). Many drugs will need to be diluted prior to administration purely to allow accurate measurement of the required dose (Northern Neonatal Network 1998).

It is clear that dose selection is a complex issue in the neonate. It is important to remember that as well as the rapid changes that occur in renal and hepatic function the baby's weight can also change quite rapidly. The dose of a drug may need to be adjusted to take this into account (Walson *et al.* 1993). It is not sensible to adjust doses on a daily weight change but doses should be reviewed on a weekly basis depending on the drugs prescribed and the patient involved. Similarly, as the infant matures the dosage frequency may need to be increased, for example, with penicillin.

Particularly in babies who are very sick and requiring intensive care, vascular access and fluid volumes may become a problem. It may be necessary to co-infuse drugs. Although this should be avoided wherever possible, there will be situations where it is unavoidable. Some drugs can be given together via a Y-connector which is placed as near to the patient as is possible to minimise the contact time between the drugs (Trissel 1998; Zenk 1999). Drug compatibilities should always be confirmed with a pharmacist. If drugs are considered to be incompatible then they should be separated by a bolus injection of sodium chloride or glucose depending on the drugs involved. Compatibility of the drug with the infusate must also be established. Drugs should not be co-infused with blood or other blood products—for example, albumin and platelets.

The type of line used for access may influence how a drug is given. It may often be possible for a drug to be given in more concentrated solutions if it is administered via a central venous catheter than if it were given via a peripheral cannula. Concentrated glucose solutions (greater than 10%) should not be given via a peripheral cannula as there is a risk of phlebitis and extravasation injury (Duck 1997).

Conclusion

The use of medicines in the neonatal population is a complex issue and it is an area of understanding which is rapidly developing. Neonates need no longer be considered as 'therapeutic orphans', as they have been described in the past. Drug therapy is increasingly complicated and the neonatal nurse needs to have a good grasp of the basic pharmacokinetic principles to allow the safe administration of medicines and monitoring of the prescribed treatment. It is important, therefore, that there is a team approach to the use of medicines in neonates; the doctor, nurse and pharmacist all have a role to play.

Case study: prescription of medication for a baby born at 25 weeks' gestation

Baby Kim weighed 775 g when born at 25 weeks' gestation. Her mother had ruptured membranes at 21 weeks and received one course of dexamethasone. At birth the baby was floppy, bradycardic and gave a single gasp. She was ventilated and had one dose of surfactant.

Plan: FBC, blood culture. Double dose penicillin + gentamicin, im vitamin K.

Day 2: TPN ordered and enteral feeds started at 0.5 ml/h.

Day 3: Bilestained aspirates, feeds stopped. Penicillin stopped, amoxycillin and metronidazole added.

Day 4: Hypernatraemic dehydration, sodium=160 mmol/l.

Day 5: Abdominal distension, bilestained aspirates.

Further investigations revealed necrotising enterocolitis.

Q.1. What factors do you need to consider when prescribing medication for a baby born at 25 weeks' gestation?

Q.2. Which of the medicines prescribed warrant special monitoring? Would this present any problems?

Q.3. The baby suffered from hypernatraemic dehydration. Would this affect the likelihood of toxicity developing with any of the medication?

Q.4. Could any of the antibiotics prescribed be given orally? What are the important points to consider?

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Appendix

Normal values in the neonate

Listed below are the 'average' normal values expected in the first days of life. However, the infant's gestational age and day of life and clinical condition need to be considered before treatment is instigated.

	Term	Preterm
Temperature (°C)		
Rectal	36.5–37.2	36.5–37.2
Axillary	35.6–37.3	36.5–37
Abdominal skin	35.5–36.5	36.2–37.3
Apex beat (per minute)	100–160	120–175
Blood pressure (mmHg)		
Systolic	50–72	47–57
Diastolic	25–45	22–35
Mean	30–55	25–47
Blood		
pH	7.3–7.4	7.3–7.38
PCO_2 (kPa)*	4.5–6.0	4.5–6.5
PO_2 (kPa)	8.0–12	7.5–10

* To convert kPa to mmHg multiply by 7.5

Blood (cont.)	Term	Preterm
Bicarbonate	18–25 µmol/l	18–25 µmol/l
Base excess (mmol/l)	-7 to +3	-5 to +5
Creatinine (µmol/l)	37–113	76–156
Calcium (mmol/l)	2.1–27	1.9–2.7
Glucose (mmol/l)	2–5.5	2.6–5.5
Lactate (mmol/l)	0.5–1.4	0.5–1.4
Magnesium (mmol/l)	0.7–1.0	0.62–1.2
Phosphate (mmol/l)	1.7–2.6	1.8–3.2
Potassium (mmol/l)	3.5–5.5	4.6–6.0
Sodium (mmol/l)	135–146	135–146
Urea (mmol/l)	3.4–6.7	1.0–5.0
Haemoglobin (g/dl)	16.8–18.0	16.8–18.0
Packed cell volume	0.53–0.58	0.53–0.58
Platelets ($\times 10^9$/l)	150–400	150–350
Prothrombin time (seconds)	10–16	13–23
White cell count ($\times 10^9$/l)	9.0–35	9.0–35
Neutrophils	50–80%	50–80%
Lymphocytes	25–40%	25–40%
Monocytes	5–8%	5–8%
Urine		
Osmolality (mosmol/l)	100–300	100–300
Sodium (mmol/l)	<1	<20
Specific gravity	1.006–1.015	1.006–1.015
Cerebrospinal fluid		
Protein (mg/l)	30–250	100–300
Glucose (mmol/l)	1.5–5.5	1.5–5.5
NB: Glucose approximately 80 per cent of serum level		
Red cells (per mm^3)	0–50	0–300

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