Handbook of Clinical Anaesthesia

Fourth Edition

Edited by

Brian Pollard
Gareth Kitchen

CRC Press
Taylor & Francis Group

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For Claire, Mum and Dad, your unwavering support makes endeavours like this possible.
To my children, Joseph and Amelia – follow your dreams; anything is possible.

GK

For all of our patients. May this book improve both your safety and your experience of anaesthesia.

BJP

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Preface

Welcome to the fourth edition of the *Handbook of Clinical Anaesthesia*. We have retained the overall structure as in the first three editions. The book continues to be a collection of individual entries each covering a particular topic, condition or problem which may be encountered in clinical anaesthesia. The philosophy of the book has been retained in that all of the information is presented in a concise form without unnecessary information or 'padding'.

Over its lifespan between the first and the fourth editions, this book has undergone a significant evolution which we believe has served to improve it. The original idea was conceived by John Goldstone and Brian J Pollard in 1994. John unfortunately had to withdraw from the project at the second edition. For the fourth edition a second editor has been introduced again, Dr Gareth Kitchen. The choice of Gareth is clear. He is a young academic anaesthetist who has been able to instil new thoughts into the book and assist in driving it forwards and bringing on board a number of new names as experts in their fields.

In the first two editions, the authors of the various sections and monographs were drawn almost exclusively from the UK. In the third edition, the authorship was widened into a much more international field. In this fourth edition, we have returned to it being a UK-based field for the authors. Not only that but as we, the editors, are based in the Northwest, we have selected our authors principally from this area as there is a huge amount of expertise here.

Remember that this book is not an exhaustive treatise. It does not cover every eventuality; no book can do that. The *Handbook of Clinical Anaesthesia* is a distillation of facts and guidance and is intended to complement the major texts in the subject. Individual entries are referenced where appropriate but the references are limited to a small number of key sources and include up-to-date reviews wherever possible.

Over the years this book has proved popular with trainees preparing for examinations in the speciality. It has also proved very popular with established consultants and specialists who keep it beside the phone, on the office desk or in the operating theatre suite for straightforward advice on problems or situations encountered.

Finally, we would like to pay tribute to the many authors involved in the first three editions of this book. A significant proportion of their text and information has been retained where the advice has not materially changed. Many sections have nevertheless been rewritten as appropriate and updated as necessary. The authors involved in the first three editions are too numerous to mention but to each and every one we thank you for your input to the previous editions and hope that you approve of this new version and its updated information.

BJP and GK

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List of abbreviations

A&E	Accident & Emergency		ECT	Electroconvulsive therapy
AAGBI	Association of Anaesthetists of Great Britain and Ireland		EDV	End diastolic volume
			EEG	Electroencephalogram
ABG	Arterial blood gas		EF	Ejection fraction
ACE	Angiotensin converting enzyme		EMG	Electromyogram
Ach	Acetylcholine		ESA	European Society of Anaesthesiology
AChE	Acetyl cholinesterase		ESC	European Society of Cardiology
ACS	Acute coronary syndrome		ETT	Endotracheal tube
ACTH	Adrenocorticotrophic hormone		FBC	Full blood count
ADH	Antidiuretic hormone		FEV_1	Forced expiratory volume in 1 second
AF	Atrial fibrillation		FiO_2	Inspired fraction of oxygen
AHA	American Heart Association		FRC	Functional residual capacity
AKI	Acute kidney injury		FVC	Forced vital capacity
ALS	Advanced life support		GABA	Gamma hydroxybutyric acid
AMP	Adenosine monophosphate		GCS	Glasgow comas scale
APTT	Activated partial thromboplastin time		GFR	Glomerular filtration rate
ATP	Adenosine triphosphate		GH	Growth hormone
BIPAP	Bilevel positive airways pressure		GMP	Guanosine monophosphate
BMI	Body mass index		GP	General practitioner
BPM	Beats per minute		GTN	Glyceryl trinitrate
CABG	Coronary artery bypass graft		Hb	Hemoglobin
CAD	Coronary artery disease		HDL	High-density lipoprotein
CCF	Congestive cardiac failure		HDU	High dependency unit
CNS	Central nervous system		HIV	Human immunodeficiency virus
CO	Cardiac output		HOCM	Hypertrophic obstructive cardiomyopathy
CO_2	Carbon dioxide			
COPD	Chronic obstructive pulmonary disease		I/E	Inspired : expired ratio
CPAP	Continuous positive airways pressure		IABP	Intra-arterial blood pressure
CPET	Cardiopulmonary exercise test		ICP	Intracranial pressure
CRF	Chronic renal failure		ICU	Intensive care unit
CSF	Cerebrospinal fluid		IG	Immunoglobulin
CT	Computed tomography		IHD	Ischemic heart disease
CVA	Cerebrovascular accident		IM	Intramuscular
CVC	Central venous catheter		IPPV	Intermittent positive pressure ventilation
CVP	Central venous pressure			
CVS	Cardiovascular system		IQ	Intelligence quotient
CXR	Chest x-ray		IV	Intravenous
DBP	Diastolic blood pressure		JVP	Jugular venous pressure
DVT	Deep venous thrombosis		kPa	Kilopascal
ECG	Electrocardiogram		LBBB	Left bundle branch block

LDL	Low-density lipoprotein
LFT	Liver function test
LM	Laryngeal mask
LMA	Laryngeal Mask Airway ™
LMN	Lower motor neuron
MAC	Minimum alveolar concentration
MAP	Mean arterial pressure
MSH	Melanocyte stimulation hormone
NICE	National Institute of Health and Care Excellence
NIV	Noninvasive ventilation
NMJ	Neuromuscular junction
NO	Nitric oxide
NREM	Nonrapid eye movement
NSAID	Nonsteroidal anti-inflammatory drug
NSTEMI	Non-ST-elevation myocardial infarction
NYHA	New York Heart Association
O_2	Oxygen
P_{50}	Partial pressure of oxygen at 50% saturation
PaO_2	Arterial partial pressure of oxygen
PAC	Pulmonary artery catheter
$PaCO_2$	Arterial partial pressure of carbon dioxide
PAP	Pulmonary artery pressure
PEEP	Positive end-expiratory pressure
PEFR	Peak expiratory flow rate
PET	Positron emission tomography
PFT	Pulmonary function test
PONV	Postoperative nausea and vomiting
PT	Prothrombin time
PTH	Parathyroid hormone
PTT	Partial thromboplastin time
PVR	Pulmonary vascular resistance
RBBB	Right bundle branch block
REM	Rapid eye movement
SaO_2	Arterial saturation of oxygen
SAP	Systolic arterial pressure
SLE	Systemic lupus erythematosus
STEMI	ST-elevation myocardial infarction
SVR	Systemic vascular resistance
TB	Tuberculosis
TIA	Transient ischemic attack
TIVA	Total intravenous anesthesia
TNF	Tumor necrosis factor
TNM	Tumor nodes metastases
TOE	Transesophageal echocardiogram
TSH	Thyroid stimulating hormone
TTE	Transthoracic echocardiogram
U&E	Urea and electrolytes
UK	United Kingdom
UMN	Upper motor neuron
USS	Ultrasound scan
V/Q	Ventilation : perfusion
VC	Vital capacity
VF	Ventricular fibrillation
VT	Tidal volume
WHO	World Health Organization

PART

1

PATIENT CONDITIONS

Respiratory system

MATTHEW STAGG

1

ASTHMA

A very common respiratory disorder characterized by recurrent attacks of paroxysmal dyspnoea with reversible variable airflow obstruction and increased bronchial hyper-responsiveness to a range of stimuli. Aetiology, pathology and clinical presentation are heterogenous, but an underlying inflammatory response is usually present. There is an immense range of clinical pathology from children with reversible bronchospasm through to elderly patients in whom bronchospasm is superimposed on chronic respiratory disease. The incidence of intraoperative bronchospasm is low and tends to occur in older asthmatics and those with active or poorly controlled asthma at the time of operation.

EPIDEMIOLOGY

Variable geographical distribution, affecting about 5% of the population as a whole but up to 10% of children.

MORBIDITY

Increased risk of postoperative respiratory complications, especially in the older patient with chronic airways disease in whom cardiac problems may also be present.

Mediator	Bronchospasm	Oedema	Mucus secretion
Histamine	+	+	+
Prostaglandin	+	+	
Leukotrienes C4, D4, E4	+	+	+
Thromboxane	+	+	
Platelet activating factor	+	+	

PATHOPHYSIOLOGY

Nonspecific airway hyper-responsiveness is common. There is an increased response to methacholine, exercise, histamine, cold-air challenge, hyperventilation or extreme emotional stimulus. Airway obstruction is due to constriction of airway smooth muscle, mucus secretion and oedema of the airway wall. Mechanisms include neural and cellular pathway activation. The neural pathway involves afferent irritant receptors in airways, causing reflex stimulation of postganglionic parasympathetic fibres, resulting in smooth muscle constriction and mucus secretion. C fibre stimulation releases local neuropeptides; substance P changes membrane permeability and mucus secretion; neurokinin A causes bronchoconstriction. Cellular pathway activation involves immunoglobin E mediated histamine release from mast cells. Eosinophils, neutrophils, macrophages and lymphocytes CD8 and Th1 may also release mediators including leukotrienes, LTB4 and the cysteinyl leukotrienes, CysLT. LTB4 is a pro-inflammatory mediator with potent neutrophil chemotaxic properties while CysLTs are potent bronchoconstrictors that increase vascular permeability, cause mucus secretion, mucociliary dysfunction, stimulate eosinophil recruitment and increase bronchial responsiveness. At a cellular level, smooth muscle tone is controlled by intracellular cyclic AMP and possibly cyclic GMP, with lower levels leading to bronchoconstriction. The effect on ventilatory function can be extreme, with increased work of breathing, air trapping, exhaustion, hypercapnia and potentially fatal V/Q mismatch resulting in life-threatening hypoxia. This may be sustained for several days.

PREOPERATIVE ASSESSMENT

Optimise treatment in consultation with a respiratory physician. Severity and frequency of attacks, hospital admissions, exercise tolerance, current medication and trigger factors are essential information. Frequency of inhaler use may inform about severity and stability of their asthma. Steroid use, time of last exacerbation, timing and duration of any hospital admission are important.

Factors that indicate increased propensity to bronchospasm include recent or current upper respiratory tract infection, steroid use and past history of respiratory complications related to surgery. Previous ICU admission, especially one requiring intubation and ventilation, should act as a 'Red Flag'. In non–asthmatics, a family history of atopy or of asthma should alert to the possibility of intra-operative bronchospasm.

Some patients with COPD may have a significant reversible component. The presence of wheezes might indicate inadequate control and suggest medication review. The presence of a respiratory tract infection is a relative contraindication to anaesthesia.

INVESTIGATIONS

Chest X-ray – Look for hyperinflation; chronic lung changes or concomitant cardiac problems in older patients; evidence of right ventricular predominance, suggesting long-standing major problems.

ECG – Look for evidence of long-standing right ventricular hypertrophy or cor pulmonale. Such patients constitute a very high-risk group.

Lung function tests – FEV_1 reduced more than FVC (FEV_1 normally 50 mL kg^{-1}, and 70%–80% FVC).

Blood gases – Useful in asthmatics with COPD to be used as a baseline to guide postoperative target goals.

MEDICATION

The range of agents that can maintain control of asthma is considerable (Table 1.1). Many are long acting. Patients should continue on their maintenance therapy throughout their hospital stay if possible.

Preoperative management strategies.

Table 1.1 Agents used to maintain control of asthma

Drug type	Example agents	Side effects
Stabilizing agents	Sodium chromoglycate	
Bronchodilators		
β_2 agonist short-acting 4–6 h	Salbutamol/levosalbutamol	Tremor anxiety tachycardia
	Terbutaline	Hypokalaemia/hypomagnesaemia
β_2 agonist long acting >12 h	Arformeterol	Less side effects
	Salmeterol	No antiinflammatory action
Phosphodiesterases	Aminophyline	Tachycardia/arrhythmias
Inhaled steroids	Becotide, flixotide, budenoside	
Inhaled anticholinergics	Ipratropium	
Leukotriene antagonists	Zileuton montelukast pranlukast	
IgE immunotherapy	Omalizumab	
Others	Ketamine	Sympathomimetic
	Magnesium	Smooth muscle relaxation
Gases	Volatile agents	Bronchodilation
	Heliox	Reduced airway resistance
		$FiO_2 < 1$

Clinical	Preoperative intervention
Asymptomatic	Nothing needed
No medications	
No recent asthma episodes	
No obstruction on spirometry	
Occasional bronchodilators	Probably nothing needed
No steroids	Dose prior to induction suggested
Inhaled steroids	Continue inhaled steroids
	Give bronchodilator prior to induction
Spirometry below baseline	Consider short course oral steroids
Oral steroids	Continue same dose preoperatively
	Consider extra dose at induction
	Probable hydrocortisone postop for several days

PREMEDICATION

Sedation is useful as anxiety may provoke an attack in some patients. Atropine or glycopyrro-late inhibits vagally mediated bronchospasm but produces tachycardia. Preoperative bronchodilators and steroids reduce the likelihood of postoperative complication, so consider an additional dose of bronchodilator by inhaler or nebulizer prior to induction. Patients on steroids should receive steroids and if on high doses (>1500 μg day^{-1} in adults; less in children) give peri- and postoperative replacement as adrenal suppression may be present. Neither wound healing nor infection problems are relevant with these short periods of increased steroid use.

CHOICE OF ANAESTHESIA

Regional anaesthesia is recommended but anxiety can trigger bronchospasm so patient acceptance is important. If general anaesthesia is necessary, avoid stimulation of the respiratory tract and drugs known to cause bronchospasm.

INDUCTION

Avoid agents that may release histamine. Thiopentone is safe although it can cause histamine release and does not block airway reflexes. Propofol has bronchodilator properties and suppresses airway reflexes. Etomidate is safe. Ketamine is suitable for induction and maintenance by infusion in the asthmatic patient with bronchospasm requiring emergency anaesthesia,

although it may produce tachycardia and increased secretions. It may also be used to treat status asthmaticus. Sevoflurane is widely used and well tolerated.

INTUBATION

Spraying the larynx with lidocaine, prior to intubation, may help although it can itself stimulate bronchospasm (not histamine mediated). The use of a laryngeal mask avoids airway stimulation and the need for muscle relaxants. If control of hypercapnia, airway protection or emergency ventilation is required, an endotracheal tube is the only option.

MAINTENANCE

Halothane, enflurane, isoflurane and sevoflurane are all potent bronchodilators. They have been used in the treatment of refractory asthma and are ideal for maintaining anaesthesia.

MUSCLE RELAXANTS

Suxamethonium is a potent histamine releaser, so avoid if possible. Atracurium and mivacurium are associated with bronchospasm from histamine release. Pancuronium, vecuronium and cisatracurium appear safe while rocuronium has been associated with some severe reactions although in high does may be used as an alternative to suxamethonium. Reversal with anticholinesterases can trigger bronchospasm although atropine or glycopyrrolate given concurrently reduces the severity of this.

ANALGESIA

Local and regional techniques are recommended, but are not always feasible. Morphine and diamorphine release histamine and should be avoided. Pethidine has been widely used although may have some histamine-releasing potential. Fentanyl and alfentanil are safe. Exercise caution with NSAIDs unless previous exposure has not yielded problems.

POSTOPERATIVE MANAGEMENT

Problems in older asthmatics usually relate to underlying chronic lung disease. Effective analgesia assists physiotherapy and coughing so preventing the development of atelectasis and concurrent infection. Warm, humidified air and bronchodilators minimize the impact of mucus retention and plugging.

THE EMERGENCY CASE WITH CURRENT SYMPTOMATIC BRONCHOSPASM

A potentially disastrous situation, but fortunately rare. Surgery must be absolutely life or limb threatening to warrant proceeding. If possible, use a regional technique. Treat bronchospasm aggressively with IV steroids, magnesium sulphate 2 gm IV and/or aminophylline IV. The induction agent of choice is ketamine, followed by ketamine infusion, although other induction agents are often used effectively and safely. Suxamethonium may release histamine but its use may be difficult to avoid, unless high dose rocuronium is an option. Fentanyl is recommended for analgesia. Inhalational agents (sevoflurane or halothane) are effective in treating bronchospasm. Once deep on these agents, the patient may be better controlled than prior to induction. Continued bronchospasm with high airway pressure may necessitate IV beta agonists or even epinephrine (nebulizer or intravenously) in extreme circumstances.

Ventilation may pose problems, as airway pressures are likely to be high. Manipulate tidal volume, rate and I/E ratio to minimize peak airway pressure but maintain adequate minute ventilation. Permissive hypercapnia is reasonably tolerated. The possibility of a pneumothorax must be continuously considered. Postoperative management should be in ICU.

DEVELOPMENT OF INTRAOPERATIVE ASTHMA

Not all wheezing is asthma. Tube contact with the carina or a main bronchus can produce wheezing. Airway obstruction may result from tube blockage, secretions or blood. Aspiration, tension pneumothorax, anaphylactic or anaphylactoid reaction may all produce bronchospasm.

Salbutamol (2–5 $\mu g\ kg^{-1}$) or aminophylline (5 mg kg^{-1}) slow IV may be given. Steroids (e.g.

hydrocortisone) will not have an immediate effect but may assist in gaining control. Airway pressures may have been very high so beware of a pneumothorax.

The end of the case is a critical time when bronchospasm may appear in an awakening patient. Extubation deep may reduce the likelihood of bronchospasm but in many cases is inappropriate. Reversal with neostigmine can provoke bronchospasm but atropine or glycopyrrolate reduce the risk. Avoiding all reversal agents is ideal. Sugammadex may be a viable alternative.

DRUGS TO AVOID

Tubocurarine
Morphine, diamorphine and other histamine-
    releasing opiates
Beta blockers
Aspirin and other NSAIDs which are prostaglandin
    mediated

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BRONCHIECTASIS

Bronchiectasis is characterized by long-standing abnormal dilatation of bronchi with chronic inflammation. This chronic inflammatory process results in patients being extremely productive of sputum with a predisposition to either chronic infection or colonisation with intermittent acute episodes of infection.

Historically bronchiectasis was a consequence of chronic recurrent infection. Pneumonias, measles, whooping cough, TB and fungal infections were the main causes. Now with antibiotics, vaccination and better nutrition it is far less common. Cystic fibrosis and smoking are now the main causes. Sometimes patients will present for surgical treatment of their bronchiectasis. There are some specific associated syndromes including Kartageners (the combination of situs inversus, sinusitis and bronchiectasis).

Diagnosis is by high-resolution CT scan and anaesthesia for bronchography has been relegated to history.

PATHOPHYSIOLOGY

Following childhood pneumonia or recurrent adult infections.

Congenital:
    – Cystic fibrosis
    – Bronchial cartilage deficiency
    – Abnormal ciliary motility (Kartageners)
    – Hypogammaglobulinaemia
Distal to bronchial obstruction:
    – Inhaled foreign body
    – Tumour

Clinical features are variable. In severe bronchiectasis there is up to 500 mL of purulent sputum per day, which gets dramatically worse during an acute exacerbation. Other features include haemoptysis from areas of severe inflammation with altered local circulation arising from bronchial and intercostal arteries. In long-standing disease pulmonary hypertension and cor pulmonale may develop. Metastatic abscess formation can occur. Amyloidosis is a rare complication.

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MANAGEMENT

Chest physiotherapy with percussion and postural drainage is key but early intervention with antibiotics may prevent acute exacerbations. These patients are often chronically colonised with resistant organisms due to frequent antibiotic exposure. *Pseudomonas aeruginosa* and *Haemophilus influenzae* are particularly common.

PREOPERATIVE ASSESSMENT

Exercise tolerance (compared with their usual state), sputum production and frequency of acute exacerbations predict the severity. Information about colonising organisms and antibiotic history are important.

INVESTIGATIONS

Blood gases – To determine present baseline, and to guide postoperative target goals.

Chest X-ray – Probably not of benefit. A recent CT scan is helpful.

Pulminary function tests – Generally not very helpful.

ECG – Look for signs of right ventricular strain or cor pulmonale.

Echocardiogram – Helpful in assessing right ventricular hypertrophy, myocardial function and raised pulmonary pressures.

PREOPERATIVE MANAGEMENT

The patient will need extensive physiotherapy and be exacerbation free prior to surgery. Discussion with chest physician and microbiologist should determine the appropriate antibiotic to use preoperatively.

ANAESTHETIC MANAGEMENT

The surgery will determine the most appropriate form of anaesthesia. If possible, use regional techniques. Use routine monitoring commensurate with the anaesthetic and surgery. Have a very low threshold for an arterial line.

There are no particular agents that are contraindicated. Try to keep the oxygen saturations high (>90%) to maintain a safety margin. End tidal CO_2

is likely to be different from the arterial value but should provide trend measurements.

Sputum retention is likely to be a problem and will predispose to secondary infection. Humidify all gases and persist with regular tracheal suction. It may be necessary to use a bronchoscope to remove inspissated secretions and sputum. In cases with very severe localised bronchiectasis, it may be feasible to try to isolate that part of the lung with a bronchial blocker.

Proper attention to sterile technique is important, particularly in those with Kartageners syndrome as they also have a defect in neutrophil chemotaxis. Nasal tubes should be avoided in view of the accompanying sinusitis.

POSTOPERATIVE CARE

Arrange early postoperative physiotherapy in advance. In cases of cystic fibrosis, HDU care may be helpful to ensure mobilization and physiotherapy. Good analgesia is essential and patient-controlled devices, epidural analgesia or NSAIDs are all useful. Entonox may be helpful. Avoid postoperative ventilation wherever possible.

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Preoperative assessment – specific medical
problems, Chapter 25
Cardiopulmonary exercise testing, Chapter 25

BRONCHOGENIC CARCINOMA

Lung cancer is the most common cause of cancer mortality worldwide for men and women, causing approximately 1.2 million deaths per year (Table 1.2). The most common symptoms are unexplained persistent cough, haemoptysis, shortness of breath, chest pain, bone pain and weight loss. They may develop from airways or parenchyma.

The main types are *non-small cell lung carcinoma* (NSCLC) and *small cell lung carcinoma* (SCLC). Early stage (stage 1–2) NSCLC is treated with surgery, while SCLC is treated by chemotherapy and radiation. Other tumours including large cell, neuroendocrine (carcinoid), bronchioloalveolar and rarer forms can all present as lung malignancies. The most common cause is long-term exposure to tobacco smoke. Lung cancer in non-smokers (15% of cases) is often attributed to a combination of genetic factors, radon gas, asbestos, air pollution and passive exposure to cigarette smoke.

Derived from the epithelium, squamous cell carcinomas are the most common NSCLC. They are usually centrally located at the carina or in the 1–3rd generation bronchi. Adenocarcinoma is less common with peak incidence in men in their fifties.

Presentation includes airway obstruction, lung collapse, and distal infection or through spread via the peribronchial tissues with subsequent invasion of the mediastinum. It spreads by both lymphatic and haematological routes and distal metastasis is common in liver, adrenals, bone and brain.

All forms of treatment can be associated with notable toxicity. Patients with significant impairment due to their lung cancer or comorbid conditions may not be fit to undergo resection or even aggressive chemoradiotherapy. Performance status can be assessed by a variety of methods including the Karnofsky Performance Status (KPS) or the World Health Organisation (WHO) status.

Anaesthetic involvement is mainly for lung resection (e.g. lobectomy, pneumonectomy). However, newer indications for palliative interventional bronchoscopic procedures are increasing. Debulking/disobliteration of central symptomatic obstructive lesions followed if necessary by tracheobronchial stents can ameliorate some symptoms of advancing disease. This may be done by rigid or flexible bronchoscopy, using a number of different modalities such as electrocautery, laser, cryotherapy/cryoextraction, argon plasma coagulation or mechanical debulking.

PREOPERATIVE ASSESSMENT

Patients may be asymptomatic or may present with a range of symptoms and signs including:

Local – Chest pain, cough, dyspnoea, haemoptysis, hoarseness, pleural effusion.
Distal – Metastases with associated problems.
Other – Ectopic hormonal activity from paraneoplastic tumours (e.g. ACTH, PTH, ADH, insulin and glucagon). Some manifestations of Cushing's syndrome can occur with hypokalaemia although the full clinical features of Cushing's syndrome are rarely seen as they do not have time to develop. Lambert–Eaton syndrome has been reported. Serotonin secreting adenomas may present as episodic sweating, wheeze and

Table 1.2 Lung cancer and its incidence

Characteristic	Squamous cell (epidermoid)	Adenocarcinoma	Large cell	Small cell
Approximate incidence	25%–30%	30%–35%	15%–20%	20%–25%
5-year survival	25%	12%	13%	1%
Operability	43%–50%	35%	35%–43%	Rare
Potential for metastasis	Low to moderate	Moderate	Moderate	High
Response rate to systemic treatment	Low	Low	Low	Moderate

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breathlessness. These patients are usually smokers and COPD is a common concomitant problem.

INVESTIGATIONS

- *Chest X-ray* – May not reveal the tumour but may show signs of concomitant problems such as COPD. A pleural effusion or pericardial effusion would suggest mediastinal invasion.
- *ECG* – Thoracic surgery can result in rhythm disturbance, especially atrial fibrillation. Smokers have a high incidence of asymptomatic heart disease.
- *Electrolytes* – May indicate ectopic ADH secretion with low sodium which will eventually produce clinical signs of confusion and weakness. Ectopic ACTH secretion can result in hypokalaemia or hyperkalaemia with or without hypernatraemia. PTH produces hypercalcaemia but so do widespread bony metastases with elevated alkaline phosphatase. Glucose values can be adversely influenced by ectopic insulin or glucagon.
- *Lung function tests* – Important if any significant lung resection is planned. FEV_1 and FVC are most useful, whilst low gas transfer (below ~30%) may have implications for risk of postoperative respiratory failure. CPET may be helpful and baseline arterial blood gases on air should be taken.

Patients will have likely presented through a lung multidisciplinary team. A chest CT and or CT-PET scan, and tissue sampling by bronchoscopy, transbronchial needle aspiration, mediastinoscopy or interventional radiology will have staged the disease enabling appropriate management.

INOPERABILITY

The TNM staging system of the international union against lung cancer (Table 1.3) will determine which primary lung cancers are theoretically operable. In general, stage 1 and 2 disease is operable. Some classical indicators of inoperability exist which indicate stage 3 or 4 advanced disease. These include SVC obstruction or other great vessel involvement, nerve palsies including left recurrent laryngeal and phrenic nerve damage, carinal or tracheal involvement, oesophageal invasion, vertebral involvement and Pancoast's syndrome.

Pancoast's syndrome is an apical carcinoma invading the eighth cervical and first thoracic nerves. Severe pain and wasting in the upper limbs occur with stellate ganglion involvement. The patient often has Horner's syndrome (ptosis, enophthalmos, miosis, impaired sweating on face).

Very often these patients have palliative stents placed for debulked endobronchial disease or symptomatic compressive extrinsic disease. They can be silicon or metallic-nitinol alloy (placed via rigid bronchoscopy or interventional radiology) requiring general anaesthesia. Nitinol bronchial stents can be placed via flexible bronchoscopy under general anaesthesia or conscious sedation, or through endobronchial tubes. Complications include migration, misplacement, infection, biofouling and stent fractures (in older generation stents). These procedures usually offer immediate relief of symptoms and at least short-term benefit in the acute setting. They have even been attributed to liberation from mechanical ventilation after acute respiratory failure.

PREOPERATIVE PREPARATION

Optimize respiratory function – beta 2-adrenergic agonists, anticholinergics, active physiotherapy and steroids as indicated.

Any sizeable effusions should be drained. Electrolytes and haemoglobin should be corrected. While a restrictive approach to transfusion should be adopted, these patients are at risk of ischaemic heart disease so aim for Hb > 10 g/dL.

In patients having debulking techniques or stenting, careful consideration of anatomical placement of the stent should be discussed with the operator prior to anaesthesia. Modern imaging provides useful information that often correlates with functionality. Patients will often be dyspnoeic and may have partially collapsed lung segments. They are usually dramatically improved by the procedure but if the collapse has been long-standing it may be

Table 1.3 TNM staging system for lung cancer (7th edition)

Primary tumour (T)

T1	Tumour ≤3 cm diameter, surrounded by lung or visceral pleura, without invasion more proximal than lobar bronchus
T1a	Tumour ≤2 cm in diameter
T1b	Tumour >2 cm but ≤3 cm in diameter
T2	Tumour >3 cm but ≤7 cm, or tumour with any of the following features:
	Involves main bronchus, ≥2 cm distal to carina
	Invades visceral pleura
	Associated with atelectasis or obstructive pneumonitis that extends to the hilar region but does not involve the entire lung
T2a	Tumour >3 cm but ≤5 cm
T2b	Tumour >5 cm but ≤7 cm
T3	Tumour >7 cm or any of the following:
	Directly invades any of the following: chest wall, diaphragm, phrenic nerve, mediastinal pleura, parietal pericardium, main bronchus <2 cm from carina (without involvement of carina)
	Atelectasis or obstructive pneumonitis of the entire lung
	Separate tumour nodules in the same lobe
T4	Tumour of any size that invades the mediastinum, heart, great vessels, trachea, recurrent laryngeal nerve, oesophagus, vertebral body, carina, or with separate tumour nodules in a different ipsilateral lobe

Regional lymph nodes (N)

N0	No regional lymph node metastases
N1	Metastasis in ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes, including involvement by direct extension
N2	Metastasis in ipsilateral mediastinal and/or subcarinal lymph node(s)
N3	Metastasis in contralateral mediastinal, contralateral hilar, ipsilateral or contralateral scalene, or supraclavicular lymph node(s).

Distant metastasis (M)

M0	No distant metastasis
M1	Distant metastasis
M1a	Separate tumour nodule(s) in a contralateral lobe; tumour with pleural nodules or malignant pleural or pericardial effusion
M1b	Distant metastasis

Stage groupings

Stage IA	T1a–T1b	N0	M0
Stage IB	T2a	N0	M0
Stage IIA	T1a,T1b,T2a	N1	M0
	T2b	N0	M0
Stage IIB	T2b	N1	M0
	T3	N0	M0
Stage IIIA	T1a,T1b,T2a,T2b	N2	M0
	T3	N1,N2	M0
	T4	N0,N1	M0
Stage IIIB	T4	N2	M0
	Any T	N3	M0
Stage IV	Any T	Any N	M1a or M1b

irrecoverable and predispose to infection. Careful planning is required.

PREMEDICATION

Minimise stress to the patient with an anxiolytic if necessary. A drying agent may help.

ANAESTHETIC TECHNIQUE

In patients with tracheal or bronchial compromise, coughing may become problematic and threaten airway patency. Inhalational techniques are likely to precipitate problems. General anaesthesia with muscular relaxation and mechanical ventilation is usually required. Almost any induction technique is suitable. Short- to medium-acting relaxants which do not accumulate are ideal with neuromuscular monitoring. Volatile agents are bronchodilators. Some advocate the use of heliox during induction if there is significant airway narrowing. Remifentanil has been recommended.

For lung resection, a double lumen endotracheal tube allows single lung ventilation and optimises surgical field. Alternatively an endobronchial blocking balloon may be placed under bronchoscopic vision.

Partial or complete central airway obstruction or symptomatic trachea-broncho-oesophageal fistulae can sometimes be palliated by debulking and/or stenting, respectively. Stents require appropriate and careful planning regarding position, size and type. Bronchial stents may be deployed awake or under general anaesthesia. Rigid bronchoscopy with a Sanders injector is a well-established technique, as is the suspension laryngoscope. Remember that the Sanders injector can result in high pressure air trapping if there is partial obstruction. Adequate neuromuscular reversal is vital prior to extubation. At the end of the case ensure there is a good cough reflex.

PATIENTS WITH PREEXISTING STENTS NEEDING ANAESTHESIA

Ensure the stent position is known, image if possible, seek an opinion from whoever placed the stent and ideally view the stent bronchoscopically prior to intubation The aim is to avoid dislodging the stent. In an emergency, try to visualise it before intubation if possible.

ACUTE POSTOPERATIVE CENTRAL AIRWAY OBSTRUCTION

It may be difficult to reestablish spontaneous breathing. The appearance has been likened to inadequate neuromuscular reversal with an ineffective breathing pattern that is largely abdominal. Desaturation ensues often associated with a deteriorating level of consciousness which may be in part due to hypercarbia. Blood gases show hypercarbia and hypoxia. Assume airway obstruction. Control the airway and with the aid of a surgeon go to rigid bronchoscopy as secretions at the carina or in the trachea are the most likely cause. The differential diagnosis is tension pneumothorax after airway instrumentation but that is very rare from stent placement in experienced hands.

POSTOPERATIVE CARE

This depends on the nature of the surgery, requirement for ventilation, preoperative respiratory function and other co-morbidities. Even without ventilation, these patients will often require specialist postoperative care.

Epidurals, oral opioids and PCA are most commonly used for analgesia. Pain will impair chest movement so good analgesia is key to recovery.

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Pneumonectomy, Chapter 15
One lung anaesthesia, Chapter 28
Intraoperative bronchospasm, Chapter 30
Preoperative assessment of pulmonary risk,
    Chapter 25
Cushings syndrome, Chapter 6
Myasthenia, Chapter 3

CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

A common chronic inflammatory disease of the lungs, there is a spectrum associated with expiratory airflow obstruction including emphysema and chronic bronchitis. It has pulmonary and systemic manifestations. Management guidelines are well established with evidence-based recommendations for chronic disease and acute exacerbations.

In 1990, COPD was ranked 12th as a burden of disease by the WHO; by 2020 it is projected to rank 5th. Cigarette smoking is its primary cause and up to 25% of smokers are likely to develop COPD.

Respiratory disease accounts for more than 25% of acute hospital admissions, of which more than half are acute exacerbations of COPD. Hospitalization carries up to 26% mortality, rising to 66% within 2 years.

The prevalence of COPD is 5%–10% among general surgical patients, 10%–12% in cardiac surgery and 40% in thoracic surgery as compared to 5% in the general population.

The pulmonary component of COPD is characterised by expiratory airflow limitation that is not fully reversible. The diagnosis, severity assessment and monitoring rely heavily but not exclusively on spirometry. In smokers, lung function decline is accelerated beyond the natural 20–30 mL annual loss.

Airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung. Respiratory failure in COPD may be type 1 (predominantly hypoxic) or type 2 (associated hypercapnia). Chronic respiratory failure is often related to chronic hypoventilation, and may lead to cor pulmonale if untreated.

Patients with COPD, particularly severe disease, are at significant risk of postoperative complications. Preoperative recognition and optimisation can reduce these risks.

PATHOPHYSIOLOGY

Inflammatory small airways disease, destruction of alveolar units, inflammatory bronchiolitis and excess mucus production lead to airflow obstruction. Airways are no longer held open due to reduced elasticity and tone of the parenchyma. The combination of airway collapse prior to full emptying, bronchospasm and secretions produces expiratory airflow limitation and gas trapping. Loss of alveolar units decreases gas transfer.

An early manifestation is an increase in residual volume. The natural history is progressive gas trapping with decreasing vital capacity (VC). This results in a decline in forced expiratory volume in 1 second (FEV_1), further exacerbated by rapid shallow breathing, which leads to dynamic hyperinflation (Figure 1.1).

This increase in work of breathing is in part responsible for dyspnoea and exercise limitation. Due to differential transluminal pressures within the small conducting airways and at the alveolar level, lung units have different time constants for emptying. Across the whole lung, this results in retained intrathoracic pressure and so called 'intrinsic PEEP'. In the longer term, an increase in residual volume and chronic hyperinflation with reduced efficiency of resting diaphragm position and function results. In those with chronic carbon dioxide retention, there is a predisposition to developing pulmonary hypertension and right heart failure (cor pulmonale). Associated conditions are malnutrition, musculoskeletal disorders, cardiovascular disease,

Expiration

Hyperinflated chest wall

Reduced respiratory excursion

Active forced expiration

Increased pressure on bronchiole from increased intrathoracic pressure

Incomplete emptying

Increased residual volume

Intrinsic PEEP

Cor pulmonale
Pulmonary hypertension

Secretions

Loss of rigidity in wall
Less elasticity
Collapsing bronchioles

Transmural pressure favours collapse

Flat less efficient diaphragm

Figure 1.1 Respiratory effects of COPD during expiration.

diabetes and depression. Reduced weight, peripheral muscle strength and chronic sputum production portend a worse prognosis.

Ultimately, the final common pathway of decreased gas transfer, alveolar hypoventilation and respiratory muscle disadvantage produces ventilation/perfusion mismatch (V/Q) resulting in hypoxaemia and/or hypercarbia.

There is no clear correlation between lung function and blood gas features. Patients with hyperinflation, and resting tachypnoea, often have low arterial oxygen tensions and low gas transfer, but do not retain carbon dioxide on increasing oxygen therapy – these tend to be the emphysema spectrum patients. Conversely, it is the chronic bronchitic patients who tend to hypoventilate, are comfortable at rest, have chronic ventilatory failure with stable hypercapnia, but better spirometry, less gas trapping and preserved gas transfer that are at risk of hypercapnic narcosis with high inspired oxygen levels.

PREOPERATIVE ASSESSMENT

The risk of postoperative pulmonary complications and postoperative respiratory failure is high while

lesser complications such as atelectasis or infection are common.

HISTORY

Enquire about:

- Exercise tolerance, breathlessness, orthopnoea, sputum productivity.
- Exacerbations requiring noninvasive ventilation, oral steroids, hospital and ICU admissions.
- Symptoms of sleep disordered breathing (excessive daytime sleepiness or tiredness, snoring and witnessed apnoeas).

EXAMINATION

Look for:

- Hyperinflation
- Right heart dysfunction
- Incipient infection in the oropharynx
- Wheezes or rhonchi (correlate with complications so consider bronchodilators or steroids to eliminate wheezing)
- Ischaemic heart disease

There is a significant risk of sudden death in uncontrolled heart disease. Heart failure, in particular, is a prognostic indicator with a 30% five-year survival rate. The exclusion and treatment of reversible ischaemia is paramount. The use of beta-blockers is controversial because of the risks of bronchospasm. A discussion between cardiologist and respiratory physician should determine the benefit risk ratio. A cardioselective beta blocker combined with inhaled steroid/bronchodilator may be indicated. Optimise statin and anti-platelet treatment.

INVESTIGATIONS

- Blood tests according to local guidelines.
- FBC to look for polycythaemia.
- Respiratory function tests. Compare current values with pre-existing results to identify any deterioration. Peak flow, FEV_1 and mid-expiratory flow rates are useful as a marker of the severity of limitation when considered with exercise tolerance. The residual volume to total lung capacity ratio is a useful indicator of gas trapping and potential surrogate of dynamic hyperinflation when approaching 50%. Reduced gas transfer, particularly kCO, may be indicative of emphysema.
- Blood gases will give the normal values for the individual, and the likelihood of chronic carbon dioxide retention, with high bicarbonate levels.
- ECG is essential. Include exercise testing to identify reversible ischaemia, and echocardiography if concerns of secondary pulmonary hypertension associated right heart dysfunction or cor pulmonale exist.
- Sleep studies should be considered if an element of sleep apnoea is suspected.

PREOPERATIVE OPTIMISATION

1. *Timing*

   Unless surgery is urgent, time is helpful in improving the preoperative state. Involve a chest physician and investigate cardiovascular disease.

2. *Stop smoking*

   Current smokers are at greater risk of complications. Smoking should be stopped at least eight weeks before surgery. There is some evidence to suggest that cessation or reduction <8 weeks before surgery increases the risk of complications.

3. *Optimise drug treatment*

   Most patients have some reversibility of lung function, or functional improvement with bronchodilators – refer to NICE guidance. The use of short-acting beta agonists, with long-acting muscurinic agonists is now routine – it reduces exacerbations and improves quality of life. When FEV_1 is less than 60% predicted and associated with two or more exacerbations per year, then an inhaled steroid/long acting beta agonist combination is recommended. Oral mucolytics are used as adjuncts in chronic deteriorating disease, whilst oral methylxanthines do not have a favourable evidence base and are no longer advised in acute or chronic settings, mainly due to increased risk of arrythmias.

   Oral steroids, for a minimum of a week, are known to reduce the duration of exacerbations, reduce reattendance rates after hospital admissions, and prevent admission at the sign of infection, when combined with antibiotic in those with severe disease. Surgery should be delayed if possible.

   A few days before surgery, in those with severe disease, a short course of oral steroids can be considered, if there are no objections related to surgical wound healing. Special care with diabetic patients is advised. An alternative is IV hydrocortisone at induction. Nebulised bronchodilator therapy should be given perioperatively. The role of the nebulised mucolytic N acetylcysteine in the perioperative or postoperative setting is not established. It is considered as an adjunct to physiotherapy in those with retained mucus and limited expectorating ability. It should be used with bronchodilators because of a risk of bronchoconstriction.

4. *Preoperative physiotherapy*

   Important for airway clearance. Continue postoperatively to reduce retained sputum and segmental collapse. In severe disease, noninvasive ventilation may be considered in the

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postextubation period together with breaks for airway clearance.

5. *Pulmonary rehabilitation*

Exercise tolerance and lung function are improved up to 6 months after completion. There is also emerging data to suggest improvements with pulmonary rehabilitation after exacerbations. Its value in the shorter term is not clear.

6. *Thromboprophylaxis*

These patients have an increased risk of venous thromboembolism, so appropriate thromboprophylaxis is important, as well as early mobilisation and appropriate hydration.

REGIONAL ANAESTHESIA

Regional anaesthesia circumvents many problems. The patient must be able to tolerate lying relatively flat. Position, procedure and duration are important. These patients are often dependent on abdominal excursion and have prolonged active expiration when breathing normally and so regional techniques that extend as high as T8 may be problematic. Exercise care when using interscalene blocks as the potential for phrenic nerve and diaphragm palsy exist.

GENERAL ANAESTHESIA

Indications include major or prolonged procedures where regional or other techniques are inadequate or inappropriate, the need for muscle relaxation and when the patient's condition necessitates ventilation. Laryngeal mask ventilation is being increasingly used to preserve laryngeal reflexes, particularly relevant to the need for effective postoperative airway clearance.

At induction attempts to modify the bronchoconstrictive effects of intubation include the local application of lidocaine or the use of beta sympathomimetics. Use drugs unlikely to cause histamine release or exacerbate bronchospasm – e.g. propofol, thiopentone ketamine and etomidate. The muscle relaxant used should also be chosen carefully. Morphine may release histamine and also may have long-acting sedative properties. Fentanyl or the use of regional or central analgesic blocks, with

or without infusion catheters, may be preferred. In severe COPD, doses of all drugs should be tempered by the predisposition of these patients to cardiovascular instability. Inhalational agents have good bronchodilating properties except desflurane which may provoke coughing, bronchospasm and tachycardia. The beneficial effects may, however, be offset by a delayed recovery. TIVA may be considered. As optimal mobility and coughing is important, a technique that results in a rapidly awake alert and comfortable patient has advantages.

MONITORING

ROUTINE MONITORING TO AAGBI STANDARDS

Additional monitoring depends on magnitude and type of surgery. An arterial line is recommended both for pressure monitoring and for repeated blood gases.

IPPV may cause air trapping and increase intrathoracic volume. Careful monitoring of ventilator parameters is therefore important. The increase is unpredictable as is the consequent increase in intrinsic PEEP. This may impede venous return and hence cardiac output. Raised pulmonary hypertension associated right heart dysfunction is a theoretical risk, and may manifest as rhythm changes, as a result of left ventricular impairment. BIPAP, reduced frequency rates and long expiratory times, with 'permissive hypercapnia' may be considered to minimise dynamic hyperinflation and its cardiovascular impact.

Capnography is essential as it will clearly show if the CO_2 trace does not reach a plateau. This indicates ongoing incomplete emptying of alveoli. There will be a large difference between end tidal and arterial CO_2. If air trapping is occurring, there will be some degree of intrinsic PEEP. Ventilators that can measure intrinsic PEEP are useful.

The use of extrinsic PEEP is controversial. It may increase air trapping. Alternatively it may splint airways open reducing trapping and reducing the inspiratory effort to reopen collapsed bronchioles.

The use of bronchodilators should be considered intra-operatively if difficulties in ventilation arise.

Lung volume reduction surgery in this population has specialist anaesthetic implications beyond the remit of this chapter.

POSTOPERATIVE CARE

These patients pose difficult postoperative management problems and pulmonary complications are common. The risk of postoperative respiratory failure (>48 h mechanical ventilation) is 3%–3.5%. The presence of severe COPD increases the risk 1.5 times, whilst lesser complications such as atelectasis or infection are more common. These are more likely in thoracic or head and neck procedures than abdominal. Tissue trauma, fluid shifts and blood transfusion are risk factors. Population factors for adverse outcome include age >70 years, ASA ≥ 3, smoking and congestive cardiac failure. The 30-day mortality rate after postoperative respiratory failure is 26%.

ANALGESIA AND PHYSIOTHERAPY

Postoperative needs include good deep breathing, coughing and early mobility but too much sedation will impair these activities and may be detrimental. An epidural may be useful for abdominal or thoracic surgery but high epidurals may embarrass breathing. There are also risks from co-morbidities such as arrhythmias; atrial fibrillation is common. On the second and third days postoperatively there are often recurrent hypoxic episodes that have been attributed to pharmacologically disturbed sleep patterns. Later complications include ileus and pseudo ileus; the resultant splinting of the diaphragm from a distended abdomen can be dangerous.

Nasogastric decompression of the stomach is important, particularly if NIV or CPAP are used, and if thoracic or abdominal surgery has been performed.

Incentive spirometry or intermittent positive pressure breathing are important adjuncts to physiotherapy.

POSTOPERATIVE MONITORING

The main problems are iatrogenic respiratory depression, sputum retention and respiratory failure. While some problems are immediate many occur in the days following surgery. Knowledge of normal and abnormal respiratory patterns (in particular, either fast and shallow or very slow) is crucial as they are early warning signs. Oxygenation is important and easily tracked with pulse oximetry but CO_2 is probably more important and hence an arterial line is helpful. Body temperature should be maintained as hypothermia may induce ischaemia.

OXYGEN THERAPY

Most patients are not hypoxic drive dependent and hypoxia is a greater threat than hypercarbia. In patients with chronically elevated CO_2 who are hypoxic drive dependent, too much oxygen may result in hypercarbia and narcosis. Recent work suggests that saturations of 90% or just above are likely to be safe. It is prudent to pursue a safe target rather than limit oxygen and risk hypoxia. Patients with chronically elevated CO_2 and high bicarbonate tend to be at more risk of hypercarbic narcosis. They appear comfortable at rest, not hyperinflated, with relatively preserved spirometry, gas transfer and less gas trapping.

NONINVASIVE VENTILATION AND CPAP

The aims are lung volume recruitment and maintenance of that state while normal spontaneous ventilatory function and airway clearance mechanisms are restored in the postoperative period.

Noninvasive positive pressure ventilation is the treatment of choice for AECOPD associated with hypercapnic respiratory failure not requiring emergency intubation. It reduces the risks of deteriorating respiratory failure, mechanical ventilation, infectious complications, length of hospital stay, and death, and is health economical. It is also an important weaning tool in mechanically ventilated COPD patients on ICU. Whilst its role in the postoperative period is not yet defined, it is intuitive and common to have it available for use immediately after extubation in the anesthetised patient with COPD who is at risk of postextubation compromise.

In COPD patients with associated obstructive sleep apnoea, access to their home device perioperatively is advisable. In those with suspected but undiagnosed OSA-COPD overlap, postoperative bilevel NIV with higher levels of expiratory positive airway

pressure (EPAP), which is akin to CPAP, should be used. Both treatments aid breathing and improve oxygenation, as there is an increased risk of profound postoperative desaturations in these patients, as a result of the residual effects of sedation and sleep deprivation on hypoxic arousal mechanisms.

OUTCOMES

Complications, especially pulmonary, are common. The hypercapnic group has significantly impaired function as they cannot easily clear CO_2 and may have altered drive. It is not always the disease state but comorbidities and the nature of the surgery that define outcome.

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Intraoperative bronchospasm, Chapter 30
Preoperative assessment – specific medical problems, Chapter 25

CYSTIC FIBROSIS (CF)

Cystic fibrosis is the most common genetic Caucasian disease with an incidence in northern Europeans of about 1 in 3000 births. The gene involved encodes CF trans-membrane conductance regulator protein (CFTR). It functions as a chloride channel on the apical border of epithelial cells lining most exocrine glands and affects many transport systems including sodium, ATP channels, intracellular vesicle transport and bicarbonate-chloride exchange which is critical to mucin structure and activities. There have been at least 1500 mutations identified that affect CFTR function in a variety of ways, but the genotype is a poor predictor of disease severity and outcome.

Diagnosis is usually made in infancy and the sweat test is easy and reliable. A chloride concentration greater than 60 mmol/L is diagnostic. With improved intensive management of affected individuals, the median age of survival is now 38 years.

There are often some very clear 'red flags' for the diagnosis, although clinical presentation can be very varied and non-specific so a high index of suspicion should always occur. Table 1.4 identifies the common symptoms. In infancy and childhood, gastrointestinal problems are common such as meconium ileus, intussusception and pancreatic insufficiency. Respiratory problems are slightly later and infections commence during childhood. Later in childhood and adulthood the full panoply of gastrointestinal, respiratory and renal manifestations may be seen. The respiratory problems (as summarised in Table 1.5) are chronic infection, with recurrent acute exacerbations leading to bronchiectasis, and chronic colonisation often with resistant organisms. Pseudomonas is particularly likely to develop in the uncleared plaques of mucus, especially with impairment of the normal mechanisms that inhibit bacterial binding to epithelium combined with faulty immunological responses to the bacteria, which then goes on to form resistant biofilms. Airway inflammation is a notable finding. An allergic response to aspergillus fumigatus occurs in some patients.

Diabetes is a common endocrine problem, associated with many pancreatic exocrine functions.

Despite this plethora of problems, modern treatment is continually improving. Nebulised hypertonic saline, macrolide antibiotics, beta agonists and ibuprofen are useful in disease management. Hypertonic saline helps by pulling fluid into the airways and helps hydrate the peri-ciliary layer and improve mucociliary clearance.

PREOPERATIVE ASSESSMENT

Patients are always under the care of a specialist unit and always have insight and are well informed about their disease state. Ask about the normal level of function and exercise capacity, whether there are any current infective problems, cough, sputum quality and quantity or wheezing. It is important to be cognisant of pancreatic and bowel dysfunction but also any endocrine problems such as diabetes.

Key features are

- Current chest status of the CF
- Exercise tolerance
- Recent hospitalisation
- Current or recent antibiotics, including any intravenous antibiotics

Table 1.4 Clinical manifestations and surgical presentation

Infancy	Childhood	Adulthood
Infection	Sinusitis	Haemoptysis
	Polyposis; polypectomy	Pneumothorax
	Allergic aspergillosis	Infection
	Intravenous access difficulties	Sinusitis and nasal polyposis
		Allergic aspergillosis
		Need for lung transplant
Meconium ileus/peritonitis; intestinal atresia	Distal intestinal obstruction	Biliary fibrosis; obstructive jaundice – need for cholecystectomy
	Intussusception	
Pancreatic insufficiency	Biliary fibrosis	Cirrhosis; varices, coagulopathy
Rectal prolapse	Hepatic steatosis	Distal intestinal obstruction
	Malabsorption	Adenocarcinoma bowel
	Diabetes	Diabetes
Hyponatraemic hypochloraemic alkalosis	Renal calculi	Renal calculi
	Hyponatraemic hypochloraemic alkalosis	Renal failure
Dehydration		Hyponatraemic hypochloraemic alkalosis
		Vasculitis
		Hypertrophic pulmonary osteoarthritis
		Osteoporosis, fractures

Table 1.5 Respiratory pathophysiology

Reduced mucociliary clearance	Physiotherapy, multidisciplinary team care.
Mucus plugging	Mucolytics
Atelectasis	
Colonisation; *Pseudomonas*, *Staphylococcus*, *Haemophilus*, *Stenotrophomonas*, *Burkholderia cepacia* and *Aspergillus*	Aerosolised antibiotics, such as tobramycin, colistin. Targeted treatment of acute infection
Obstructive airway pattern reduced FEV_1, reduced peak flow and increased residual volumes	Beta adrenergic agents. Oral or inhaled steroids
Bronchiectasis, emphysema, fibrosis	
Apical blebs – pneumothorax	
Pulmonary hypertension	Oxygen therapy
Cor pulmonale	NIV/CPAP/BiPAP

INVESTIGATIONS

Chest X-ray looking for hyperinflation, extent of bronchovascular markings and evidence of cysts or bronchiectasis. A CT scan may be more informative.

Lung function tests may show an obstructive pattern.

Blood gases if indicated. As the disease progresses, chronic hypoxia and hypercapnia predispose to raised pulmonary artery pressures and vascular resistance which leads to right ventricular strain and cor pulmonale. These patients may require home oxygen or may be on NIV. This needs to be known so that access to their devices postoperatively is possible.

Renal and liver function should both be checked as these may deteriorate insidiously. In advanced disease, there may be abnormal clotting.

PREOPTIMISATION

Engage physiotherapists who will have a plan to ensure the patient is as good as they can be – they and the patient will know. Request physiotherapy immediately prior to going to theatre. Bronchodilators, steroids as required and hydration are all important. Current antibiotics or those recommended by microbiology for the surgery.

Bowel preparation to avoid constipation. H_2 antagonists or similar as reflux is common.

Plan the anaesthetic technique to suit the surgery. Use regional anaesthesia where possible either as the entire technique (difficult in children), or as an adjunct to general anaesthesia so emergence is rapid

and pain free at the end of surgery. Try to have minimal impact on respiratory function and also plan to be able to commence physiotherapy immediately postoperatively if possible. Use humidified gases and care should be taken with any nasal tubes as most patients have hypertrophic sino-nasal mucosa with or without polyps.

Monitoring should be appropriate to fit the surgery and the patient. If there is evidence of pulmonary hypertension or impaired myocardial function, invasive monitoring may be appropriate. Watch the airway pressure as it may be an indicator of plugging or collapse. End tidal CO_2 and oximetry are both useful but may need to be supplemented by arterial blood gases and, if diabetic, the blood sugar should be monitored. In neonates, transcutaneous monitors can be used.

INDUCTION

If the patient has reflux a rapid sequence technique should be used. Pre-oxygenation followed by propofol as it wears off rapidly. Some anaesthetists will avoid nitrous oxide as there is a small risk of pneumothorax, and a high FiO_2 is often required. A volatile agent that is non-irritant is ideal. Sevoflurane has the advantage of bronchodilation and will also facilitate intubation where a minimal but adequate dose of a non-histamine releasing relaxant such as vecuronium or cisatracurium can be used.

Positive pressure ventilation is usually not a problem unless there is very severe disease. Suctioning

may be necessary to clear the secretions perioperatively. Intraoperative physiotherapy may be useful on occasions. Only extubate when the patient will breathe well and be able to cough as avoiding atelectasis is important. Prior to extubation, instillation of saline may be helpful for the physiotherapy following extubation.

POSTOPERATIVE MANAGEMENT

Rapid emergence and good analgesia, with a combination of opioids, NSAIDs and local anaesthesia where appropriate will enable early physiotherapy and mobilisation. These patients are at high risk of postoperative complications particularly pulmonary complications from sputum retention, plugging and consequent atelectasis. An enhanced recovery area is ideal unless more intensive monitoring and care is needed. If necessary, CPAP and noninvasive ventilation may be required. Positive pressure ventilation can produce significant problems in these patients with barotrauma and a tendency to air trapping, detrimental changes in V/Q and increasing dead space so it is best avoided if possible.

Proper hydration and opiate-sparing techniques may avoid the complication of distal intestinal obstruction.

PREGNANCY

The normal physiological changes of pregnancy, such as increased minute ventilation and oxygen requirements, may stress respiratory function while fluid shifts may exacerbate problems with right ventricular strain. Prognostic factors include weight gain <4.5 kg, FVC <50%, colonisation with *B. cepacia*, frequent respiratory infections and hospitalisations, diabetes and pancreatic insufficiency. If there is evidence of cor pulmonale, this is likely to get much worse with pregnancy and there is a recognised mortality.

Regional techniques are clearly preferable, in particular combined techniques where a good block can provide postoperative analgesia. There are circumstances in patients with severe disease where this may not be feasible but the decision to use general anaesthesia should not be taken lightly in

particular if it may exacerbate the incipient respiratory infection and failure.

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CROSS-REFERENCES

RESTRICTIVE LUNG DISEASE

A range of conditions produces a restrictive picture on lung function with reduced total lung capacity, reduced resting volume yet often with normal airways resistance and airflow.

Restrictive lung diseases may be classified as intrinsic or extrinsic. Intrinsic restriction is characteristic of a group of over 200 diverse conditions affecting the pulmonary interstitium (i.e. the space bounded by the alveolar epithelium and the pulmonary capillary bed and including the perivascular and perilymphatic tissues) and encompassed

by the term diffuse parenchymal lung disease (DPLD) (Figure 1.2). These are usually characterised by impaired gas transfer factor and reduced gas transfer coefficient (Kco), as a result of impaired exchange between alveolar–capillary units within the interstitium.

The most commonly encountered DPLDs in clinical practice are the so-called idiopathic interstitial pneumonias (IIP), which separate into idiopathic pulmonary fibrosis (IPF) (previously known as cryptogenic fibrosing alveolitis), and the non–IPF diseases, which generally have a better prognosis. Other DPLDs are subclassified as granulomatous, exposure related (organic or inorganic), drug and radiation induced, associated with collagen vascular or rheumatological diseases, pulmonary-renal and vasculitides, and rare orphan diseases (e.g. histiocytosis X, lymphangioleiomyomatosis)

Extrinsic restriction of lung function is usually associated with reduced gas transfer but normal or increased gas transfer coefficient corrected for lung volume (Kco). Essentially, the reduced lung volumes are due to limited excursion of the chest wall, pleura or neuromuscular impairment of the respiratory system. Note that left ventricular

dysfunction is also a cause. Table 1.6 summarises the main causes of intrinsic and extrinsic restrictive lung diseases.

PATHOPHYSIOLOGY

The volume of the functional residual capacity (FRC) is determined by the balance of inward elastic recoil of the lungs and outward elastic recoil of the chest wall. Impairment of either will restrict movement and result in a lower FRC. Total thoracic compliance is the combined compliance of lung and chest wall which is reduced. Particularly in advanced DPLD, there is V/Q mismatch and oxygen transfer reduction, leading to hypoxaemia. This is often apparent earlier following exercise.

The restrictive nature of the system means that smaller tidal volumes necessitate a higher respiratory rate to maintain effective minute ventilation and acid base homeostasis. This is generally true in intrinsic disease but extrinsic restrictive conditions such as neuromuscular disease or obesity have a propensity to respiratory muscle fatigue and alveolar hypoventilation, which may over time lead to type 2 or hypercapnic respiratory failure and pulmonary hypertension. The efficiency of ventilation is reduced
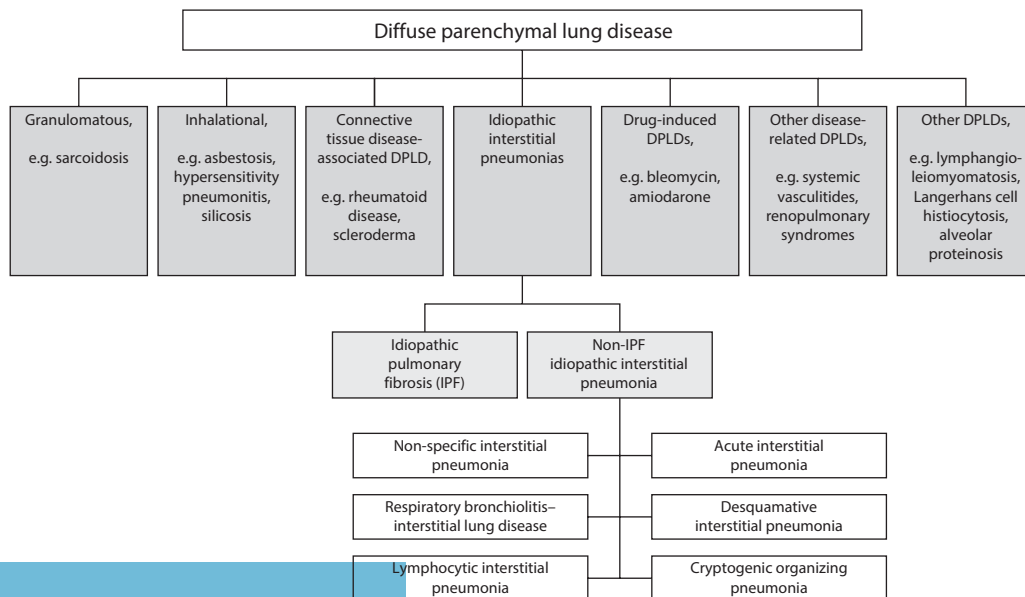

Figure 1.2 A classification scheme for diffuse parenchymal lung disease (DPLD).

Table 1.6 Restrictive lung diseases

Type	Mechanism	Condition
Intrinsic	DPLD	See Figure 1.2
Extrinsic	Limitation of chest wall excursion	Kyphoscoliosis, ankylosing spondylitis, thoracoplasty, pleural effusion, obesity
	Respiratory muscles/neuromuscular	Polio, Guillain–Barre, muscular dystrophy
	Pleural thickening	

by the smaller volumes as the effective dead space rises in relation to the tidal volume. The underlying disease process will further add to lung dysfunction.

ANAESTHESIA

The problems posed are the restricted lung volumes which reduce the ability of the lung to respond to stress. There is limitation of gas transfer and a predisposition to infection. Compliance is reasonable over a limited range of lung volumes above which it reduces dramatically so ventilation must remain within these limited volumes.

PREOPERATIVE PREPARATION

It is important to elicit the underlying cause of the restrictive picture.

HISTORY

With likely DPLD, exertional breathlessness, cough and reduced exercise tolerance may be apparent depending on the type and severity of disease. The history should elucidate the exercise tolerance and the degree of dyspnoea at rest and on exercise. Features of pulmonary hypertension and right ventricular failure such as ankle oedema may be present. Viral prodromal-like respiratory illnesses often characterise the clinical history and may be difficult to distinguish from respiratory tract infections. The past medical history should identify disorders associated with DPLD (e.g. rheumatoid arthritis and connective tissue disease). Radiotherapy for breast or thoracic malignancy can result in pulmonary fibrosis. Patients with a past history of granulomatous disease, e.g. ulcerative colitis, are at increased risk of developing sarcoidosis.

Chemotherapy such as bleomycin and other drugs such as amiodarone, methotrexate, gold, and homeopathic or complementary medications can cause DPLD.

Occupational history of exposures (organic and inorganic), and systemic features that may indicate connective tissue, vasculitis or rheumatological disease should be determined.

With suspected extrinsic diseases, weight-related problems, sleep-disordered breathing, left ventricular failure and neuromuscular weakness should be asked about.

EXAMINATION

Assess the degree of dyspnoea, look for cyanosis and evidence of finger clubbing (indicative of idiopathic pulmonary fibrosis). Look for features of systemic disease such as Raynaud's or polyarthropathy. There may be fine bilateral 'velcro-like' crackles heard on auscultation. Evidence of pulmonary hypertension, right heart dysfunction (i.e. loud pulmonary second heart sound, tricuspid regurgitation, raised jugular venous pressure [JVP] and ankle swelling), oropharyngeal indicators of sleep apnoea and left ventricular dysfunction should be excluded.

INVESTIGATIONS

Chest X-ray often shows a reticulonodular appearance, with characteristically small lung fields. The distribution of changes is indicative of the aetiology. Upper zones are associated with granulomatous or acute exposure-related DPLD. Lower zone predominance is usually related to the idiopathic interstitial pneumonias. Honeycombing and loss of clarity of the heart borders is generally a sign of advanced disease.

High-resolution CT scan is the diagnostic investigation of choice in suspected DPLD. The patterns of distribution of ground glass, interstitial thickening, traction bronchiectasis, and consolidative conglomerates are often sufficient to allow diagnosis without need for lung biopsy. However, this is usually in the context of secure clinical features and ultimately the profile of longitudinal functional behaviour.

Respiratory function tests show decreased vital capacity and FEV_1 so the ratio remains normal. FRC is reduced. The carbon monoxide diffusing capacity (DLco) is reduced in intrinsic lung disease as is the gas transfer coefficient Kco. Progressive decline in DLco (<40% predicted) is an independent predictor of poor prognosis in idiopathic interstitial pneumonia. Preserved or high Kco associated with low DLco is evidence of extrinsic disease. In neuromuscular disease, maximum inspiratory pressures (both volitional 'sniff' and nonvolitional diaphragm studies) are dramatically reduced. The vital capacity (VC) is a helpful serial measure of progression of DPLD (especially if >10% change). In neuromuscular weakness, a serial fall in VC may warrant a discussion about assisted ventilation in the acute or postoperative setting.

In patients with coexistent emphysema, lung volumes may be preserved. A mixed obstructive/restrictive defect may sometimes be seen in sarcoidosis, lymphangioleiomyomatosis (LAM), respiratory bronchiolitis interstitial lung disease (RB-ILD) and hypersensitivity pneumonitis.

Arterial blood gases may show hypoxaemia. CO_2 rises in extrinsic disease and sometimes with advanced DPLD.

Exercise tests such as the 6 minute walk test are useful in IPF. Desaturation to 88% or 200 m portend a poor prognosis. Exercise tolerance is reduced so exercise testing with oximetry will indicate oxygen requirement and can be used to follow disease progression.

PREOPERATIVE OPTIMISATION

Reverse any airflow limitation with bronchodilators; steroids may be needed. Treat cardiac failure appropriately well in advance of surgery. Treat any possibility of infection. If there are limiting factors such as pleural effusions, then drainage may be very helpful.

Involve the physiotherapists. A cardiological opinion is essential. If pulmonary hypertension may be present, perform echocardiography.

THE ANAESTHETIC

Plan the anaesthetic in terms of the procedure and the limitations of the patient. Consider if the procedure is amenable to regional technique. If a regional technique is used, then beware the height of the block may impair ventilatory muscle function, both chest and abdomen, so not above a level of T10 depending on the patient.

GENERAL ANAESTHESIA

Some advocate anticholinergic agents. Monitoring should encompass oximetry, capnography and the ability to do blood gas sampling. Cardiovascular monitoring will be defined by the cardiovascular stability of the patient and the nature of the procedure. In patients with kyphoscoliosis or ankylosing spondylitis, difficult intubation should be anticipated. A further problem in these patients with chest wall abnormalities is surgical positioning.

Ventilation may be difficult. Small tidal volumes will be necessary and if exceeded can result in very high airway pressures and a risk of pneumothorax. Oxygenation may also be problematic despite ventilation so high inspired oxygen may be necessary.

POSTOPERATIVE MANAGEMENT

As with other severe lung diseases, the postoperative period is a potential source of problems. Sputum retention and basal atelectasis will both contribute to the restrictive picture and may have significant effects on the already poor lung function. Extubate when compliant and awake so that coughing, mobilisation and physiotherapy are possible early. Adequate analgesia is essential with the usual difficult balance between analgesia and sedation. A high dependency area is ideal postoperatively.

Beware of hypoxia and of insidious hypercapnia. Noninvasive ventilation, either CPAP or bilevel, can be used to facilitate postoperative lung volume recruitment and relieve the work of breathing as necessary.

The physiotherapists are key to the management for several days postoperatively until the patient is fully mobile. Adjuncts like incentive spirometry, intermittent CPAP or intermittent positive pressure breathing may be helpful as bridges to recovery.

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SARCOIDOSIS

Sarcoidosis is a systemic, granulomatous disease of unknown aetiology. It seems likely that the granulomas form through an interaction between antigens, as yet unknown, and T cells. It has geographical variation. Slightly more common in women, its peak onset is in the twenties and thirties. Presentation is variable but 90% of patients have lung involvement, often with bilateral hilar lymphadenopathy or pulmonary infiltrates. Skin, lymph node, eye and liver are the next most affected organs in that order.

Cardiac involvement is less common but potentially fatal. There may be radiological appearances, particularly involving the small bones of the hand and feet, or symmetrical arthritis of large joints. Occasionally there is neurological involvement.

While imaging and a plethora of tests can imply sarcoid, such as elevated ACE levels, a raised calcium, raised immunoglobulins and 'gallium lit' lesions, the only real way to diagnose the condition is by biopsy which will show noncaseating granulomas. TB and fungal infection are often the differential diagnosis.

The implications to the anaesthetist mainly relate to the cardiac and pulmonary involvement which may involve fibrotic lung changes and a restrictive pattern usually with reduction of diffusing capacity. Most patients will have an abnormal chest X-ray at some stage in the disease and usually hilar lymphadenopathy. Occasionally there may be obstructive lesions in the airways themselves.

There may be nasopharyngeal and laryngeal involvement affecting the arytenoids and supraglottic area and patients occasionally present with dysphonia, then stridor and dyspnoea which may necessitate emergency tracheostomy.

Cardiovascular involvement is an uncommon manifestation in clinical practice at 2%, but 25% of postmortem examinations of known cases of sarcoid have cardiac involvement. Preferential granulomatous involvement of the conduction system is manifested as a variety of dysrhythmias, including complete heart block. Congestive cardiac failure with features of a dilated cardiomyopathy may also be present.

Renal involvement is uncommon at less than 2%. It may occur through hypercalcaemia or nephrocalcinosis or both. It may also cause either interstitial or membranous nephritis.

Hepatic and pancreatic involvement have been reported.

The neurological system may be affected in 5%–15% of patients with sarcoid although, again, the postmortem evidence suggests far more. Most common are cranial nerve palsies, which account for 65% of the neurological manifestations. Headache is also common but fitting is uncommon. Rarely, mono- or polyneuropathies can develop which may cause sensory or motor deficit, or a combination of both. Cerebellar symptoms can also occur.

Neuropsychological disturbance is also uncommon. Spinal involvement is rare but may present with various forms of paresis including cauda equina syndrome.

PREOPERATIVE ASSESSMENT

In such a protean disease it is hard to suggest a universal approach. A clear history of the range of problems that are known should be elicited but awareness of occult cardiology and neurology should be borne in mind.

The prevalence of respiratory system involvement indicates careful respiratory assessment. Specific attention is focused on a history of stridor (suggesting laryngeal involvement), swallowing difficulties (hinting at neurological problems), or any breathlessness indicating the more common interstitial-type lung disease. A chest X-ray and blood gases will be useful to identify any overt respiratory issues. Pulmonary function tests may help clarify degrees of restrictive lung injury defects. The covert nature of cardiac involvement mandates taking a history of any palpitations or fainting episodes, an ECG to look for any signs of actual or potential heart block, and echocardiogram to assess cardiac function. Pacing may be indicated.

Renal function is relatively easy to assess as is the measurement of calcium looking for hypercalcaemia. Neurological involvement needs to be elicited especially if it is intended to use a regional technique. Table 1.7 summaries the most likely pre-operative findings.

PERIOPERATIVE ANAESTHETIC MANAGEMENT

Given the massive range of potential problems that may be an issue, it is vital to tailor the anaesthetic to the patient. If feasible, the use of a regional technique may be advantageous in the presence of significant respiratory disease but may be difficult if there is neurological involvement. Laryngeal involvement with stridor is a special case that needs careful planning. The most common problem generally is the respiratory system. As with other respiratory conditions,

Table 1.7 Investigations and results

Investigations	Results (if system involved)
Chest X-ray	Bilateral hilar lymphadenopathy, reticulonodular shadowing, pleural effusions, cardiomegaly, atelectasis
Electrocardiogram	Conduction defects, ventricular hypertrophy
Arterial blood gases	Reduced Po_2 on room air
Lung function tests	Restrictive/obstructive defects
Electrolytes	Raised calcium/potassium
Echocardiography	Ventricular hypokinesis, mitral valve involvement, septal thickening and bright echoes (consistent with fibrogranulomatous infiltration)

caution with sedation is advised. Avoiding and preventing hypoxia is the aim and the liberal use of supplemental oxygen is recommended. These patients may already be on steroids, but if not, steroids may be of benefit.

POSTOPERATIVE MANAGEMENT

If the predominant area of risk is respiratory, the focus should be on good analgesia, mobilisation and physiotherapy. Renal issues may need attention to avoiding prerenal insults. With such a myriad of presentations and potential problems, there is no specific area in which sarcoid is different from its component parts. The role of steroids and other agents needs careful attention as these may need to be continued and probably increased in the postoperative phase.

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CROSS-REFERENCE

ANAESTHESIA AND SLEEP APNOEA SYNDROME (SAS)

In obstructive sleep apnoea (OSA), breathing during sleep is periodically interrupted by closure of the upper airway for 10–45 second intervals. This partial obstruction results in periods of reduced ventilation. Most individuals with OSA have a combination of apnoeas and hypopnoeas and respiratory effort-related arousals. The result is fragmented sleep which leads to excessive daytime sleepiness, fatigue, or poor concentration. Partners comment on apnoeas, snoring, restlessness and resuscitative snorts.

Grading of severity is based on the frequency of apnoeas and hypopnoeas per hour (apnoea/hypopnoea index [AHI]) and consequent symptoms. Alternatively, the frequency of dips in oxygenation during sleep is used (oxygen dip rate). AHI 5–14 correlates with mild symptoms, AHI 15–30 moderate and AHI > 30 severe.

Sleep apnoea can also be defined as obstructive (cessation of flow in the presence of respiratory effort), central (no flow and no effort) or mixed (a combination of the two).

Risk factors for OSA include obesity, craniofacial abnormalities and upper airway soft tissue abnormalities. Potential risk factors include heredity, smoking, nasal congestion and diabetes mellitus.

SAS affects 2%–4% of middle aged males and 1%–2% of adult females. Only 20%–30% of affected individuals have currently been diagnosed in the UK and the prevalence is increasing with increasing obesity.

Patients are at increased risk for organ system dysfunction and impaired neurocognitive performance due to chronic nocturnal hypoxaemia and repeated arousals over months and years.

An increased incidence of cardiovascular and cerebrovascular events and an emerging association with endocrine abnormalities including diabetes and sex hormone dysfunction exists due to chronic hypoxaemia-related microvascular dysfunction. Patients with SAS have a 7–10 times increased chance of road traffic accidents compared with other drivers.

Patients with OSA have an increased risk of peri- and postoperative complications. Prolonged apnoeic events may follow reduced consciousness due to iatrogenic loss of protective arousal mechanisms that overcome upper airway obstruction. Severe respiratory complications and unexpected deaths have occurred. The possibility of REM rebound-associated nocturnal desaturations, perhaps after hospital discharge, increases further the risk of unrecognised or untreated OSA. Preoperative diagnosis and optimisation are important. Three scenarios are identifiable:

- Known SAS on treatment
- Known SAS non-compliant with treatment
- Undiagnosed SAS

This last group, particularly who may not have typical features of SAS, can present with difficult airways, difficult intubation, or hypoxaemia postoperatively. Patients with clinical features suggestive of SAS and those with unexplained hypoxaemia, polycythaemia or pulmonary hypertension warrant diagnostic sleep testing. It is likely that the majority of patients with SAS are undiagnosed or untreated. The prevalence of SAS is thought to be very high in the morbidly obese, perhaps up to 70%. However, factors predicting this have poor specificity and not all patients in this category have SAS.

The most effective treatment for symptomatic SAS is CPAP which improves symptoms and reduces

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cardiovascular comorbidity in those using it effectively. However, adherence remains a challenge in more than 30%–40% of patients. They should be managed as part of a specialist sleep clinic.

OBSTRUCTIVE SLEEP APNOEA

Breathing is normally a function of generating a negative pressure in the thorax and entraining air. Narrowing of the airway associated with a tendency for tissues to collapse inwards are the key features for obstruction. The three areas in the pharynx subject to collapse are the retropalatal pharynx, the retroglossal pharynx and the retroepiglottic pharynx. Narrowing may be due to excess soft tissues in obesity and OSA correlates with neck circumference.

Narrowing of the airway produces increased turbulence and local tissue vibration while increased velocity through narrow passages generates a Bernoulli effect pulling tissues inwards (see Figure 1.3). When conscious, muscle tone prevents this but a reduction in muscle tone, through sleep or sedatives, may be all that is required for loss of a patent airway. In those predisposed to OSA, normal nocturnal tone and function of the dilator muscles is impaired. This may be due to both turbulent flow and a deficiency of orexins (neurohormones that govern wakefulness).

REM sleep is associated with decreased muscle tone which recovers as the patient wakes and then returns as they fall asleep again. There are usually four periods of NREM sleep which culminate in an episode of REM with its marked slowing of the EEG. Drugs that influence muscle tone either through peripheral or central actions may increase obstruction. Predisposing factors to the development of SAS are summarised in Table 1.8.

CENTRAL SLEEP APNOEA

This is a control effect and so is associated with conditions affecting ventilatory drive, e.g. neuromuscular disorders or neurological damage of the respiratory centre through stroke or head injury. Obesity may also affect the chest wall and reduce lung volume. Sedatives or opiates, which inhibit central control, will worsen these effects.

The sequence of physiological events that follows an obstructive apnoea is

- A fall in oxygen tension.
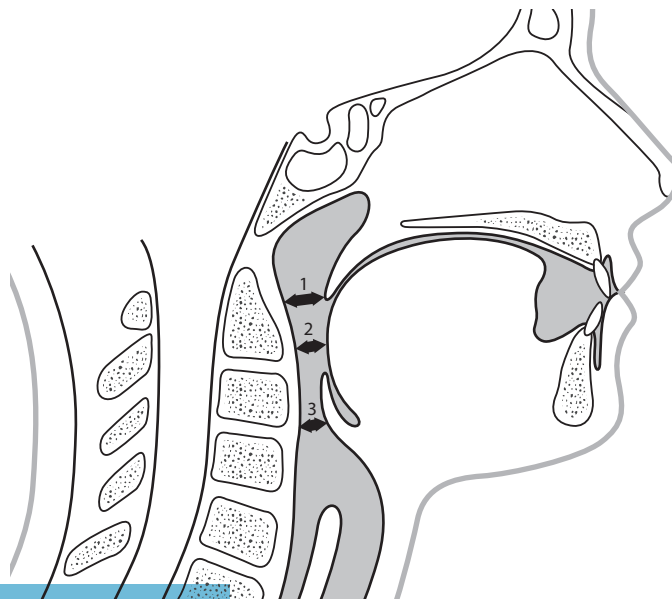- A rise in carbon dioxide tension.

Figure 1.3 Airway obstruction in sleep apnoea. 1, Nasopharynx – tensor palatine; 2, oropharynx – tongue enlargement and posterior tissues; 3, laryngopharynx – tissues around epiglottis and base of tongue.

Table 1.8 Predisposing conditions for SAS

Condition	Mechanism
Obesity; acquired; genetic (Prader–Willi syndrome)	Soft tissues, anatomical
Smoking	Pharyngeal inflammation and oedema
Nasal obstruction	Polyps, septal deviation, nasal congestion
Laryngeal tracheal obstruction	Laryngo/tracheo malacia
Neuromuscular; cerebral palsy, stroke, head injury	
Endocrine; thyroid Cushings acromegaly	Soft tissue
Connective tissue; Marfans syndrome	Upper airway tissues
Mucopolysaccharidoses	
Craniofacial; Pierre Robin, Treacher-Collins, Downs	

- Increasing ventilatory effort.
- Increasing negative inspiratory airway pressure.

All four mechanisms trigger 'reflex' activity, increase EEG activity (seen as twitching, movement, etc.) and cause arousal.

Immediate effects during an apnoea include:

- Low PaO_2 associated with tachycardia or bradycardia.
- Associated nocturnal angina, and myocardial infarction.
- Diurnal pulmonary and systemic hypertension.

Consequences of SAS include excessive daytime sleepiness, impaired concentration, mood changes, morning headache, waking with a choking sensation and dry mouth. Signs include snoring, excessive daytime sleepiness, nocturnal sweating and witnessed apnoea. Secondary effects include polycythaemia, pulmonary hypertension and right heart failure. In patients with excess weight there are likely to be significant comorbidities. Other nonspecific effects include gastro–oesophageal reflux, hypertension, ischaemic heart disease and in diabetics increased instability. There is an increased incidence of sudden death in untreated patients with SAS, compared with age-matched controls.

SLEEP, ANAESTHESIA AND APNOEA

REM sleep is the time with the most influence on sleep apnoea. In neonates it accounts for up to 50% of sleep while by middle age it is about 20%. It diminishes with age or with medications such as antidepressants.

THE PERIOPERATIVE IMPACT OF SLEEP APNOEA

Repetitive episodes of upper airway obstruction during sleep, with sleep disruption, hypoxaemia and autonomic arousals, contribute to cardiovascular risk. Anatomic narrowing in the pharynx due to excess tissue, tonsillar hypertrophy or craniofacial variations can lead to airway difficulties. Desaturations of sufficient intensity may precipitate arrhythmias or acute coronary syndrome in susceptible individuals. The CNS depressant effects of sedatives, analgesics and anaesthetics suppress the natural arousal mechanism induced by hypoxaemia or hypercapnia in patients with SAS leading to prolongation of postoperative apnoeic episodes. Another potential concern is the impact of restoration of sleep after a period of perioperative sleep deprivation. The phenomenon of rebound REM sleep, with its associated profound desaturations may be under-recognised in the postoperative period.

Although no prospective randomised trials of anaesthetic risk in patients with SAS exist, there are reports of postoperative cardiac arrhythmias, myocardial infarction, cerebrovascular events and hypoxaemia-induced organ dysfunction. There have been sporadic reports of fatalities in patients with OSA in the postoperative period.

THE EFFECT OF SEDATION AND ANAESTHESIA ON PATIENTS WITH SLEEP APNOEA

The effects of sedatives and sedating analgesics (e.g. opioids) mimic those on the upper and lower

respiratory tracts in sleep. There is a reduction in FRC and atelectasis. This has potential implications for preoxygenation. In patients with OSA associated with obesity, this reduced pharyngeal anatomical space, together with the functional disturbance of the dilator muscles (particularly genioglossus), is accompanied by a reduction in lung volumes as a result of fat distribution around the diaphragm in central obesity. This may reduce the traction on the pharynx exerted by the trachea. The usual neural mechanisms in wakefulness, to compensate for these anatomical imbalances, are lost during sleep. The pharynx is more susceptible to closure in these patients, potentially exacerbating the upper airway risk.

Sedatives reduce the phasic activity of pharyngeal muscles just prior to inspiration, mimicking the response to REM in patients with OSA. Thus, during general anaesthesia, there is a loss of the protection against upper airway collapse (caused by the lower respiratory tract muscles generating negative pressure on the airway). Moreover, sedatives depress the compensatory arousal responses to hypoxia,

hypercapnia and upper airway collapse that characterise the repeated sleep/wake cycle in OSA (Figure 1.4). The risk of prolonged apnoeas and desaturation then increases, as has been noted in many patients with OSA undergoing sedation.

In the postoperative period, residual central depressant effects of these agents may cause prolonged apnoeas and desaturation when reduced monitoring is present. Disruption of sleep has also been documented in postoperative periods following surgery. Thus, reduced total sleep time with less REM and non-REM slow wave sleep are reported, which may take several days to return to normal.

ASSESSMENT

History

SAS is still predominantly under-recognised. Look for a history of snoring, daytime sleepiness or lethargy and witnessed apnoeas. Identify other features such as depression, neurocognitive or functional decline. Seek a corroborative history from a partner,
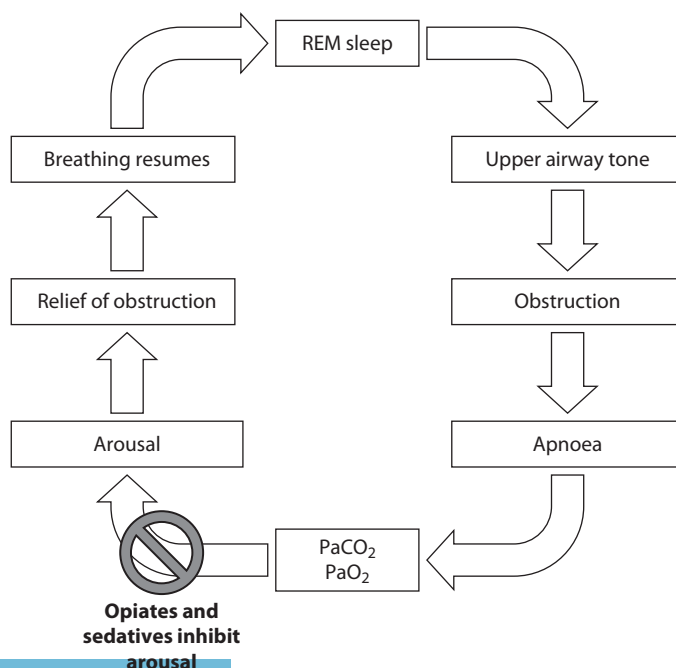

Figure 1.4 The pathophysiology of obstructive sleep apnoea and how sedatives can suppress the natural arousal responses of hypoxaemia and hypercapnia. REM, rapid eye movement.

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as patients will often deny symptoms due to insidious onset or being unaware of their own sleep disturbance. Identify comorbidities (e.g. hypertension, diabetes mellitus) and risks (skilled mechanical work, HGV driving, pilots, etc.). The Epworth sleepiness scale is a self-reported indicator of sleepiness, when other confounders to adequate sleep are excluded (e.g. prostate problems, symptomatic nocturnal acid reflux, noise or light disturbance). It has 8 questions of different situations each with a weighted scale of 0–3 of likelihood of dozing off. A score >10/24 indicates a pathological reason for excessive sleepiness. However, it does not correlate well with severity of SAS. Other questionnaires have been validated in different settings such as the Berlin and STOPBANG scoring models.

Physical examination

Look for signs of excess soft tissue, retrognathia, short distance between hyoid and mandible, crowded oropharynx, kissing tonsils, or thick uvula. Look for signs of undiagnosed or undertreated hypothyroidism, pulmonary hypertension, polycythaemia due to chronic hypoxaemia or right heart failure.

Investigations

If surgery is not urgent, then investigation of severity and associated comorbidity should be undertaken. Referral to a sleep clinic requires overnight polysomnography with sleep staging. However, a limited respiratory multichannel study without sleep staging can be performed at home, and is diagnostic when the clinical probability of SAS is high. Information it provides includes the number, duration and severity of obstructive events and levels of desaturation, average, nadir and duration. Heart rate, ECG, actigraphy (movement as a surrogate for wakefulness) and position are also available. In the absence of other confounders such as underlying lung disease, the characteristic episodic desaturation pattern of overnight oximetry is sufficient for a diagnosis of OSA.

If OSA is diagnosed some advocate using CPAP preoperatively to 'train' the patient for its use postoperatively. There is also some evidence from dynamic imaging to suggest a remodelling of the upper airway after 8–12 weeks of effective use of CPAP. In long-term CPAP use, they should bring their own devices into the hospital for use pre- and postoperatively.

The risk of postoperative problems in patients with SAS having peripheral surgery is twice that of normal patients.

ANAESTHESIA

SAS is a risk factor for a difficult airway and difficult intubation. Large tongue, limited mouth opening, large tonsils and short neck indicate caution. Prepare for a difficult intubation. Intraoperative end tidal CO_2 monitoring may act as a guide to potential use of postextubation assisted ventilator requirements (i.e. CPAP or BIPAP).

PREMEDICATION

Avoid premedication if possible. Benzodiazepines contribute to loss of muscle tone and predispose to sleepiness postoperatively. If essential, it should be given where the patient can be on the CPAP machine or at least closely observed and where the agent can be reversed. More important is discussion with the patient of what is to be done and good preoxygenation. Most patients will be able to be intubated and difficulty is fortunately uncommon.

INDUCTION

Use a local or regional technique if possible. All sedative agents depress the respiratory reflexes and reduce muscle tone. The only exception may be dexmedetomidine.

If a general anaesthetic is required, then use controlled ventilation. Patients can breathe spontaneously but need close monitoring, will need CPAP to prevent atelectasis and with most techniques will remain drowsy postoperatively. Use short-acting agents.

SURGICAL ISSUES

Thoracic or abdominal surgery compromises the chest. Some procedures such as uvulopalatopharyngoplasty and tonsillectomy result in upper airway problems postoperatively. There may be physical narrowing through haematoma, oedema or

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bleeding. Analgesics and opiates increase respiratory depression. Nasal surgery with packs may impair the airway dramatically and pose problems from apnoea and from the packs. Occasionally it will be sensible to leave the patient intubated until they can be extubated while awake. Rarely the safe option will be elective tracheostomy.

Extubate the patient semi-upright especially in the obese patient with OSA. Complete reversal of neuromuscular block is important. Obstruction postextubation is more common and there is a risk of negative pressure pulmonary oedema.

Pharyngeal oedema has been reported. The cause is uncertain but a small amount of swelling may be critical in a narrowed airway. Haemodynamic instability may need controlling with beta blockers and other antihypertensives.

POSTOPERATIVE MANAGEMENT

Supplemental oxygen is important. The balance between analgesia and good breathing is important. These patients need careful monitoring in the postoperative phase until their analgesic requirements are minimal and they are mobilising. The use of effective CPAP will allow adequate analgesia. In patients with SAS and obesity hypoventilation, type 2 respiratory failure can ensue. They are also more prone to greater desaturations, even on CPAP. These patients should have been identified, and provision for noninvasive ventilation planned.

Early complications include airway obstruction, bleeding into the airway, vomiting and aspiration and respiratory depression with airway obstruction. Later complications include insidious respiratory depression with hypercapnia, increased episodes of hypoventilation or even episodes of apnoea and hypoxaemia. Later effects include those of basal atelectasis with late secondary chest infection.

Patients with home CPAP should continue its use. Some patients may benefit from preoperative training. In the early phase postextubation it may impair coughing, suctioning, communicating and give a false sense of reassurance. It may be counterproductive and the real benefit comes in later in the recovering patient, particularly for sleeping at night.

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CROSS-REFERENCES

SMOKING AND ANAESTHESIA

Recognition of the problems of smoking has taken a long time and is only just beginning to alter behaviour; therefore, a significant proportion of patients will be smokers or have a long, but recently stopped, smoking history.

PATHOPHYSIOLOGY

The effects of smoking on the respiratory system include:

- Airway hyper-reactivity, especially small airways
- Reduced mucociliary clearance
- Increased mucus secretion
- A change in epithelial permeability
- Altered surfactant and hence compliance
- Small airway narrowing
- V/Q mismatch

The effects of smoking on the cardiovascular system include:

- Hypertension (due to chronic nicotine exposure and atherosclerotic change).
- Increased catecholamine levels (15–50 ng/L).
- Reduced oxygen uptake and shift in the oxygen dissociation curve to the left caused by carboxyhaemoglobin.
- Increased haemoglobin values in long-standing smokers secondary to relative hypoxaemia and carboxyhaemoglobin.
- A predisposition to thrombosis due to the increased haemoglobin together with damage to vascular endothelium. Endothelin is released resulting in a negative effect on nitric oxide dynamics and altering superoxide production. It acutely affects clot dynamics and thrombin structure and thus is thrombogenic. Curiously, cigarette smoke may have a synergistic effect with clopidogrel reducing platelet aggregation but in general should be considered thrombogenic.

The immunological effects of smoking are diverse but include reduced phagocytic and cytotoxic T-cell activity. There is some evidence for impaired immune defences.

Smoking is associated with enzyme induction so there may be an altered response to some drugs although the clinical relevance of these effects is questionable.

In the postoperative phase, smokers are more prone to hypoxaemia, have slightly higher PCO_2, have more change in pulmonary function tests, with a reduction in FEV_1/FVC ratio suggesting greater small airway obstruction. Pulmonary complications were doubled in one series and in major surgery time to extubation, ICU stay and hospital stay were all increased.

PREOPERATIVE ASSESSMENT

- Ideally, a patient should have stopped some weeks previously.
- Examine for chronic respiratory disease.
- Examine for cardiovascular disease, especially hypertension and ischaemic heart disease.
- There is an association with excess alcohol intake.
- In heavy smokers with airway disease, nutritional state may be affected.

INVESTIGATIONS

Routine investigations according to local policy (Table 1.9). Depending on the clinical picture, also consider:

- ECG with echocardiography if indicated
- Pulmonary function tests
- Arterial blood gases
- CPET
- Look for signs of infection

PREMEDICATION

An anticholinergic may be helpful as these patients will probably have irritable airways due to hypersecretion.

In those who have given up, there may be signs of nicotine withdrawal with agitation so anxiolytics may be considered.

H_2 antagonists or antacids should be considered.

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Table 1.9 Effects of heavy or long-term smoking on preoperative investigations

Investigation	Result
Full blood count	Increased haematocrit
ECG	Signs of ischaemic heart disease
Chest X-ray	Chronic airways limitation Infection Malignancy
Arterial blood gases	Hypoxaemia, hypercarbia
Lung function tests	Decreased FEV_1, FVC, PEFR

Table 1.10 Benefits of stopping smoking in the perioperative period

Time before surgery	Benefit
2 h	Nicotine blood levels fall
12 h	Carbon monoxide blood levels fall
Days	Sputum volume reduced; Haematocrit falls
Weeks	Ciliary activity restored towards normal Epithelial permeability returns towards normal
Months	Immune system recovery Drug metabolism restored towards normal

PERIOPERATIVE MANAGEMENT

MONITORING ACCORDING TO AAGBI GUIDELINES

Oxygen saturation monitor may overestimate SaO_2 if there is significant carboxyhaemoglobin content. Smoking greater than 20 cigarettes a day is associated with a carboxyhaemoglobin <4% and this will fall fairly rapidly after stopping smoking, so the P_{50} will be returning to normal at 12 hours, or faster if receiving an increased inspired oxygen concentration. Table 1.10 highlights several benefits of smoking cessation.

If there is evidence of impaired respiratory function or heavy smoking right up to the point of anaesthesia, then increased FiO_2 is recommended.

CHOICE OF ANAESTHETIC TECHNIQUE APPROPRIATE TO THE PATIENT AND THE PROCEDURE

In the presence of airway problems associated with smoking, a regional technique may be preferred where feasible and may allow more effective physiotherapy postoperatively.

GENERAL ANAESTHESIA

Main considerations are the irritable airways and there may be a nicotine-mediated exaggerated pressor response to intubation. This may be obtunded with lidocaine applied locally. Spontaneous breathing, unless deep, may be problematic with coughing.

POSTOPERATIVE MANAGEMENT

This should reflect the comorbidities. Early active physiotherapy should be instigated if there are chronic lung problems. The risk of secondary infection is increased as is the likelihood of postoperative complications generally. It is wise to continue enhanced oxygen by mask for 24 hours minimum. The incidence of postoperative nausea and vomiting is reduced amongst smokers.

STOPPING SMOKING AND POSTOPERATIVE OUTCOME

The issue of smoking and complications is more contentious than it appears. Most studies show current smokers to have higher complication rates than non-smokers or previous smokers. The complication rate in some series amongst smokers is doubled. Wound healing is also impaired. Not all studies show this; for lung resection for carcinoma, the incidence of complications was not different between those who stopped smoking and those that did not. This was seen as a reason not to delay surgery. It is reasonable to assume smoking is associated with more postoperative complications and impaired wound healing.

More difficult is the issue of advice as to when to stop smoking (Table 1.10). Stopping 6–8 weeks prior to surgery is beneficial in terms of reduced

complication rate (52%–18%), but stopping for less time might be detrimental. Benefit has also been demonstrated for 3–4 weeks. One study has shown no benefit but no detriment for cessation at 1–3 weeks. Current advice should be that postoperative complications are reduced and wound healing improved by stopping smoking even for as little as 3 weeks. Shorter intervals may not be helpful but have no detriment. This was not seen in a recent paper in patients undergoing thoracotomy.

In 1944, Morton reported a sixfold increase in the incidence of postoperative respiratory morbidity in smokers over non-smokers. These findings have been confirmed in several other studies more recently. Every opportunity should be taken to discourage smoking in the perioperative period.

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CROSS-REFERENCES

COPD and anaesthesia, Chapter 1
Ischaemic heart disease, Chapter 2
Preoperative assessment of cardiac risk, Chapter 25
Preoperative assessment of pulmonary risk, Chapter 25

2

Cardiovascular system

REDMOND P TULLY AND ROBERT TURNER

AORTIC VALVE DISEASE

AORTIC STENOSIS

Aortic stenosis (AS) remains the most common valvular lesion worldwide. Prevalence is estimated at between 9.2% and 16% in people over 75 years of age and severe AS is estimated at 3.4%. Approximately 75% of patients with severe AS are symptomatic, with 40% requiring surgical intervention leaving a significant proportion either unrecognized or untreated.

AETIOLOGY

Calcific aortic valve disease is the most common cause, ranging from aortic sclerosis, with thickening of the leaflets, to aortic stenosis where obstruction of the left ventricular outflow occurs. Calcification can occur with a structurally normal aortic valve, although progression is accelerated in the presence of congenital valvular abnormalities, e.g. bicuspid aortic valve (in approximately 2% of cases). Infective endocarditis and rheumatic heart disease account for the remaining cases.

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PATHOPHYSIOLOGY

In systole, the aortic valve offers little resistance to outflow with near identical pressures in the aorta and LV. Thickening of the valvular leaflets and cal-cification lead to progressive narrowing of the open valve area. Once this has reached 50% of normal size (<2.0 cm^2), a pressure gradient develops resulting in pressure overload of the left ventricle. To compen-sate for the increased pressure required to maintain stroke volume, concentric left ventricular hypertro-phy (LVH) occurs.

The hypertrophied left ventricle becomes impaired or 'stiff' with delayed isovolumetric relax-ation, and consequent shortening of filling time – diastolic dysfunction. In turn, this increases filling pressures required for a given volume (Figure 2.1) meaning there is a greater reliance upon atrial con-traction for adequate LV filling. Consequently, these patients are particularly susceptible to reductions in preload and atrial dysthymias, e.g. AF. These dia-stolic changes can also result in a 'fixed cardiac out-put state' where the cardiac output (CO) cannot be increased in response to systemic vasodilation.

Angina and an increased risk of myocardial infarction are present in a significant proportion of patients with AS despite up to a third having nor-mal coronary arteries. This is largely a consequence of increased myocardial mass and hence oxygen demand, and the shifted balance between myocar-dial oxygen debt (systole) and myocardial oxygen repayment (diastole).

As the disease progresses, the pressure required to overcome the pressure gradient of the valve will no longer be balanced by the LVH resulting in increased wall stress, and eventually dilatation of the left ventricle cavity. These changes result in conges-tive cardiac failure.

PREOPERATIVE MANAGEMENT

Increased perioperative cardiac risk occurs in the presence of moderate or severe AS. This is increased further in the presence of symptoms. Hence, symp-tomatic patients for all elective noncardiac sur-gery and asymptomatic patients in the presence of severe or critical AS undergoing high-risk surgery, or the presence of concurrent CAD or coexisting moderate-severe MR, should be considered for valve replacement preoperatively. In patients unsuitable for surgical intervention, transcutaneous approaches may be considered (TAVR) or balloon valvuloplasty. If considered ineligible for the above, the patient should be counselled about the risks and benefits of proceeding.
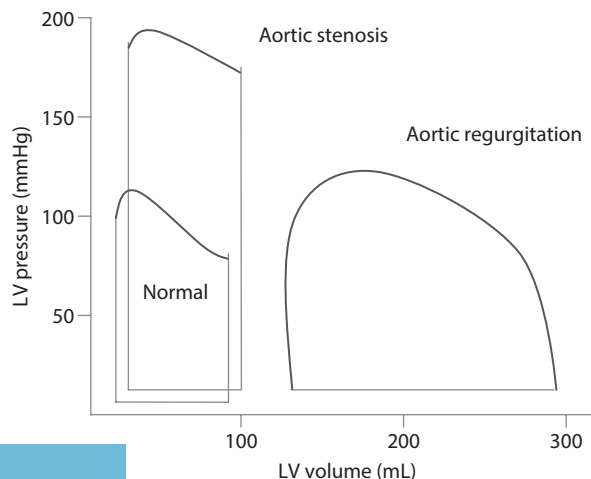

Figure 2.1 Aortic valve disease pressure volume loops.

HISTORY

- Look particulary for symptoms of AS or cardiac failure. If a new diagnosis of AS is made, or the presence of new symptoms or poor symptom control elicited, obtain a cardiologist's opinion.
- Review all medications.
- Obtain a history of comorbid diseases.
- Review recent cardiovascular investigations, in particular echo and exercise tolerance tests.
- Syncope, angina and dyspnoea (SAD) are the classical triad of symptoms. The presence of symptoms does not correlate well with severity of AS. The presenting symptom does correlate with mortality: CCF 2 years, syncope 3 years, angina 5 years.

EXAMINATION

Look for

- Slow rising low volume pulse with narrow pulse pressure.
- Sustained heaving apical impulse in the presence of LVH, or displaced apex in the presence of CCF.
- Ejection systolic murmur heard throughout the precordium, loudest in the aortic area and radiating to the neck. As the stenosis worsens, S2 will become singular and then reverse splitting occurs and S2 can be obliterated by the murmur. S4 may also be heard as atrial contraction against the stiff LV.
- Features of CCF.

INVESTIGATIONS

- *ECG* – Evidence of LVH/strain, LBBB or RBBB.
- *Echo* – Evaluation of severity of valvular lesion and LV function (Table 2.1) – note in the presence of LVF the pressure gradient can underestimate severity.
- *Cardiac atheterization* – Evaluation of LV function, valve gradient and concurrent CAD.
- *Exercise testing* – Consider to identify and evaluate coexisting CAD.

Table 2.1 Grading of severity of aortic stenosis by echocardiography

	Valve area (cm^2)	Mean valve gradient (mmHg)
Mild	>1.5	<20
Moderate	1–1.5	20–40
Severe	0.6–1	40–60
Critical	<0.6	>60

MONITORING

- *ECG* – At least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced due to underlying LVH.
- *Invasive arterial pressure (preinduction)* – Allows rapid recognition of haemodynamic changes
- *TOE* – Allows accurate evaluation of ventricular filling and provides the most useful information where rapid volume shifts are expected, can also identify regional wall motion abnormalities (RWMA), an early sign of ischaemia. However, this requires an experienced operator.
- *Pulmonary artery catheter* – Not recommended as measurement of filling will be inaccurate and can induce dysrhythmias.
- *Noninvasive cardiac output monitoring* – Not validated, and values for stroke volume and cardiac output will be inaccurate although trends pre-/post-fluid boluses have been shown to be accurate with oeesophageal Doppler and pulse contour wave analysis.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a low-normal rate. Avoid tachycardia – Reduces diastolic LV filling, coronary artery perfusion and LV ejection time in systole, resulting in reduced cardiac output and myocardial ischaemia.
- *Blood pressure* – Aim for normal blood pressure and high-normal SVR using direct alpha-agonists (metaraminol and phenylephrine). Pay meticulous attention to volume status to ensure adequate filling. Consider preloading with intravenous fluid boluses when fluid shifts are expected.

- *Dysrhythmias* – Treat promptly. A defibrillator should always be available in theatre if not connected to the patient for rapid DC cardioversion if required.

ANAESTHETIC TECHNIQUE

Rapid changes in SVR and ventricular filling are poorly tolerated due to a relatively fixed cardiac output. Falls in SVR result in reduced cardiac output, profound hypotension and reduced coronary blood flow with resulting myocardial ischaemia. Pain will cause increased catecholamine levels and resultant tachycardia; therefore, good analgesia is essential.

General anaesthesia +/- regional limb blocks is considered the best approach, although drugs should be titrated carefully. Avoid inappropriate use of premedication. A central venous catheter (CVC) may be considered for safe administration of vasoactive substances in symptomatic patients or patients undergoing high-risk surgery.

Central neuraxial blockade potentially causes significant reduction in SVR. This should generally be avoided in patients with severe AS, and caution exercised if undertaking this approach.

POSTOPERATIVE MANAGEMENT

Maintain a low threshold for transferring the patient to ICU even following a minor procedure, due to inexperience of ward staff in dealing with patients with severe AS. Continue direct arterial pressure monitoring postoperatively, with the same considerations regarding haemodynamic parameters as intraoperatively. Provide adequate postoperative analgesia and a plan for escalation if this is not achieved.

AORTIC REGURGITATION

The incidence of aortic regurgitation (AR) increases with age, with a peak at >80 years old. Estimates of prevalence range from 2%–30% for all severities, with less than 1% of the population having severe disease. The Framingham study demonstrated a significant gender difference with prevalences of 13% in men and 8.5% in women, although this may be accounted for by the higher incidence of bicuspid aortic valves and Marfan syndrome in males.

AETIOLOGY

Congenital or degenerative diseases of the valve leaflets or aortic root are the most common causes (Table 2.2). However, worldwide, rheumatic fever remains a significant cause of AR.

PATHOPHYSIOLOGY

Aortic regurgitation causes both volume and pressure loading of the left ventricle due to regurgitant flow from the aorta back into the LV during diastole. The resulting increased LV end-diastolic volume is compensated for by an increased stroke volume, hence increased systolic LV pressure and pulse pressure (Figure 2.1). The radius of the regurgitant orifice is the primary determinant of regurgitant flow, with up to 60% of the stroke volume returning to the LV in severe disease.

The LV remodels to accommodate this, with an increase in cavity dimension and mixed eccentric and concentric hypertrophy resulting in a more spherical shape. These changes and increased heart rate allow preservation of the ejection fraction (EF) until late stages of disease. Whilst the remodelling means patients can be asymptomatic for long periods, eventually the eccentric hypertrophy will fail to compensate for the increased volume and the concentric hypertrophy will fail to maintain normal wall stress. This results in raised filling pressures, reduced cardiac output and development of congestive cardiac failure.

Table 2.2 Causes of aortic regurgitation

Valvular	Aorta
Bicuspid aortic valve	Marfan's syndrome/ Ehlers–Danlos Syndrome
Infective endocarditis	Syphllis
SLE	Hypertension
Rheumatic fever	Ankylosing spondylitis/ RA/Behcet disease
Appetite suppressant drugs	Trauma

CLINICAL MANAGEMENT

PREOPERATIVELY

In asymptomatic patients with AR and preserved LV function, there is no increased risk of cardiovascular complications in noncardiac surgery. If patients have symptomatic CCF, NYHA III or IV, or EF <30%, they should be considered for valve replacement surgery. If not suitable for surgical replacement, medical optimisation, with ACE-I's, ARB's or mineralocorticoid antagonists and titrated cardio-selective beta-blocker therapy should be undertaken prior to elective surgery. For new diagnoses of heart failure, where possible, surgery should be delayed for 3 months to allow optimization.

HISTORY

Many patients will by asymptomatic; however, the most common symptom is dyspnoea. Patients may also develop other features of CCF and can develop angina. Pay particular attention to symptoms of AR or cardiac failure. Medications should be reviewed and a history of comorbid disease sought. Recent cardiovascular investigations, in particular echo and exercise testing, should be reviewed and noted as part of the preoperative assessment.

EXAMINATION

- Collapsing pulse and wide pulse pressure.
- Displaced apex inferolaterally.
- Early diastolic high-pitched murmur classically maximal at lower left sternal edge although if aortic root involvement is present, this may be heard in the aortic area. The volume of murmur is not indicative of severity of regurgitation. The murmur is best heard with the patient sitting-up, leaning forward in expiration.
- An Austin-Flint murmur may be heard in mid-diastole at the apex due to regurgitant flow directed at the anterior MV leaflet.
- **De Musset's sign** – Head nodding with each pulse.
- **Corrigan's sign** – Visible carotid pulsation.

INVESTIGATIONS

ECG – LVH/strain, LAD
CXR – Cardiomegaly +/– pulmonary congestion +/– dilated ascending aorta
Echo – Measurement of vena contracta, regurgitant fraction and evaluation of LV function.

INTRAOPERATIVELY

MONITORING

- *ECG* – At least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced due to underlying LVH.
- *Invasive arterial pressure (from preinduction)* – To allow rapid recognition of haemodynamic changes, where CCF is present.
- *TOE* – May be considered to monitor filling, although an experienced operator is required. This has a strong indication for mixed valvular disease where early detection of RWMAs is of greater importance.
- *Noninvasive cardiac output monitoring* – Not validated and values for stroke volume and cardiac output will be inaccurate.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a high normal heart rate. Avoid bradycardia as this increases diastolic time and hence regurgitant volume.
- *Blood pressure* – Aim for a low normal blood pressure with low-normal SVR ensuring adequate filling with IV fluid. Patients will be very sensitive to reduced preload.
- *Dysrythmias* – Treat promptly particularly if associated with hypotension. A defibrillator should always be available in theatre for rapid DC cardioversion of tachyarrhythmias if required. Persistent bradycardia may require treatment with anti-cholinergics or beta-agonists.

ANAESTHETIC TECHNIQUE

Unlike in AS, neuraxial techniques are well tolerated and the aim should be to maintain a low-normal afterload. Ensure good analgesia to prevent

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catecholamine release and associated hypertension. Consider a CVC for safe administration of vasoactive agents in symptomatic patients or patients undergoing high-risk surgery.

POSTOPERATIVELY

Following high-risk procedures, or where large volume shifts have occurred, monitoring in ICU is indicated with invasive blood pressure monitoring, to detect early signs of CCF.

PROSTHETIC VALVES

Patients with prosthetic valve replacement will normally undertake regular review with the cardiology team, and should have recent assessment of valvular function. If this is not available locally, echocardiography should be performed preoperatively where possible.

ANTICOAGULATION

Tissue valves do not require lifelong anticoagulation except in special circumstances. Mechanical valves will require life-long anticoagulation normally with warfarin. The risk of thromboembolic events largely depends on the site and type of valve. Older aortic valve and mitral valve replacements have a much higher embolization rate of approximately 5% per annum. For these, bridging should occur for the full period INR remains <2, with either unfractionated heparin infusions or treatment dose low molecular weight heparins. For modern bileaflet mechanical aortic valves, technological advances have reduced the thromboembolic rate to less than 5% per annum, some studies report as low as 1%. For these valves, bridge with prophylactic dose low molecular weight heparin may be considered and an alternative to full therapy in low risk procedures.

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CROSS-REFERENCES

Heart failure, Chapter 2
Preoperative assessment of cardiovascular risk, Chapter 25

ATRIAL SEPTAL DEFECTS

Atrial septal defects (ASD) account for 10% of all congenital heart disease (CHD), and 20%–40% of CHD presenting in adult life. It is twice as common in women as men. Ostium secundum is the most common (75%), with primum (15%–20%) and sinus venosus (5%–10%).

AETIOLOGY

The formation of the atrial septum occurs in several stages. The septum primum is a soft tissue structure that grows towards the endocardial cushions to form the initial division into left and right atria. The space between the septum primum is the ostium primum, which narrows as the septum primum grows. Before this is fully occluded, the ostium secundum forms due to resorption of a portion of the septum primum, allowing continued movement of blood between right and left atria.

The septum secundum is a muscular structure that develops anterior to the septum primum. As it grows along a similar path to the septum

primum, it leaves an opening, the foramen ovale, which is continuous with the ostium secundum. The septum primum gradually shrinks leaving only a small flap of tissue, the valve of the foramen ovale, which closes after birth when the lungs become functional and pulmonary vascular pressure falls. Atrial septal defects are due to errors in this process.

Ostium primum ASD – Incomplete fusion of the septum primum with the endocardial cushion leads to a defect adjacent to the atrio-ventricular valves, which may or may not be affected (mitral > tricuspid).

Ostium secundum ASD – Caused by an unusually large ostium secundum, or failure of the septum secundum to correctly align with the septum primum. These defects can be further classified into:

Patent foramen ovale – Arises due to inadequate development of the septum secundum, or excessive or abnormal resorption of the septum primum, resulting in failure of normal closure of the foramen ovale soon after birth. Occurs in up to 30% of the population.

Sinus venosus ASD – Secondary to abnormal fusion of the sinus venosus and the atrial septum, usually near to the entry of the superior vena cava into the right atrium. Partial anomalous pulmonary venous drainage can be a feature.

Coronary sinus ASD – Defect resulting in an unroofed coronary sinus and persistent left superior vena cava that drains into the left atrium. Right-to-left shunt can result in desaturation. This is diagnosed by contrast injection into the left upper extremity – the coronary sinus will opacify before the right atrium.

PATHOPHYSIOLOGY

The degree of shunting across an ASD is related to the relative compliance of the two ventricles and the cross-sectional area of the defect. In the neonate, right- and left-sided cardiac pressures are approximately equal and little or no shunting occurs. As pulmonary vascular resistance falls, a left-to-right shunt develops. This is normally well tolerated; however, with large shunts the right heart will become volume-loaded and pulmonary flow will increase resulting in pulmonary hypertension and

its sequelae. If left untreated, eventually the compliance of the right side of the heart will decrease and pressures will equalize between the right and left side of the heart. This allows bi-directional shunting and further increases in pulmonary artery pressure (PAP) will result in a right-to-left shunt (Eisenmenger's syndrome). Paradoxical emboli are now able to enter the systemic circulation from the right side of the heart, increasing the risk of systemic emboli and resultant CVA or MI. Surgery can be undertaken to close the defect and halt the progression to cyanotic disease; however, if this is delayed and the pulmonary hypertension (PH) is permanent, right ventricular (RV) failure will follow.

ASD CLOSURE

In childhood an ASD secundum may close spontaneously; however, once adulthood has been reached this is extremely unlikely. The decision to repair an ASD is based on clinical and echocardiographic information, including the size and location of the ASD, the magnitude and haemodynamic impact of the left-to-right shunt, the presence and degree of pulmonary arterial hypertension, previous paradoxical emboli, and the presence of orthodeoxia-platypnea. In general, closure is recommended in the following circumstances:

- Presence of right ventricular enlargement with or without symptoms
- Following the occurrence of a paradoxical embolic event
- Documented ortodeoxia-platypnoea
- In patients with PH if the pulmonary vascular resistance (PVR) <5 Wood units, or >5 Wood units with PAP <2/3 SVR

CLOSURE METHOD

There are two main options for closure of an ASD – percutaneous and surgical. For an ASD primum, surgical closure is performed due to the larger size of the defect and possible involvement of the mitral valve cleft. Coronary sinus ASDs and sinus venosus ASDs are usually closed surgically although percutaneous devices are used as well. For ASD secundums

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<40 mm with a rim of tissue at least 5 mm around the defect, percutaneous device closure and surgery have comparable mortality data although reintervention is slightly higher for the device closure group.

PERCUTANEOUS DEVICE CLOSURE

Percutaneous closure is classically done under general anaesthesia via a femoral venous approach under intracardiac echo (ICE) or TOE guidance. If ICE is utilized, a supraglottic airway device may be used but if TOE is needed intubation is required. Following closure, dual antiplatelet therapy is continued for at least 6 months.

SURGICAL CLOSURE

For surgical closure, a midline sternotomy is routine but a minimally invasive approach with or without the aid of robotic surgery is in development. Complete thorascopic procedures have also been undertaken. Patients <25 years have better long-term outcomes. Some centres propose anticoagulation for up to 3 months following closure, to minimise the risk of thrombus attaching to the atrial patch and subsequent embolic complications. This is more common in patients who have had intraoperative arrhythmias and thus may benefit from postoperative anticoagulation.

CLINICAL MANAGEMENT

PREOPERATIVE MANAGEMENT

History

Pay particular attention to symptoms of cardiac failure, pulmonary hypertension, recurrent chest infections in children and cyanotic episodes with or without a relationship to posture. Review medications and any comorbidities. Review cardiovascular investigations, in particular echo and ECGs. Uncomplicated defects are likely to be relatively asymptomatic and the only indication of pathology may be the incidental finding of a murmur.

Examination

- Soft ejection systolic murmur loudest over the pulmonary area

- Pansystolic murmur loudest at the left sternal edge if tricuspid regurgitation (TR) has developed
- Fixed splitting of S2 due to increased pulmonary flow
- Pulse may be regular or irregularly irregular
- A right ventricular heave may be palpable at the left sternal edge
- JVP may be normal or raised, giant CV waves may be seen in the presence of TR, or cannon A-waves in the presence of right ventricular hypertrophy
- Features of syndromes associated with ASDs may be present, e.g. Down's syndrome

Investigations

- *ECG* – Look for AF; first-degree heart block; evidence of RVH/strain; incomplete RBBB; left axis deviation (LAD – seen with primum defects); right axis deviation (RAD – with secundum defects).
- *CXR* – Cardiomegaly, with atrial enlargement and pulmonary congestion.
- *ECHO* – Transthoracic echocardiography (TTE) can often reveal the defects – if not visible initially, bubble studies may be performed. ICE should be used to assess for the suitability of device closure.
- *Cardiac catherisation* – Can provide detailed information on location and function of the defect, pulmonary systemic shunting, PAP and ventricular function.

INTRAOPERATIVE MANAGEMENT

Monitoring for noncardiac procedures is the same as for a patient without an ASD. For closure procedures, use invasive arterial monitoring, ICE or TOE.

The recirculation of blood from intracardiac shunts may lead to a slower onset of intravenous induction and an increased dose may be required. Moderately soluble inhalational volatile agents (e.g. sevoflurane and desflurane) will have a more rapid increase in alveolar concentration due to a left-to-right shunt. Standard hypnotic agents are considered safe for induction and maintenance and inhalational agents provide a smaller reduction in

SVR than TIVA. Physiological targets should be similar to those for patients with established PH in order to prevent increases in PVR and potential shunt reversal.

POSTOPERATIVE MANAGEMENT

Postoperative care should be the same as for a patient without an ASD, depending on the procedure undertaken and comorbid disease. Pay particular attention to volume status and electrolyte management due to the increased risk of AF.

ASD AND EISENMENGER'S SYNDROME

Patients with right-to-left shunts will appear cyanosed and have finger clubbing. Pulmonary regurgitation, if present, causes a decrescendo diastolic murmur on auscultation. Chest radiography shows right ventricular hypertrophy, prominent pulmonary arteries and increased lung markings. Cardiac catheterization will confirm increased right ventricular and pulmonary artery pressures.

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Congenital heart disease in adult life, Chapter 2
Herat failure, Chapter 2
Patients with pacemakers and implantable
    defibrillators, Chapter 2
Pulmonary hypertension, Chapter 2

CORONARY ARTERY DISEASE

Although coronary artery disease (CAD) is no longer the greatest cause of mortality in the UK it remains a major cause of perioperative mortality and morbidity. Cardiac complications account for up to 42% of deaths within 30 days of surgery. It remains a major burden on healthcare systems in the developed world and has increasing prevalence in developing countries; it is estimated that the presence of CAD increases the perioperative risk of major complications by approximately 2.5-fold compared to the general population. This is particularly evident in open vascular surgery where the perioperative myocardial infarction rate is 5% compared to 1% nonvascular/noncardiac surgery. Timing of surgery after a previous myocardial infarction is important, with 30-day mortality 14.2% in the first 30 days reducing to 10.5% after >60 days.

AETIOLOGY

Atheromatous disease remains the most common cause. Plaques consisting of lipids with localized smooth muscle proliferation restrict blood flow within the coronary arteries. Ischaemia results when myocardial oxygen demand increases beyond supply or when there is rupture of plaque which can precipitate thrombosis and result in complete occlusion of an artery. Risk factors are illustrated in Table 2.3.

PATHOPHYSIOLOGY

Perioperative cardiac complications are caused by an imbalance between cardiac muscle oxygen supply and demand resulting in ischaemia. Increased

Table 2.3 Risk factors for CAD

Fixed	Modifiable
• Age: >45 men, >55 women	• Smoking
• Gender: male 5x risk vs. female at age >50	• Hypertension
	• Diabetes mellitus
• Race	• Obesity
• Family history of early IHD	• Increased LDL:HDL ratio

myocardial oxygen demand may result from tachycardia (increases myocardial VO_2 and reduces diastolic filling time), increased contractility (e.g. pain causing a sympathohumoral response), and increased wall tension (inotropic therapy, hypertension). Alternatively supply may be reduced by tachycardia, vasospasm, hypotension, increased LVEDP (reduces coronary blood flow), hypoxia or anaemia. This is particularly important in the context of perioperative blood loss. Patients undergoing surgery are also at increased risk of an occlusive event due to the hypercoagulable and proinflammatory state.

PREOPERATIVE ASSESSMENT

AHA and ESC/ESA 2014 guidelines have suggested a stepwise approach to the assessment of preoperative risk evaluation and perioperative management of cardiac patients undergoing noncardiac surgery.

If the urgency of surgery prevents necessary cardiac testing or treatment, surgery should proceed with adequate perioperative surveillance for cardiac and medical treatment as appropriate. A plan should be instigated for immediate postoperative monitoring, further investigations and management; this would often include observation in an ICU.

If there is no requirement for immediate surgery, the patient should be screened for the presence of any unstable cardiac conditions:

1. Unstable angina pectoris
2. Acute heart failure
3. Significant cardiac arrhythmias (e.g. high-grade heart block, symptomatic monomorphic ventricular rhythms, polymorphic ventricular rhythms, new VT, SVT with ventricular response >100 at rest, new prolonged QT_c)
4. Symptomatic or severe valvular heart disease
   a. Severe or critical aortic stenosis (valve area <1 cm^2)
   b. Symptomatic mitral stenosis, or symptomatic in the presence of severe stenosis – valve area <1.5 cm^2 with pulmonary artery pressure >50 mmHg
   c. Symptomatic aortic or mitral regurgitation, or asymptomatic with LVEF <30%
5. Recent myocardial infarction (<30 days) or residual ischaemia

If any of these are present, the benefit of further evaluation and potential optimization should be weighed against any deleterious effects of delaying surgery by discussion involving anaesthetic, surgical and cardiology teams.

Where no unstable cardiac conditions are present, the procedural risk of the surgery should be considered in combination with the functional capacity of the patient. If the surgery is considered to have a 30-day MI and cardiac death risk <1%, or >1% with the functional capacity of the patient >4 metabolic equivalents (METs), risk factors should be identified and recommendations advised on lifestyle and medical therapy in line with current ESC/NICE guidance. A preoperative ECG should be considered. In patients with known IHD or previous myocardial ischaemia, low-dose titrated cardio selective beta-blockade should be considered or titrated preoperatively. Where ventricular systolic dysfunction has been identified, consideration should be given to an ACE-I preoperatively and in all patients undergoing vascular surgery statin therapy should be considered.

In situations where the functional capacity of the patient is ≤4 METS, the patient should be considered for noninvasive stress testing if one or more risk factors from Table 2.4 are present.

NON-INVASIVE TESTING

RESTING 12-LEAD ECG

A resting standard configuration ECG may reveal underlying rhythm disturbances or evidence of ischaemia. It is recommended that a resting ECG is

Table 2.4 Clinical risk factors according to revised cardiac risk index

Heart failure

Stroke or TIA

Diabetes mellitus requiring insulin therapy

Ischemic heart disease angina pectoris and/or
   previous myocardial infarction

Renal dysfunction (serum creatinine >170 umol/L or
   CrCl <60 mL/min/1.73 m²

Peripheral arterial disease (ankle brachial ratio <0.9,
   or previous revascularization)

Source: Modified from Lee TH et al. (1999). *Circulation*
      100:1043–1049.

performed on all patients with known CAD or risk factors undergoing intermediate or high-risk surgery. It may also be considered for patients undergoing low-risk surgery and patients over 65 with no risk factors undergoing intermediate or high-risk surgery. It is not recommended for patients undergoing low-risk surgery unless clinical suspicion of arrhythmias exists.

ASSESSMENT OF VENTRICULAR FUNCTION

Resting LV function may be assessed by SPECT, cardiac gated CT or MRI, radionucleotide ventriculography or echocardiography. TTE provides the most versatile and readily available imaging modality, which can also provide useful information about valvular disease. It is not recommended as a routine for all patients undergoing surgery, however, in the absence of signs of cardiac disease it may be considered for patients undergoing high-risk surgery. Patients with signs or symptoms of new valvular disease or cardiac failure should have TTE evaluation as should patients with known disease with symptomatic change in the presence of existing CAD.

STRESS TESTING

Bicycle or treadmill testing can detect inducible ischaemia and provides an estimate of functional capacity; however, the accuracy of detection of ST-segment changes during exercise varies significantly between studies and operators. Also preexisting ST-segment abnormalities or bundle branch blocks hinder reliable analysis. Results provide a graded response with onset of changes at low workloads associated with a significantly increased risk of perioperative mortality, cardiac events and long-term cardiac events. Inducible changes at high work-loads indicate a minimal increase in risk from a normal test.

In patients with limited exercise tolerance or reduced mobility, stress echocardiography, stress myocardial perfusion scanning, or stress cardiac MR can be carried out. This is achieved by undertaking imaging pre-/postpharmacological stressor (dipyridamole, dobutamine or adenosine). Stress echocardiography has high negative predictive value but low positive predictive value and failure to reach target heart rate is common. These allow inducible ischaemia to be demonstrated where areas of the myocardium are at risk, and fixed scarring where revascularization would be impossible.

Stress testing is recommend for patients undergoing high-risk surgery with poor functional capacity (<4 METS) and two or more risk factors (Table 2.4). It may also be considered in high or intermediate risk surgery in patients with one or more risk factors and poor functional capacity (<4 METS).

CORONARY ANGIOGRAPHY AND REVASCULARIZATION

Invasive coronary angiography is rarely indicated for patients undergoing noncardiac surgery and when undertaken inappropriately can result in unpredictable delay to planned surgery. Although CAD may be present in a significant proportion of patients undergoing surgery, the indications are similar to those in a nonsurgical setting. Urgent angiography is recommend where a patient has acute ST-elevation, new acute bundle branch block not requiring urgent surgery or where clinical benefit from angiography outweighs that of surgery. Urgent or early angiography should be undertaken in patients who have ACS or NSTEMI not requiring urgent surgery. In patients requiring urgent surgery, late revascularization should be considered postoperatively and the benefit of timing of angiography and surgery should

be discussed with the specialist teams on a case-by-case basis.

Elective preoperative angiography is recommended in patients with proven MI and unstable angina with maximal medical therapy. It can also be considered in patients with stable ischaemic cardiac disease undergoing nonurgent carotid endarterectomy. In the perioperative period two-thirds of myocardial events were not due to plaque fissure or intraluminal thrombus, but were type II MI where a low flow high demand situation occurred intraoperatively.

Surgical timing postrevascularization should be considered with current recommendations advising, where possible, a delay of 2 weeks following balloon angioplasty, a minimum delay of 4 weeks following bare metal stent insertion (but ideally 3 months) and a minimum of 6 months for new generation drug eluting stents (DES) or 12 months for older DES. If surgery is required to be performed within these windows it should be discussed with the specialist cardiology team on a case-by-case basis and discussion concerning antiplatelet therapy is essential with risks outlined to the patient.

PHARMACOLOGICAL INTERVENTIONS

Currently there is an extensive and ever-expanding array of pharmacological agents used in the treatment of CAD. As a result, in-depth discussion of each agent is not possible within the scope of this book. For complex cases, there should be discussion between the anaesthetic, surgical and cardiology teams in a timely manner preoperatively to allow optimization of therapy. Below some of the larger groups are discussed but this is not an exhaustive list of therapies.

BETA-BLOCKERS

Recent trials such as POISE and POBBLE have questioned the role of beta-blockade in the perioperative period. Both trials demonstrated increased mortality but a reduction in cardiac events. However, these results have been questioned by the maVS and DIPOM studies. As a result, the current ESC and AHA guidelines recommend continuation of beta-blocker therapy in patients on established therapy,

and preoperative initiation may be considered in patients with known IHD or patients with >2 risk factors (Table 2.4) or ASA >3 undergoing high-risk surgery.

STATINS

Statins should be continued in the perioperative period if patients are established on this therapy. Current ESC guidelines also recommend initiation of statin therapy in patients with peripheral occlusive arterial disease prior to surgery and in patients undergoing vascular surgery who are statin naïve. This should be done 2 weeks prior to surgery to allow early detection of any complications such as statin-induced myopathy and rhabdomyolysis. For maximal benefit, statins should be continued for a minimum of 1 month post-surgery.

ACE INHIBITORS/ARBS

Continuation of ACE-I therapy perioperatively provides much discussion due to the risk of hypotension under anaesthesia. Observational studies have demonstrated a less frequent reduction in hypotension when ACE-I/ARB therapy is discontinued 24 hours prior to surgery when used for treatment of hypotension although this benefit remains debatable. Current guidance recommends that, in the presence of heart failure and LV dysfunction, AECE-I/ARBs continuation in the perioperative period under close monitoring should be undertaken. When used in the treatment of isolated hypertension without heat failure or LV dysfunction, consideration should be given to discontinuation of the therapy 24 hours preoperatively and until the patient's blood pressure and volume status are stable postoperatively.

INTRAOPERATIVE STRATEGIES

ST-SEGMENT MONITORING

The occurrence of perioperative ST-segment changes has been associated with cardiac morbidity and mortality also in patients undergoing noncardiac surgery. Intra- and postoperative ST-segment monitoring with computerized ST-segment analysis is considered

useful for patients with known coronary artery disease or those undergoing vascular surgery.

PULMONARY ARTERY CATHETER

Perioperative use of a pulmonary artery catheter remains a controversial issue. While significant information can be obtained from its use, no differences have been observed in survival or cardiovascular morbidity compared to standard care in patients who underwent major noncardiac surgery.

TRANSOESOPHAGEAL ECHOCARDIOGRAPHY

The use of transoesophageal echocardiography has gained wide acceptance in the setting of cardiac surgery. However, to date there is not sufficient evidence to support its routine use as a diagnostic monitor or to guide therapy during noncardiac surgery.

ANAESTHETIC MANAGEMENT

Neuraxial techniques can result in sympathetic blockade and cause a decrease in preload and afterload. Although initially some randomized controlled trials suggested that the use of neuraxial techniques might have beneficial effects on outcome, these data have not been unequivocally confirmed in more recent studies on larger patient populations.

A comparison of the effects on outcome of general anaesthesia with opioid analgesia to combined general-epidural anaesthesia and analgesia in intra-abdominal aortic, gastric, biliary and colonic surgery revealed no overall differences in death or major complications. It seems that to date, there is insufficient evidence to confirm (or deny) that postoperative analgesic techniques affect major postoperative morbidity and mortality.

In recent years, increasing evidence has indicated that volatile anaesthetic agents may have cardioprotective properties. In the setting of coronary artery surgery, the use of these drugs was shown to be associated with a better preservation of postoperative myocardial function and less evidence of postoperative myocardial damage. In noncardiac surgery, however, there is at the moment no such evidence.

Other measures to be taken in the perioperative period that may help to improve outcome include maintenance of normothermia and adequate perioperative pain management.

POSTOPERATIVE STRATEGIES

PAIN MANAGEMENT

Postoperative pain may increase sympathetic drive and therefore constitute a risk factor for the development of postoperative cardiac complications. However, the potential benefits of invasive analgesic techniques should be weighed against the potential dangers of their application. This is especially a concern in patients on antithrombotic or anticoagulant drugs.

Patient-controlled analgesia may be an alternative for postoperative pain relief. Nonsteroidal anti-inflammatory drugs and cyclo-oxygenase-2 inhibitors may promote heart and renal failure as well as thromboembolic events and should therefore be avoided in patients with myocardial ischaemia.

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Cardiopulmonary exercise testing, Chapter 25
Coronary artery bypass grafting, Chapter 16
Perioperative assessment of cardiovascular risk in
   noncardiac surgery, Chapter 25

CARDIAC CONDUCTION DEFECTS

Cardiac arrhythmias are common during anaesthesia. Underlying conduction abnormalities that are benign in the resting heart may be exposed due to the additional physiological stress and pharmacological interventions experienced perioperatively. The presence of structural or ischaemic heart disease increases further the risk of perioperative arrhythmias. Conduction abnormalities can be classified into:

- Atrioventricular blocks
- Bundle branch blocks
- Accessory pathways
- Long QTc syndromes

ATRIOVENTRICULAR BLOCK

Atrioventricular (AV) block can be defined as a delay or interruption in the transmission of electrical impulses from the atria to the ventricles caused by a problem at the level of the AV node (AVN) or His-Purkinje System. This may be a transient or permanent alteration to normal conduction and has many causes (Table 2.5).

FIRST-DEGREE AV BLOCK

In a first-degree block, there is delayed transmission of impulses through the AVN resulting in prolongation of the PR interval to >200 ms. This heart block is normally asymptomatic and may be treated with atropine or glycopyrollate acutely if required. Chronically some patients benefit symptomatically from a dual chamber pacemaker.

SECOND-DEGREE AV BLOCK

Mobitz type I (Wenckebach) – There is delayed conduction through the AVN with progressive lengthening

Table 2.5 Classification of causes of AV block

Causes of AV block	Mechanisms
Cardiac	MI, fibrosis in the conduction systems (Lev and Lenegre syndromes), cardiomyopathies, CHD, e.g. ASD, PDA, Ebstein's, congenital heart block, cardiac surgery, valvular heart disease, myocarditis
Drugs	Beta blockers, calcium-channel blockers, digoxin
Systemic disease	Collagen vascular disorders, e.g. SLE, RA. Neuromuscular disorders, e.g. myotonic dystrophy
Other	Hypothermia, hyperkalaemia, hypoxia, hypo/hyperthyroidism

of the PR interval, until a beat is dropped (nonconducted P wave). This rhythm is usually asymptomatic and does not require treatment.

Mobitz type II – The block is usually at the level of the His bundle rather than the AVN. The ratio of P waves to conducted QRS complexes is often used to describe this block (2:1, 3:1, 4:1, variable). It can be associated with symptoms, usually pre/syncopal in nature. Mobitz type II may progress to complete heart block.

Management of reversible causes should be undertaken, e.g. electrolyte correction, treatment of myocardial ischaemia and cessation of drugs that increase nodal delay. If no reversible cause is found, and the rhythm persists, a permanent pacemaker is indicated and caution should be taken to avoid drugs which slow nodal conduction.

THIRD-DEGREE AV BLOCK (COMPLETE HEART BLOCK)

This occurs when no impulses are conducted from the atria to the ventricles and an escape pacemaker takes over. The ECG will show no relationship between P waves and QRS complexes. If QRS complexes are narrow and their rate is 45–55 bpm, this indicates an AVN block and a junctional or His-bundle escape rhythm. If QRS complexes are

broad and the rate is slower, at 30–40 bpm, it suggests an infra-nodal block with a distal ventricular escape rhythm. This block should always be treated. Removal of any aggravating medications, correction of electrolytes, and initial therapy with an isoprenaline infusion should commence while arrangements for a temporary or permanent pacemaker are made. Atropine can be used with caution in haemodynamically unstable patients; however, this will only increase rate if the block is at the level of the AVN. When the block is below the level of the AVN the unopposed vagolysis will increase the atrial rate, potentially slow the ventricular rate, and can lead to potentially dangerous ventricular arrhythmias.

BUNDLE BRANCH BLOCKS

Bundle branch blocks result from damage to the His-Purkinje system. This causes broadening of the QRS complex (>120 ms). The most common causes are age-related fibrotic changes, ischaemic heart disease, hypertension, cardiomyopathies, infiltration from systemic disease, cardiac surgery or trauma, and, for RBBB, pulmonary embolism or cor pulmonale. Diagnosis relies on careful examination of the ECG to ensure a supraventricular origin of the impulse.

Left bundle branch block (LBBB) – The left bundle is made up of two branches, the smaller anterior fascicle supplied by septal braches of the left anterior descending artery (LADA) and the posterior fascicle supplied from both the LADA and right coronary artery (RCA). A delayed depolarisation of the left ventricle gives rise to prominent notched R waves in all leads and an 'M' shape is often seen in V6.

If involvement is limited to the anterior fascicle (anterior fascicle hemiblock), then the ECG will show left axis deviation (LAD), and minimal prolongation of the QRS. If limited to the posterior fascicle (posterior fascicle hemiblock), right axis deviation (RAD) greater than 120 degrees will be evident with minimal prolongation of the QRS. Most commonly LBBB is seen with ischaemic heart disease, and development of a new LBBB should be considered as an acute ischaemic event equivalent to ST-elevation, and treated as such.

Right bundle branch block (RBBB) – Delayed depolarisation of the right ventricle produces an RSR pattern in V1 and a prominent S wave in leads I and V6. RBBB is relatively common in the adult population and has been reported in up to 2% of inpatients. However, if a new RBBB develops, it should be considered to be the result of acute ischaemia until proven otherwise and treated as equivalent to ST-elevation.

Bifascicular block – RBBB and L anterior or posterior hemiblock. May progress to trifascicular block or complete heart block. No specific management is required unless the block progresses at which point a pacemaker should be considered.

Trifascicular block – Bifascicular block and first degree AV block. This rhythm may progress to complete heart block. If the first degree block is due to AVN disease, then it is less likely to progress to complete heart block. Assessment by a cardiologist should be sought for patients with this rhythm, and if symptomatic a permanent pacemaker should be inserted.

ACCESSORY PATHWAYS

Conduction from the atria to the ventricles normally occurs via the AVN and His-Purkinje system. Patients with additional conduction tracts are said to have pre-excitation syndromes, because the tracts allow rapid bi-directional conduction of electrical impulses.

The most common of these is the Bundle of Kent. It results in the typical Wolff-Parkinson-White ECG findings of PR shortening (<120 ms), broad QRS (>120 ms) and delta-waves. Patients with accessory pathways are predisposed to AV re-entrant and unstable tachyarrhythmias, which may occur particularly in the context of AF. This can deteriorate to VT or VF.

Such patients should be managed by a cardiac electrophysiology specialist prior to elective procedures. Intra-operative AF can be treated with procainamide or amiodarone. If the patient is compromised by the rhythm, prompt synchronized DC-cardioversion is recommended. AV nodal reentrant rhythms can be treated with IV verapamil or lignocaine, in the absence of haemodynamic instability, or synchronized DC cardioversion if

this is present. For patients with pharmacologically managed arrhythmias, all anti-arrhythmic drugs should be continued in the perioperative period. Intraoperative vagolytic drugs should be avoided and adequate analgesia and depth of anaesthesia should be ensured, particularly during laryngoscopy and surgical stimulus to avoid catecholamine surges precipitating tachyarrhythmias.

LONG QT SYNDROME

The long QT syndrome (LQTS) is a disorder of myocardial repolarization characterized by a prolongation of the QT interval on the ECG. Patients with LQTS usually report palpitations, syncope and seizures and are at high risk of developing torsades-de-pointes and sudden cardiac death. The LQTS may be congenital or acquired. The congenital forms are caused either by autosomal dominant or less commonly by autosomal recessive genetic mutations, almost all of which encode for abnormal cardiac ion channels. The acquired form is usually caused by drug therapy, hypokalaemia or hypomagnesaemia.

ANAESTHETIC MANAGEMENT

Preoperative management by a specialist cardiac electrophysiology team is recommended due to the high risk of sudden cardiac death. Commonly implantable cardiac defibrillators are used to mitigate the risk of fatal arrhythmias. Beta-blockers have also been shown to reduce the QT interval and some patients may be managed with this as single therapy. Responders to beta-blockers as a single therapy appear to have lower risk of malignant arrhythmias intraoperatively. A baseline 12 lead ECG should be performed preoperatively, and correction of electrolytes undertaken, with particular attention paid to potassium and magnesium.

INTRAOPERATIVE MANAGEMENT

General and regional anaesthesia are considered safe although catecholamine surges should be avoided. Where local anaesthetic agents are used, adrenaline should be avoided. Premedication with a narcotic

and benzodiazepine on the morning of surgery is recommended.

Antiarrhythmic drugs, a defibrillator with pacing facility and a temporary transvenous pacemaker should be available in the operating room with defibrillator pads attached to the patient. In addition to routine monitoring, a minimum of two ECG leads should be continuously monitored (one limb and one chest lead), invasive arterial monitoring is advisable and continual temperature measurement is recommended. The allows rapid identification of arrythmogenic factors such as temperature changes, heart rate changes, worsening in ST-segment elevation in Brugada syndrome or lengthening QT interval.

Intraoperative ventricular dysrhythmias in patients who respond to beta-blockers are usually responsive to further beta-blockade. Primidone, bretylium or verapamil may be used in those who do not respond. In both groups, premature ventricular contractions usually respond to lidocaine. Standard advanced cardiac life support protocols (with the possible exception of using epinephrine last) should be followed for ventricular tachycardia or fibrillation.

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CROSS-REFERENCE

Patients with pacemakers and implantable defibrillators, Chapter 2

CARDIOMYOPATHIES

A cardiomyopathy is defined as a change to the heart muscle that results in structural and functional abnormalities in the absence of coronary artery disease, valvular disease, hypertension or congenital heart disease sufficient to explain the observed myocardial abnormality.

The WHO classifies cardiomyopathies in terms of anatomy and physiology as follows; each of these can be further classified into idiopathic or acquired:

- Dilated cardiomyopathy (DCM)
- Hypertrophic cardiomyopathy (HCM)
- Restrictive cardiomyopathy (RCM)
- Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D)
- Unclassified cardiomyopathies

DILATED CARDIOMYOPATHY

Idiopathic DCM has a reported prevalence of 0.4 per 1000, and DCM overall has a likely prevalence of 1 per 2500. It is most commonly diagnosed in the third and fourth decades of life (Table 2.6).

HYPERTROPHIC CARDIOMYOPATHY

Prevalence estimated at 1 in 500 adults.

AETIOLOGY

Usually autosomal dominant inheritance of a number of mutations in genes encoding sarcomeric proteins such as beta-myosin heavy chain and troponin T, including *MYH7, MYBPC3, TNNT2, TNN13*.

Other causes of hypertrophic cardiomyopathy include chronic hypertension and aging.

Table 2.6 Aetiology

Causes of dilated cardiomyopathy	Examples
Hypertensive disease	Secondary to chronic hypertension and cardiac remodeling.
Valvular heart disease	Haemodynamically significant lesions (MR, AS, AR, MS) can lead to cardiac remodeling, hypertrophy, chamber dilatation and DCM.
Viral myocarditis and other infections	~50% of those receiving a diagnosis of acute viral myocarditis develop DCM. Common causes: coxsackievirus B, adenovarius, parvovirus. Other causes: HIV, Lyme disease, *trypanosoma cruzi*, toxoplasmosis and malaria.
Toxic cardiomyopathies	Alcohol (4% of cases), cocaine, amphetamines, anthracycline chemotherapy, e.g. doxorubicin, Herceptin, postpartum cardiomyopathy
Metabolic conditions	Malnutrition, vitamin and nutrient deficiencies, e.g. B vitamins, adrenocortical insufficiency, hyper/hypothyroidism, acromegaly and phaeochromocytoma.
Stress cardiomyopathy	Takosubo cardiomyopathy
Familial cardiomyopathy	Up to 25%, usually autosomal dominant, X-linked autosomal recessive and mitochondrial inheritance also reported.

RESTRICTIVE CARDIOMYOPATHY

Uncommon in the West (<5% of all cardiomyopathies), estimated prevalence of between 1 in 1000 to 1 in 5000. However, endomyocardial fibrosis is a significant cause of heart failure in parts of Africa (Table 2.7).

Table 2.7 Aetiology

Causes of restrictive cardiomyopathy	Examples
Idiopathic/primary	Endomyocardial fibrosis or Loeffler eosinophilic endomyocardial disease
Infiltrative	Haemochromatosis, amyloid, sarcoid, cardiac carcinoid, glycogen storage disease affecting the heart, malignant infiltration
Treatment-related	Radiotherapy, anthracyclines

ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY/DYSPLASIA (ARVC/D)

The prevalence of ARVC/D is estimated to be between 0.2 and 1 cases per 1000 in the general population. Under-recognition remains a significant problem, so this may be an underestimate of the actual burden of disease. ARVC/D is an important cause of sudden cardiac death worldwide, representing the cause of 2%–5% of deaths in adults below the age of 40.

AETIOLOGY

The aetiology of ARVC/D remains unknown. In autopsies, the presence of inflammatory infiltrates in the myocardium has been demonstrated and ARVC/D has been documented post-myocarditis. Presently it is thought that it arises as the result of myocardial inflammation in the context of genetic susceptibility.

ANAESTHETIC CONSIDERATIONS FOR CARDIOMYOPATHIES

PREOPERATIVE MANAGEMENT

Pay particular attention to symptoms of cardiac failure, palpitations, syncope and associated conditions. Review medications and comorbidities. Elicit an accurate family history particularly with regards to sudden cardiac death or early onset CCF. Recent cardiovascular investigations in particular

echocardiogram should be reviewed. In the early stages of the disease patients with cardiomyopathy may have no signs and symptoms. However, with the progression of the disease the classical clinical symptoms of cardiac failure will present.

INTRA- AND POSTOPERATIVE MANAGEMENT

Patients with cardiomyopathies generally behave like patients with heart failure of any cause, and should be managed as such in the perioperative period. Notable exceptions are patients with HOCM, restrictive cardiomyopathies and ARVC/D. Elective admission to a critical care environment should be considered postoperatively for patients with cardiomyopathies.

In patients with HOCM, the LVOT is prone to obstruction, resulting in loss of CO and fatal arrhythmias. This can be exacerbated by reduced LV filling. As such, in HOCM patients CO is said to be filling-dependent. In the perioperative period it is essential to avoid hypovolaemia and tachycardia, and hypotension should be primarily treated with fluid administration. In these patients, the use of invasive arterial BP monitoring and noninvasive cardiac output monitoring or TOE is strongly recommended to guide volume status.

In patients with restrictive cardiomyopathy, the stroke volume is relatively fixed and small, so rapid changes in SVR are not well tolerated. Nodal infiltration may also occur in diseases such as cardiac amyloid resulting in bradyarrhythmias. These should be fully investigated preoperatively. A high-normal heart rate and SVR should be targeted, and bradycardias treated promptly with vagolytic drugs.

In patients with ARVC/D, fatal arrhythmias are the most common cause of death. Such arrhythmias may be triggered during anaesthesia, and usual precautions with optimization of electrolytes preoperatively should occur. If an ICD is *in situ,* the normal precautions of this should apply. An external defibrillator should be connected to patient intraoperatively and only disconnected once the ICD has been reactivated.

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CHILDREN WITH CONGENITAL HEART DISEASE FOR NONCARDIAC SURGERY

The global incidence of congenital heart disease (CHD) is approximately 1 in 125 live births. These patients may present for emergency or elective surgery to nonspecialist centres with corrected, partially corrected or uncorrected disease. The key is to understand the underlying defect, the anatomy you are dealing with, and the functional physiology, then conduct a thorough preoperative evaluation. Despite advances in understanding and management, children with severe or major CHD still have greater than twice the 30-day mortality of matched controls.

There are more than 100 different types of CHD although the underlying pathophysiology allows a useful classification of CHD from a clinical perspective (Table 2.8).

PATHOPHYSIOLOGY

SHUNT LESIONS

Left-to-right shunts

Acyanotic congenital heart disease results from shunting of blood from the left side of the heart to the right side and into the pulmonary circulation. This is the most common pathophysiology in CHD patients. These conditions often manifest in the first two weeks of life due to the high PVR *in utero*. As this falls following delivery, the shunt may become evident with features of cardiac failure. As a consequence of shunting a proportion of the cardiac output to the right ventricle becomes volume overloaded with pulmonary hyperperfusion. The magnitude of the shunt for large defects is mainly dependent on the ratio of pulmonary vascular resistance (PVR) and systemic vascular resistance (SVR). As the defect size reduces, the shunt becomes largely independent of the

Table 2.8 Classification of CHD based on pathophysiology

Shunts	Left-to-right	Right-to-left
	ASD	Tetralogy of Fallot
	VSD	Truncus arteriosus
	AVSD	Complete Transposition
	PDA	Total anomalous pulmonary venous connection
	PFO	L-to-R shunt plus Eisenmenger's syndrome
Obstructive Lesions	LVOT	RVOT
	Aortic stenosis/atresia	Pulmonary stenosis/atresia
	Mitral stenosis	Tricuspid atresia
	Coarctation of aorta	Ebstein anomaly with intact septum
	Interrupted aorta	

resistances and predominantly determined by the size of the defect.

The increased pulmonary flow over time results in pulmonary hypertension as the pulmonary circulation adapts to the increased volumes and right-sided pressures. Once this has developed (a PAP >25 mmHg at rest or >30 mmHg during exercise), these children are eight times more likely to experience a major perioperative complication.

Pulmonary hypertensive crisis may occur pre-operatively following the development of pulmonary hypertension where the PVR exceeds the SVR resulting in pressure overload of the RV and reduced pulmonary blood flow and compression of the LV +/- flow reversal across the shunt. This compromises cardiac output resulting in hypotension, hypoxia and a mixed respiratory and metabolic acidosis and ultimately biventricular failure. If this occurs, treatment to reduce the PVR should be administered: 100% O_2; inhaled nitric oxide or inhaled/intravenous prostacyclin; inotropic support of the RV; reduction in arterial $PaCO_2$.

Eisenmenger's Syndrome

Any uncorrected left-to-right shunting lesion may undergo flow reversal and become a right-to-left shunt. Once a patient has developed pulmonary hypertension in response to the increased flow and resultant damage to the pulmonary capillary beds, the RV undergoes compensatory hypertrophy to maintain forward flow. Right-sided heart pressures now exceed the left, and flow reversal occurs, leading to mixing of desaturated blood in the left heart and resultant systemic cyanosis.

Right-to-left shunts

Cyanotic heart disease results from shunting of desaturated blood from the right heart to the left and to the systemic circulation, bypassing the pulmonary circulation. These lesions carry an increased risk of paradoxical embolism, so particular care to avoid even small volume air embolism or accidental particle administration when administering IV medications and fluids should be exercised. The shunt volume may be reduced by manoeuvres that reduce the PVR and increase SVR, e.g. squatting, administration of 100% O_2, inhaled NO or prostacyclin. Conversely, this can

be exacerbated by falls in systemic vascular resistance and increase in PVR seen in anaesthesia.

Polycythaemia may occur in patients with a right-to-left shunt. Arterial hypoxia is detected by the renal erythropoietin-producing oxygen-sensing cells in the juxtamedullary cortex of the kidneys, and erythropoietin (EPO) is produced in response. The increased erythropoiesis in response to high levels of EPO causes polycythaemia, high reticulocyte count, serum hyperviscosity and a resultant increased risk of thrombosis.

OBSTRUCTIVE LESIONS

Lesions leading to outflow tract obstruction of either the right or left heart, if uncorrected, leave patients with a relatively fixed cardiac output. The increased afterload for either ventricle results in pressure loading and compensatory hypertrophy. Obstructive lesions tend to have higher rates of arrhythmias which may be refractory to classic agents. Patients with severe lesions will rapidly develop symptoms of venous congestion on exertion or in illness. There is also an increased risk of myocardial ischaemia due to the increased ventricular mass and the relatively fixed cardiac output being unable to adapt to increases in demand.

PREOPERATIVE MANAGEMENT

In this patient group is it vital that the anaesthetist has a clear understanding of the functional physiological status of the patient, prior to proceeding with the case. This can broadly be split into two sections underlying circulation and presence of any of the four key risk factors (Table 2.9).

Table 2.9 Physiological risk factors

Physiological risk factors	
Cardiac failure	Arrhythmias
Pulmonary hypertension	Cyanosis

Source: Adapted from White MC and Peyton JM. (2012). *Contin Educ Anaesth Crit Care Pain* **12**(1): 17–22.

FUNCTIONAL CIRCULATION

SERIES "NORMAL" CIRCULATION

A series circulation is where there is a separate pulmonary and systemic circulation, working together. This is seen following complete repair procedures and with some forms of unrepaired lesions such as ASD or VSD. In the latter, blood mixing may occur down the pressure gradient resulting in shunts, as discussed.

PARALLEL CIRCULATION

A parallel circulation is where there is communication between the systemic and pulmonary circulations. Here the blood flow is determined by the resistances to flow, so blood will flow down the path of least resistance. It can be thought of as a balance between SVR and PVR determining the flow of blood to lungs and body. CCF occurs if pulmonary flow is too high with low cardiac output, and cyanosis occurs in the reverse. This situation is seen in infants with large VSDs, modified Blalock-Taussig (BT) shunts and hypoplastic left heart syndrome. Caution should be taken to avoid high concentrations of O_2, and avoid large drops in SVR.

SINGLE VENTRICLE CIRCULATION

In some cases the abnormality may not be amenable to full repair so palliative surgery can be performed. This may be a BT-shunt, Glenn shunt or a Fontan circulation. In these, the blood flow to the lungs is passive down the pressure gradient from the PA to LA. Varying degrees of cyanosis are observed with each of the shunts. These procedures may often be carried out in a stepwise manner in the child's perinatal and infant life. Here careful consideration should be given to ventilation strategies because the pulmonary blood flow is augmented by the negative intrathoracic pressure generated on inspiration in spontaneous breathing. Positive pressure ventilation will restrict pulmonary blood flow so inspiratory times should be kept short, PEEP optimized and high peak pressures avoided.

PREOPERATIVE ASSESSMENT

A thorough history should be taken including the original abnormality, any surgical correction undertaken, current medications reviewed, any issues or hospital admissions since birth for cardiac complications or coexisting disease. Particular attention should be paid to a history of recent upper or lower respiratory tract infections, pulmonary hypertension, CCF, arrhythmias, cyanosis, seizures, failure to thrive, developmental delay and reactive pulmonary disease. All cardiac investigations should be reviewed carefully, and baseline oxygen saturations, heart-rate and blood pressure should be fully documented.

EXAMINATION

A full examination of the child should be undertaken, particularly looking for features of cardiac failure, pulmonary hypertension, cyanosis and evidence of recent respiratory tract infection. These children will normally have cardiac murmurs and should be further investigated if atypical for the type of CHD or repair present. A full airway assessment should be carried out as CHD may be associated with syndromes involving the upper respiratory tract and cervical spine instability.

INVESTIGATIONS

These should be guided by history and examination with the child's underlying condition taken into consideration and any previous investigations performed. Where any doubts occur, these should be discussed with the child's cardiologist and/or specialist team.

ECG – All CHD patients should have a preoperative ECG. Whilst RBBB is common and unlikely to progress to third-degree heart block, the presence of ventricular ectopics (VE) warrants further investigation and consideration of transfer to a specialist centre. This is due to the high incidence of sudden cardiac death in CHD patients with VEs.

Echo – If there is no recent imaging, or there is a recent change in the child's condition, echocardiography should be performed.

Full blood count – Consider in the presence of cyanosis to evaluate for polycythaemia. If present, coagulation and viscosity studies should be considered.

U&Es – Many of the cardiac medications will affect electrolyte balance and renal function.

CXR – Should be performed if there is clinical evidence of cardiac failure or recent respiratory infection.

Cardiac MRI/catherisation – Should be undertaken on advice of specialist team or child's cardiologist.

RISK CLASSIFICATION

A significant part of the preassessment is to allow risk stratification and to enable the surgery to proceed in the safest environment. White and Peyton have proposed a very useful risk stratification system (see reference). For elective procedures, high-risk patients should be transferred to a specialist centre, intermediate-risk patients should be discussed with a specialist centre and considered for transfer, and low-risk patients should be operated on locally if the skill-set allows. In emergency situations, all high- and intermediate-risk patients should be discussed with the paediatric transfer service for feasibility of transfer to a specialist centre. If this is not possible, the case should be discussed with the specialist team and transferred at the earliest safe opportunity. Low-risk patients may be managed locally if the skill-set allows and any concerns discussed with the specialist team.

INTRAOPERATIVE MANAGEMENT

Full monitoring should be undertaken in line with paediatric AAGBI guidance. It should be noted that in CHD the $ETCO_2$ will correlate poorly with $PaCO_2$ and the gap will vary. Consideration of invasive monitoring should be undertaken for all intermediate- and high-risk patients.

PREMEDICATION

In patients with arrhythmogenic conditions, the prevention of a catecholamine surge may be beneficial. However, it may be harmful in patients with pulmonary hypertension if there is fixed cardiac output. Volume status should be considered and prolonged

fasts avoided to prevent dehydration, as this has a twofold effect, both reducing preload and also increasing serum viscosity.

ANTIBIOTIC PROPHYLAXIS

In the current ESC guidelines on the use of prophylactic antibiotics for prevention of infective endocarditis, patients with cyanotic CHD, all CHD where a repair has been made in the previous 6 months with synthetic material or those with a residual shunt or valvular regurgitation are considered high risk. These patients should receive prophylactic antibiotics for high-risk procedures in keeping with local guidelines.

ANAESTHETIC TECHNIQUE

There is a wide variety of anaesthetic techniques described with no significant evidence that any one technique betters another. Propofol and ketamine have been well studied in children with propofol causing a reduction in SVR and CO. In some situations this may not be desirable such as in parallel circulations. Ketamine has relative small effects on SVR, PVR, PAP and MAP making it an ideal induction agent in children with pulmonary hypertension or parallel circulations. Whilst inhalational induction is considered safe, slower inductions with low concentrations of volatile agents should be considered. Intravenous access where possible should be obtained preinduction, or a second consultant anaesthetist or senior paediatrician should be present in the anaesthetic room such that IV access can be quickly secured postinduction.

Maintenance of anaesthesia with sevoflurane or isoflurane has been well studied and both have minimal deleterious effects. Ketamine and opiate infusions are also considered safe in this population although propofol infusions should be avoided due to the reduction in SVR and cardiac output. Regional anaesthesia is considered safe in CHD if it is routine practice for the procedure being undertaken.

POSTOPERATIVE MANAGEMENT

Provision of monitoring and timely intervention is key to successful postoperative recovery, with meticulous fluid balance and maintenance of baseline parameters with supplemental oxygen and drugs

as required. Elective admission to a paediatric critical care unit should be considered for all high- and intermediate-risk patients, or those with slow progression to baseline function postoperatively.

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CROSS-REFERENCES

Cardiac conduction defects, Chapter 2
Pulmonary hypertension, Chapter 2
Patients with heart failure, Chapter 2

CONGENITAL HEART DISEASE IN ADULT LIFE

With advances in surgical techniques and supportive management, there are increasing numbers of adults with grown-up congenital heart disease (GUCHD). Up to 90% now survive to adult life. In 2010 it was estimated that there were approximately 200,000 GUCHD patients in the UK and 1.2 million in Europe. There are significant physiological and anatomical consequences that impact anaesthetic assessment and practice in this specialized patient group.

Broadly speaking, GUCHD can be classified into simple, moderate or severe complexity rather than using an extensive anatomical classification (Table 2.10). These patients can also be classified into uncorrected, corrected and palliated.

Table 2.10 Congenital heart lesions by complexity

Simple	Moderate	Severe
Isolated aortic valve disease	Aorta to LV fistula	Conduits
Isolated mitral valve disease	Partial or total anomalous pulmonary drainage	Cyanotic congenital heart disease
PFO, small ASD or VSD	AV canal defects	Fontan procedure or TCPC
Previously repaired PDA	Coarctation of the aorta, or unrepaired PDA	Single ventricle circulation
Sinus venosus or secundum ASD (repaired)	Moderate to severe pulmonary stenosis or regurgitation	Transposition of the great vessels
VSD (repaired)	Tetralogy of Fallot	Mitral, tricuspid or pulmonary atresia
Mild Pulmonary stenosis	VSD with associated abnormality	Other rare congenial heart disease

Abbreviations: ASD = atrial septal defect; PDA = patent ductus arteriosus; PFO = patent foramen ovale; VSD = ventricular septal defect; TCPC = total cavo-pulmonary connection.

COMPLICATIONS OF GUCHD

Patients who have undergone surgical repair, palliation or unrepaired lesions are at risk of developing cardiac and noncardiac complications. The most common are electrophysiological disturbances, but this also includes cardiac failure, sudden cardiac death, infective endocarditis, bleeding and thromboembolic disease, renal impairment, airway stenosis, nerve palsies, altered blood flow and chronic hypoxaemia.

ELECTROPHYSIOLOGICAL CHANGES

These are both the most common and most significant consequences of GUCHD. Most commonly abnormalities of conduction are seen rather than SA nodal dysfunction. These can occur due to the altered anatomy and physiology, chronic hypoxia or as a direct consequence of surgical injury. Patients most prone to arrhythmias include those with history of ventriculotomy or lesions resulting in structural changes to the right side of the heart.

Anti-arrhythmic drugs are often tolerated poorly due to the negative ionotropic effects and should be used with caution. Bradycardia and deterioration to nonsinus rhythm is generally poorly tolerated, and should be avoided where possible. Cardioversion should only be carried out following anticoagulation and echocardiographic evaluation except in life-threatening circumstances due to the increased risk of cardiac thrombus.

CARDIAC FAILURE

Cardiac failure commonly develops as a consequence GUCHD due to the physiological and anatomical changes. Cardiac failure is generally treated in line with guidance on acquired disease and anaesthetic management should be similar to that for all patients with cardiac failure, with a low threshold for ionotropic support. For many GUCHD patients with cardiac failure, heart transplant is the only option although this surgery has increased complexity than in normal patients.

THROMBOEMBOLIC DISEASE AND BLEEDING TENDENCY

Abnormal anatomy, increases in turbulent flow and chronic hypoxia with compensatory erythrocytosis

lead to an increased risk of intracardiac thrombus formation and also venous thrombosis. The use of pharmacological and mechanical thromboprophylaxis is essential perioperatively, with early mobilization post-surgery.

Conversely, large numbers of patients have established anticoagulation or antiplatelet therapy following shunt insertion, valvular surgery, device closure or following arrhythmias. Patients will often run a low-normal platelet count and may have abnormalities of function, particularly where the cardiac abnormality is part of a systemic syndrome.

INFECTIVE ENDOCARDITIS

Patients with GUCHD have an increased risk for the development of infective endocarditis. In recent years, guidelines have restricted the scope of use of prophylactic antibiotics. In general, prophylactic antibiotics are recommended for all untreated cyanotic heart disease and in patients with postoperative palliative shunts or other prosthesis. After surgical repair where no residual defects remain once endothelialisation has occurred normally at six months the risk is significantly reduced. Patients with a prosthetic valve and GUCHD should be treated as other patients with prosthetic valves. Current European Society of Cardiology guidelines recommend that inantibiotic prophylaxis is restricted to the above groups unless high-risk procedures are being undertaken. In patients with GUCHD, strict asepsis should be observed when inserting invasive lines or undertaking any invasive procedures.

HYPOXAEMIA

In patients with congenital heart disease, chronic as opposed to acute hypoxaemia is a common symptom and is usually associated with reduced pulmonary blood flow and/or right-to-left shunting. Hypoxaemia is a concomitant feature of arrhythmias, concurrent cardiac failure and pulmonary disease.

AIRWAY ABNORMALITIES

Tracheal or bronchial stenosis is commonly seen in patients with GUCHD. This may result from

prolonged periods of intubation and ventilation or external compression from enlarged or malpositioned vessels. Prosthetic conduits may also result in scarring and stricture formation.

PREOPERATIVE MANAGEMENT

Congenital heart disease confers an increased perioperative risk from cardiac and noncardiac causes. It is vital that all but the simplest of conditions and situations be discussed with a specialist centre and transfer to a cardiac centre be considered.

A thorough history should be elicited, with particular attention to symptoms of heart failure, palpitations, arrhythmias, syncope, previous infective endocarditis and functional capacity. Details of the original abnormality and any corrective/palliative surgery undertaken and any recent reviews from their specialist team. Medications should be reviewed, and a history of comorbid disease sought. All recent cardiovascular investigations, in particular echo and exercise testing, should be reviewed. If present, arrhythmias may be symptomatic with palpations, dizziness or syncopal episodes. Features of heart failure may also be described.

On examination look for:

- Scars suggestive of previous thoracotomy or permanent pace maker.
- Pulse – Normal; low volume in low cardiac output states; irregularly irregular in AF.
- Central or peripheral cyanosis.
- JVP – May be raised, and/or distended neck veins may be seen.
- Apex bet – Displaced in the context of volume-overload.
- Cardiac murmurs.
- S_3 with gallop rhythm may be audible on auscultation.
- Basal crepitations may indicate pulmonary oedema.
- Peripheral oedema.

INVESTIGATIONS

- *ECG* – Arrhythmias, conduction defects, evidence of current or previous ischaemia.
- *CXR* – Cardiomegaly, pulmonary congestion (bat-wing shadowing), Kerley B lines (interstitial oedema), pleural effusions. Shunts, permanent pacemakers and artificial valves may be visible.
- *Echo* – Recommended for all patients undergoing intermediate or high-risk noncardiac surgery – consider presence, quantification, timing and degree of ventricular dysfunction, structural abnormalities and valvular lesions. Regional wall motion abnormalities may be seen with ischaemic heart disease, or myocardial thickening in myocarditis.
- *FBC* – Low platelet count may be present, or reactive polycythaemia.
- *U&Es* – most cardiac medications affect electrolyte balance and renal function; look for cardio-renal syndrome, AKI or CRF. Evaluate and optimise preoperatively.

INTRAOPERATIVE MANAGEMENT

MONITORING

- *ECG* – At least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced with abnormal baseline ECG.
- Invasive arterial blood pressure (start before induction).
- *TOE* – To monitor filling, although an experienced operator is required. Strongly indicated for mixed valvular disease, where early detection of regional wall motion abnormalities is of greater importance.
- *Noninvasive cardiac output monitoring* – Useful for monitoring filling status. Although absolute values are unlikely to be accurate, response to fluid boluses do show incremental changes with pulse contour wave analysis, and Doppler flow.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a normal resting heart rate. Patients are particularly sensitive to tachy- and brady-arrhythmias and rhythm changes.
- *Blood pressure* – Aim for a normal blood pressure. A low normal SVR should be

maintained by meticulous attention to volume status to ensure adequate filling but avoid overload. Consider preloading with intravenous fluid boluses when fluid shifts are expected.

- *Dysrhythmias* – Treat in line with current ALS recommendations. A defibrillator should always be available in theatre for rapid DC cardioversion if required.
- *Other* – Avoid hypoxia, hypothermia, hypercarbia and acidosis, which may increase reduced myocardial function and increase PVR.

ANAESTHETIC TECHNIQUE

Anaesthetic technique should take account of the specific anatomical and physiological impact of the malformation. The uptake of inhalation agents can be affected by intra-cardiac shunting. An R-L shunt will reduce the pulmonary blood flow and hence the partial pressure in blood going to the brain resulting in a slower uptake; however, intravenous agents will have a more rapid onset. Peripheral nerve blocks are recommended where possible to reduce the haemodynamic impact of general anaesthesia. Central neuraxial blockade should be used with extreme caution due to the sensitivity of these patients to preload and poor tolerance of bradycardia.

POSTOPERATIVELY

All patients should have supplemental oxygen and good analgesia is essential to minimise catecholamine release. Following medium- or high-risk procedures, or where large volume shifts have occurred, monitoring in ICU is indicated with invasive blood pressure monitoring, to detect early signs of decompensation. All patients with cyanotic heart disease should be managed in ICU or a specialist cardiac centre.

PREGNANCY AND GUCHD

A significant proportion of maternal deaths during pregnancy are accounted for by GUCHD patients. However, the majority of patients, particularly those with simple lesions, can undergo pregnancy without difficulty to mother or fetus. Counselling should be advised prior to pregnancy and this should be

lesion-specific by a specialist team. For example, patients with R-L shunts are more susceptible to changes in SVR and flow across the shunt will increase potential resulting in cyanosis.

During labour there is an increased risk of developing infective endocarditis. Caesarean section should be reserved for obstetric reasons as far as possible, with planning by the obstetric, cardiac and anaesthetic teams crucial throughout the pregnancy. Induction of labour should only be considered in a specialist centre if there is evidence of cardiac decompensation.

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CROSS-REFERENCES

Atrial septal defect, Chapter 2
Cardiac conduction defects, Chapter 2
Medical problems and obstetric anaesthesia, Chapter 12
Heart failure, Chapter 2
Patients with pacemakers and implantable defibrillators, Chapter 2
Pulmonary hypertension, Chapter 2
Tetralogy of Fallot, Chapter 2

HEART FAILURE

Heart failure (HF) can be defined as any structural or functional cardiovascular abnormality leading to systemic perfusion unable to meet the body's

metabolic demands without excessive increase in LV filling pressures.

Two major classification systems are in clinical use. The New York Heart Association (NYHA) scale classifies severity in terms of functional limitation and degree of activity required to elicit symptoms (Box 2.1). The American College of Cardiology/American Heart Association (ACC/AHA) guidelines (Box 2.2) categorizes the stages of heart failure into distinct groups. This emphasizes the progressive nature of heart failure and allows for intervention based on stage.

BOX 2.1: NYHA functional classification of heart failure severity

- Class I – Patients with heart disease that does not limit physical activity and an absence of symptoms (fatigue, palpitation, dyspnoea) on ordinary physical activity.
- Class II – Patients with heart disease resulting in mild limitation of physical activities or symptoms of HF developed with ordinary physical activity but absent at rest.
- Class III – Patients with heart disease resulting in significant limitation of physical activity or symptoms of HF on minimal activity but absent at rest.
- Class IV – Patients with heart disease resulting in the inability to carry on any physical activity without discomfort. If any physical activity is undertaken discomfort increases.

BOX 2.2: AHA/ACC stages of heart failure development

- Stage A – At risk of HF but without structural heart disease or symptoms of HF (fatigue, palpitation, dyspnea).
- Stage B – Structural heart disease but without signs or symptoms of HF.
- Stage C – Structural heart disease with prior or current signs or symptoms of HF.
- Stage D – Refractory HF requiring specialized interventions.

EPIDEMIOLOGY

Prevalence is estimated at 2–20 per 1000 population and increases steeply with advancing age. Median age at diagnosis in the UK is 76 years. More than 37.7 million people are thought to suffer from heart failure worldwide. The 900,000 patients in the UK account for 5% of all emergency admissions and 2% of inpatient bed-days.

AETIOLOGY

Heart failure most commonly occurs in developed countries as a sequel of chronic hypertension or valvular heart disease (Table 2.11).

PATHOPHYSIOLOGY

Changes seen in HF result from physiological changes in response to cardiac injury. Raised EDV is compensated for by LVH and increased wall stress, with initial increase in SV and contractility. This is unsustainable with continuing volume overload and leads to fibrotic changes in the myocardium and LV

Table 2.11 Classification of cardiac failure

	Causes
Left-sided heart failure	Ischaemic heart disease or hypertensive heart disease (commonest cause)
	Valvular heart disease
	Cardiomyopathy
	Myocarditis
Right-sided heart failure	Lung disease with pulmonary hypertension, e.g. end-stage COPD, ILD, and resultant cor-pulmonale
	Right-sided cardiomyopathy, e.g. arrhythmogenic RV dysplasia (rare)
Congestive or biventricular heart failure	RV failure due to pulmonary hypertension and fluid overload as a result of LV dysfunction
High output failure	Anaemia
	Pregnancy
	Hyperthyroidism
	Paget's disease of bone
	Arteriovenous malformations

dilatation with impaired active relaxation and hence ventricular filling.

Various pathways are involved in the development of HF and are targeted therapeutically. The Renin-Angiotensin-Aldosterone System (RAAS) plays a key role in pathogenesis. It is up-regulated due to renal hypoperfusion and sympathetic stimulation, resulting in increased circulating angiotensin II, which causes vasoconstriction, salt and water retention and ventricular remodeling.

CLINICAL MANAGEMENT

Patients with clinical evidence of HF, active symptoms or features present on examination are at significantly increased risk of perioperative complications. This is reflected by its inclusion in the majority of risk indices. In the developed world, the prevalence of heart failure continues to increase steadily as the population ages resulting in more patients presenting for surgery with active disease. Patients with severe heart failure LVEF <30% and/or NYHA class IV have high rates of perioperative cardiovascular complications and increased mortality. Where urgency of surgery allows, these patients should be optimized medically preoperatively and counseled about the risks. There is no clear evidence demonstrating difference in mortality for patients with isolated diastolic HF (preserved LVEF) versus systolic HF highlighting heart failure and functional limitation as key determinants perioperatively.

Acute decompensation provides the greatest risk to the patient and should be evaluated and treated by a cardiologist. Elective surgery should be deferred until this has occurred and ideally a stable baseline achieved for at least two weeks for patients with known chronic HF or at least three months following a new diagnosis of severe HF although urgency of surgery may preclude this. For emergency surgery, haemodynamic optimization should occur with echo evaluation as soon as feasible and pre-/postoperative natriuretic peptide levels. Postoperative care will likely be in an ICU with involvement of the cardiology team.

PREOPERATIVE MANAGEMENT

Pay particular attention to symptoms of HF, history of associated conditions and functional capacity.

Review medications and comorbidities. Recent cardiovascular investigations, in particular echo and exercise tests should be reviewed and noted. If a new diagnosis of HF is made, or presence of new symptoms or poor symptom control is elicited, a cardiology opinion should be sought preoperatively where possible. Symptoms include:

- Fatigue
- Exertional dyspnoea
- Paroxysmal nocturnal dyspnoea
- Orthopnoea

If present, arrythmias may be symptomatic with palpations, dizziness or syncopal episodes. If HF is secondary or in the presence of coexisting CAD, the patient may report angina episodes. Signs of gout may be present and signs of chronic renal failure in the presence of cardiorenal syndrome.

EXAMINATION

- Pulse may be regular, of low volume if a low cardiac output state is present or irregularly irregular in the presence of AF.
- JVP may be raised and/or distended neck veins may be seen.
- The apex may be normal or displaced in the context of volume-overload.
- S_3 with gallop rhythm may be audible on ausculataion of the precordium.
- Bibasal crepitations are heard on auscultation of the posterior aspect of the chest wall with pulmonary oedema
- Peripheral oedema will result in pitting ankle and sacral swelling and weight gain.
- Hepatomegaly (may be pulsatile) can result from right-sided failure with or without TR portal flow reversal on Doppler studies.

INVESTIGATIONS

- *ECG* – Look for arrhythmias, conduction defects, or evidence of current or previous ischaemia.
- *CXR* – Cardiomegaly and pulmonary congestion (bat-wing shadowing) can be seen; Kerley B lines may be present at the lung margins from interstitial oedema; bilateral pleural effusions may also be seen.

- *Echo* – The key investigation and recommended for all patients undergoing intermediate or high-risk noncardiac surgery. It allows quantification of the timing and degree of ventricular dysfunction, presence of congenital or acquired structural abnormalities and assessment of valvular lesions. Regional wall motion abnormalities may be seen with ischaemic heart disease or myocardial thickening in myocarditis.
- *Noninvasive stress testing* – Recommended for patients with new symptoms of heart failure presenting for noncardiac surgery, patients with poor functional capacity <4 METs or NYHA III/IV patients with good functional capacity >4 METs, or NYHA I/II presenting for high risk noncardiac surgery.
- *Serum natriuretic peptides* – Review preoperatively in the absence of recent levels and postoperatively, for all intermediate and high risk noncardiac surgical procedures.
- *Full blood count* – Anaemia may be present and require correction pre- or perioperatively.
- *U&Es* – The majority of cardiac medications can impact on electrolyte balance and re*ν*αl function, as well as the possibility of cardiorenal syndrome, AKI or CRF. These should be evaluated and optimized preoperatively.

INTRAOPERATIVE MANAGEMENT

- *ECG* – Monitoring of at least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced due to underlying LVH.
- *Invasive arterial blood pressure monitoring (from pre-induction)* – To allow rapid recognition of haemodynamic changes.
- *TOE* – May be considered to monitor filling, although an experienced operator is required. This is strongly indicated for mixed valvular disease, where early detection of regional wall motion abnormalities is of greater importance.
- *Noninvasive cardiac output monitoring* – May be useful for monitoring filling status although absolute values are unlikely to be accurate. Response to fluid boluses does show

incremental changes with pulse contour wave analysis and Doppler flow.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a low normal heart rate 50–70 bpm. Avoid tachycardia as this predisposes to arrhythmias and reduces diastolic LV filling and cardiac output.
- *Blood pressure* – Aim for normal blood pressure. A low-normal SVR should be maintained by meticulous attention to volume status to ensure adequate filling but avoid overload. Consider preloading with intravenous fluid boluses when fluid shifts are expected.
- *Dysrhythmias* – Treat in line with current ALS recommendations. A defibrillator should always be available in theatre for rapid DC cardioversion if required.
- *Other* – Avoid hypoxia, hypothermia, hypercarbia and acidosis which may increase reduced myocardial function and increase PVR.

ANAESTHETIC TECHNIQUE

Avoid increases in afterload and tachycardia. There is no clear evidence for benefit of general anaesthesia or neuraxial techniques. A balanced approach should be taken with consideration of normal practice for the procedure. Where EF is <30%, local or regional techniques should be utilized for peripheral surgery where possible. On the morning of surgery, all antifailure, antiarrhythmic and antianginal medications should be given. Nitrates can be administered transdermally in the perioperative period until an oral route is reestablished. Digoxin can be converted to intravenous preparations for the perioperative period.

POSTOPERATIVELY

All patients should have supplemental oxygen. Good analgesia is essential to minimise catecholamine release. Following high-risk procedures, or where large volume shifts have occurred, monitoring in ICU is indicated with invasive blood pressure

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monitoring to detect early signs of decompensation. Postoperative natriuretic peptide monitoring is advised in this patient group for intermediate or high-risk surgeries or in high-risk patients, with involvement of the cardiology team.

MANAGEMENT OF ACUTE HEART FAILURE

Acute heart failure will normally present with symptoms of pulmonary oedema, decreased saturations, increased respiratory rate and dyspnea. For intubated and ventilated patients, the only signs may be increasing O_2 requirement, decreasing saturations and altered lung mechanics.

In awake patients:

1. Rapid assessment and simultaneous interventions following the ABC framework.
2. Summon assistance.
3. Sit the patient up and administer oxygen via a reservoir mask or CPAP device with maximal FiO_2 and flowrate. This can be titrated down to effect. Intubation and ventilation may be required if unresponsive to supplemental oxygen.
4. Ensure full AAGBI recommended monitoring is applied.
5. Treat any haemodynamically unstable rhythms as per ALS algorithms.
6. Intravenous vasodilators should be considered, most commonly a GTN infusion if systolic BP is >100 mmHg.
7. Consideration of bolus dose or infusion of furosemide if volume overload is suspected. This will also have a venodilatory effect, which can be of benefit acutely.
8. If shock is present (systolic BP <90 mmHg) consider initiation of inotropic therapy. Enoximone, milrinone, dopamine, dobutamine or dopexamine may be used, and levosimendan can also be added as an adjunctive therapy.
9. Bedside echo, lung USS and CXR should be performed to confirm diagnosis and severity.
10. Patients should be reviewed by the ICU team and consider admission to ICU.

MEDICAL MANAGEMENT OF CHRONIC HEART FAILURE

This is aimed at preventing maladaptive remodeling and suppression of the neurohumoral response. Current AHA/ACCF and ESC guidelines recommend treatment based on the ACC/AHA disease staging.

- *Stage A* – Cessation of smoking, treatment of hypertension aiming for a systolic <140 mmHg and diastolic <80 mmHg, improving glycaemic control in diabetes, treatment of hyperlipidemia, supported weight loss in obesity to BMI <35, and consideration of ACE-I/ARB.
- *Stage B* – Treatments in stage A, plus all patients should receive ACE-i/ARB if not already initiated, consideration of beta-blocker therapy, avoidance of NSAIDs, and consideration of mineralocorticoid receptor antagonist. In the presence of CAD or valvular heart disease, intervention should be considered.
- *Stage C & D* – As stage A and B plus patients should receive education about HF and social support to facilitate self-care. A sodium-restricted diet should be undertaken. Graded exercise training is recommended. Addition of a mineralocorticoid receptor antagonist if not contraindicated for NYHA class II-IV disease or EF <35%. Consider addition of hydrazine and Isosorbide Dinitrate in African populations with NYHA II-IV disease or in other populations where ACE-i/ARB/beta blockers are contraindicated. Digoxin may be of benefit in the presence of AF. Cardiac resynchronization therapy is indicated for patients with ECG evidence of LBBB and QRS prolongation >150 ms, in dilated cardiomyopathy or for those with poor response to medical therapy.

Beta-blockers counteract the effects of sympathetic stimulation and prevent adverse remodeling. All patients should be considered for beta-blockade (carvedilol, bisoprolol). Caution should be exercised in the context of diabetes and bradycardia.

All patients should be considered for ACE-I therapy. These reduce circulating angiotensin-II and aldosterone levels by preventing conversion of angiotensin-I to angiotensin-II. This promotes vasodilatation and naturesis, as well having beneficial effects on myocardial remodeling.

When ACE-I therapy is contraindicated due to angioedema, intolerable cough or other sensitivity, angiotensin II receptor blockers are an alternative.

Aldosterone antagonists reduce sodium retention and have a weak diuretic effect. Mortality benefit has be demonstrated in the RALES trial for patients with NYHA II-IV or EF <35%.

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CROSS-REFERENCE

Coronary artery disease, Chapter 2

HYPERTENSION

Hypertension is the chronic elevation of blood pressure (BP) ≥140/90 if under the age of 60 or ≥150/90 over the age of 60. When untreated it is associated with an increased risk of stroke, ischaemic heart disease, heart failure, renal disease, retinopathy and mortality.

Hypertension may be discovered at preoperative screening although AAGBI guidance advises against diagnosis of hypertension in the preoperative assessment. Diagnosis is now heavily reliant on primary care physicians with increased emphasis on home and ambulatory blood pressure monitoring. The classification of hypertension has been simplified in a recent joint national committee report to four levels – normal, prehypertensive and hypertensive stages one and two. BP <120/80 is regarded as normal for all age groups. BP 120–139/80–89 in patients under 60 or 120–149/80–89 in those over 60 is defined as prehypertensive. Hypertensive stage 1 is defined as BP 140–179/90–109 under 60; 150–179/90–109 over 60. BP 180/110 or greater defines hypertensive stage 2 in all age groups.

EPIDEMIOLOGY

The Health Survey for England National 2014 report found that UK prevalence of hypertension in adults was 32% for men and 27% for women, of which 17% and 11% had uncontrolled hypertension, respectively. These figures have remained largely unchanged for the past few years. More than half of those with hypertension are over the age of 60 years.

AETIOLOGY

Hypertension is either primary (essential) or secondary. Ninety to ninety-five percent of cases are primary, which is a multifactorial process related to inheritance of various genetic polymorphisms and environmental factors such as diet, obesity, alcohol intake, etc.

Secondary cases are much less common and can be due to renal (2.5%–6%), vascular, endocrine (1%–2%), neurogenic or pharmacological causes (Table 2.12).

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Table 2.12 Cause of secondary hypertension

	Secondary causes of hypertension
Renal	Polycystic kidney disease, chronic kidney disease, renovascular disease, urinary tract obstruction, renin-producing tumour, Liddle syndrome
Vascular	Coarctation of the aorta, vasculitis, collagen vascular disease
Endocrine	Primary hyperaldosteronism, Cushing syndrome, phaeochromocytoma, congenital adrenal hyperplasia, hyper/hypothyroidism, hyperparathyroidism, acromegaly
Neurogenic	Brain tumour, Bulbar poliomyelitis, intracranial hypertension
Pharmacological	Alcohol, cocaine, calcineurin inhibitors, NSAIDs, erythropoietin, decongestants and herbal remedies containing ephedrine, herbal remedies containing licorice, nicotine
Other	Obstructive sleep apnoea, pregnancy-induced hypertension

PATHOPHYSIOLOGY

The pathophysiology is multifactorial, highly complex, and highly dependent upon the aetiology. Development of essential hypertension is largely thought to be due to failure or pathogenic changes of one or multiple pathways with roles in regulation of blood pressure. These include, but are not limited to, the renin angiotensin aldosterone system, neurogenic control via sympathetic activity, ANP-regulated natriuresis and diuresis, the kallikrein-kinin system, endothelial mechanisms (NO and ET-1), adrenal steroids, sodium and water excretion and renomedullary vasopressin. In younger patients <60 years of age, cardiac output is often elevated,

whilst in patients over 60 years, increased SVR and stiffness play a dominant role.

Current AHA/ACC guidelines list hypertension as a 'minor' risk for perioperative cardiac events, and it remains unclear if postponing surgery for correction confers any benefit to mortality or morbidity. Despite this, hypertension remains the largest single cause of cancellation or postponement of elective surgery in the UK.

PREOPERATIVE MANAGEMENT

Preoperative practices have changed in the wake of new guidance from AAGBI and British Hypertension Society. Current practice is as follows:

- Patients should be referred for elective procedures by primary care physicians if mean BP readings in primary care for the last 12 months are less than 160/100 mmHg, or if they remain hypertensive despite optimal treatment or decline antihypertensive treatment.
- Preoperative assessment clinics need not measure blood pressure if the above criteria are documented in the GP referral letter.
- If no documented blood pressure is available from primary care, BP should be measured in preoperative assessment clinic and should only be referred back to the GP for investigation and/or management if BP exceeds 180/110 mmHg.

MANAGEMENT OF CHRONIC ANTIHYPERTENSIVE MEDICATION

Oral antihypertensive medications should be continued up to the time of surgery because, with a few exceptions, this is relatively safe. Abruptly discontinuing some medications (beta-blockers and clonidine) may be associated with significant rebound hypertension. Most antihypertensive agents can be taken with small sips of water on the morning of surgery. Dose titration should be performed by the patient's primary care physician or cardiologist if preoperative optimisation is required. It should be noted that patients on antihypertensives can have larger intraoperative fluctuations of BP in response to medications and stimuli than normotensive patients.

Angiotensin converting enzyme inhibitors (ACE-I) and angiotensin II receptor blockers (ARB) can theoretically blunt the compensatory activation of the renin-angiotensin system during surgery and result in prolonged hypotension. One study of 150 vascular surgery patients found that the incidence of hypotension during induction was significantly lower in patients who stopped taking captopril or enalapril the evening before surgery, than in those who took the medication on the morning of surgery. Current practice varies between institutions and from procedure to procedure. In general it would be reasonable to withhold ACE-I/ARB medication in prolonged surgeries or where there is a risk of acute kidney injury, surgeries where large volume shifts are anticipated and high-risk surgery; otherwise, these should be continued.

Beta-blockers reduce intraoperative myocardial ischaemia. In addition to a rise in blood pressure, withdrawal in patients with underlying coronary disease can lead to accelerated angina, myocardial infarction or sudden death. Perioperative beta-blockade must be incorporated and considered in any beta-blocker protocol, with the goal of avoidance of bradycardia and hypotension.

Patients receiving calcium channel blockers may have an increased incidence of postoperative bleeding probably due to inhibition of platelet aggregation. The multiple benefits of these drugs probably outweigh the small risk of continued therapy.

Patients in whom chronic diuretic therapy has caused hypo- or hyperkalaemia may have potentiation of the effects of muscle relaxants as well as a predisposition to cardiac arrhythmias and acute kidney injury.

POSTOPERATIVE HYPERTENSION

Postoperative hypertension is a relatively common phenomenon occurring in up to 2% of cases. It is normally manifest within the first 30 minutes postoperatively and commonly lasts less than 2 hours. A history of hypertension is the most important risk factor and it occurs more commonly where antihypertensive medications have been withheld preoperatively.

INDICATIONS FOR TREATMENT

Any significant rise in blood pressure postoperatively should be treated immediately. Common causes of hypertension such as pain, agitation, hypercarbia, hypoxia, hypervolaemia and bladder distention should first be excluded or treated. Routine antihypertensive medications should be restarted postoperatively and if the morning dose was missed, an additional statin dose may need to be administered. In the context of hypertension, if any focal neurological signs or lateralizing neurology occurs, urgent evaluation by an acute stroke physician is merited. If despite the above measures the patient's BP remains significantly higher than normal or >180/110 mmHg, treatment with labetalol, esmolol, or GTN infusion should be administered with invasive arterial monitoring and transfer to ICU.

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CROSS-REFERENCES

Heart failure, Chapter 2
Intraoperative hypertension, Chapter 30

MITRAL VALVE DISEASE

MITRAL STENOSIS

The incidence and prevalence of MS in developed countries has declined significantly with the reduction in cases of rheumatic fever. Current estimated incidence is 1 in 100,000. It is around twice as common in women as men. Incidences in developing countries are far higher, e.g. India has 100–150 cases per 100,000 and Africa 35 per 100,000.

AETIOLOGY

More than 90% of cases are rheumatic in origin. Other causes include severe mitral annular calcification, infective endocarditis, inflammatory conditions (SLE and RA), left sided carcinoid syndrome, congenital causes (cor triatriatum, Shone's syndrome, mucopolysaccharidosis) and iatrogenic (methysergide or ergot alkaloid valvulopathy).

PATHOPHYSIOLOGY

The predominant cause of narrowing of the mitral valve is the rheumatic disease process. It results in commissural fusion, and retraction of the leaflets and chordae tendinae may occur. Postinflammatory calcification then occurs with a progressive course. The commissural fusion is responsible for the reduced orifice size and loss of physiological adaption to flow. The major haemodynamic consequence is pressure loading of the left atrium which initially compensates by hypertrophying. This however consequently raises the postcapillary pulmonary resistance, causing an increase in PAP. The low pressure RV hypertrophies to maintain CO until it can no longer compensate. When it dilates, symptoms of right ventricular failure and pulmonary hypertension (PH) are seen. A secondary consequence of pressure loading the LA is that when it can no longer compensate for the increased valvular resistance to flow, it will dilate resulting in a pro-arrhythmogenic state. Pressure volume loops are illustrated in Figure 2.2.
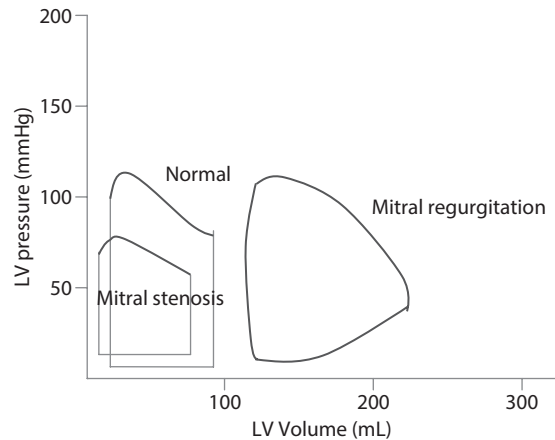
Figure 2.2 Pressure volume loops for mitral valve disease.

CLINICAL MANAGEMENT

Asymptomatic patients with minor to moderate MS undergoing noncardiac surgery will normally tolerate anaesthesia well with no significant mortality or morbidity. However, in the presence of symptoms, echocardiographic evidence of RV dysfunction and PH or in severe disease (valve area <1.0 cm^2), there is increased mortality and cardiac morbidity associated with the perioperative period. These patients should have elective surgery postponed, and referral to cardiology for evaluation and consideration of MV surgery or balloon commissurotomy.

PREOPERATIVE MANAGEMENT

Pay particular attention to symptoms of MS, PH or cardiac failure. Review medications and comorbidities. Recent cardiovascular investigations, in particular echo and ET, should be reviewed and noted. If a new diagnosis of MS is made, or presence of new symptoms or poor symptom control is elicited, a cardiology opinion should be sought preoperatively.

Symptoms of heart failure, angina and AF may be present. Rarer presentations include a hoarse voice from compression of the left recurrent laryngeal nerve by the enlarged LA (Ortner's syndrome) and features of infective endocarditis.

EXAMINATION

- General features – Malar flush, left thoracotomy scar from previous mitral valvotomy, signs of heart failure, bruising from anticoagulation.
- Pulse may be irregularly irregular in AF.
- JVP – Elevated with prominent CV wave due to tricuspid regurgitation from pulmonary hypertension.
- Apex – Undisplaced and tapping (loud S1). Left parasternal heave from pressure overloaded RV.
- Mid-diastolic low rumbling murmur best heard in the left lateral position at end-expiration. Loud S1 'closing snap' and loud S2 (from P2 due to pulmonary hypertension), and an 'opening snap' shortly after P2 due to doming of the anterior mitral valve leaflet in diastole.
- Other valvular lesions may be evident as rheumatic fever commonly affects multiple valves, and mixed MV disease is also common.

INVESTIGATIONS

- *ECG* – p-mitrale (bifid broad p waves >40 ms), atrial fibrillation and atrial flutter are common findings.
- *Echo* – Evaluation of severity of valvular lesion and ventricular function (Table 2.13). PH may be observed with RVH and/or RV dysfunction (see pulmonary hypertension section). An atrial thrombus may also be visualized due to the propensity for AF.

Table 2.13 Echocardiographic evaluation of mitral stenosis severity

	Valve area (cm^2)
Normal	4–6
Mild	>1.5
Moderate	1.0–1.5
Severe	<1.0

Note: In sinus rhythm at heart rate 60–80 bpm.

- *Cardiac catheterization* – Evaluation of RV function, presence and quantification of pulmonary hypertension, valve gradient and concurrent CAD.
- *Exercise stress testing* – Consider to identify and evaluate coexisting CAD.

INTRAOPERATIVE MANAGEMENT

MONITORING

- *ECG* – Monitoring of at least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced due to underlying LVH.
- *Invasive arterial blood pressure monitoring (from preinduction)* – To allow rapid recognition of haemodynamic changes.
- *TOE* – This allows accurate evaluation of ventricular filling and provides the most useful information where rapid volume shifts are expected, can also identify regional wall motion abnormalities. However, this requires an experienced operator.
- *Pulmonary artery catheter* – Not recommended as measurements of filling will be inaccurate and it can induce dysrhythmias.
- *Noninvasive cardiac output monitoring* – Not validated, and values for stroke volume and cardiac output will be inaccurate, although trends pre-/post-fluid boluses have been shown to be accurate with oeseophageal Doppler and pulse contour wave analysis.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a low-normal heart rate of 50–70 bpm. Avoid tachycardia as this predisposes to arrhythmias and reduces diastolic LV filling and cardiac output.
- *Blood pressure* – Aim for normal blood pressure. A high-normal SVR should be achieved using direct alpha-agonists such as metaraminol and phenylephrine. Meticulous attention to volume status should be observed to ensure adequate filling. Consider preloading with intravenous fluid boluses when fluid shifts are expected.

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- *Dysrhythmias* – Treat promptly. A defibrillator should always be available in theatre, if not connected to the patient for rapid DC cardioversion if required.
- *Other* – Avoid hypoxia, hypothermia, hypercarbia and acidosis which may increase PVR.

ANAESTHETIC TECHNIQUE

Anaesthesia is similar to that for AS. Rapid changes in SVR and ventricular filling are poorly tolerated due to a relatively fixed cardiac output. Falls in SVR result in reduced cardiac output, profound hypotension and reduced coronary blood flow with resultant myocardial ischaemia. Pain will cause increased catecholamine levels predisposing to tachycardia and arrhythmias, so good analgesia is essential.

General anaesthesia +/– regional limb blocks is considered the best approach, although drugs should be titrated carefully and inappropriate use of premedication avoided. A central venous catheter may be considered for safe administration of vasoactive substances in symptomatic patients, or patients undergoing high-risk surgery. Central neuraxial blockade potentially causes significant reduction in SVR. This should generally be avoided in patients with severe MS, and caution exercised if undertaking this approach.

POSTOPERATIVE MANAGEMENT

A low threshold should be had for transferring the patient to ICU postoperatively even following a minor procedure. These patients will often require IABP monitoring to continue postoperatively with the same considerations with regards to haemodynamic parameters as intraoperatively. Adequate postoperative analgesia should be provided and a plan for escalation if this is not achieved.

MITRAL REGURGITATION

Prevalence of moderate-severe MR in the American Strong Heart Study was just over 2% based on echocardiographic features. It is the most common type of valvular heart disease, closely followed by aortic stenosis.

AETIOLOGY

MR is degenerative, caused by damage to the mitral valve apparatus (annulus, leaflets, chordae tendinae and papillary muscles), or functional secondary to LV dilatation, usually as a consequence of chronic hypertension. Mitral valve prolapse, in which the valve leaflets undergo fibromyxomatous degeneration due to defects in the *MMVP1* and *2* genes, occurs in 1%–3% of the normal population, and can progress to MR. Other causes include rheumatic heart disease, inflammatory conditions and connective tissue disorders such as Marfan's and Ehlers–Danlos syndromes. Acute MR is seen following infective endocarditis, acute MI and trauma.

PATHOPHYSIOLOGY

The abnormal coaptation of the valvular leaflets allows regurgitate flow during ventricular systole. This results in volume overload of the LA in systole, and of the LV in diastole. When this overload occurs acutely, the LA is not able to distend acutely, leading to an increase in LA pressure, PAP, and consequently, pulmonary oedema. In the chronic setting, gradual dilatation of the LA occurs to accommodate the increased systolic volume and concentric LVH occurs to produce a supra-normal EF to maintain cardiac output. Only part of this EF passes into the systemic circulation, therefore EF will overestimate the cardiac function. With time, the LV will display evidence of diastolic dysfunction due to the volume load, and LV cavity dimensions will increase (Figure 2.2). The raised LA pressures overtime will result in post-capillary PH and consequently RVH will occur, with RV dysfunction as the disease progresses.

CLINICAL MANAGEMENT

Asymptomatic patients with MR undergoing non-cardiac surgery will normally tolerate anaesthesia well, with no significant mortality or morbidity. However, in the presence of symptoms or echocardiographic evidence of LV dysfunction, there is increased mortality and cardiac morbidity associated with the perioperative period. These patients should have elective surgery postponed, and be referred to cardiology for evaluation and consideration of MV repair or replacement surgery.

PREOPERATIVE MANAGEMENT

Pay particular attention to symptoms of MR, or cardiac failure and history of associated conditions. Review medications and comorbidities. Recent cardiovascular investigations, in particular echo and ET, should be reviewed and noted. A past medical history or family history of connective tissue disorder, e.g. Marfan's (spontaneous pneumothoraces, skeletal abnormalities, visual defects from lens displacement) may be present. If a new diagnosis of MR is made, or the presence of new symptoms or poor symptom control is elicited, a cardiology opinion should be sought preoperatively where possible.

Symptoms of MR include those of heart failure, e.g. fatigue, exertional dyspnoea, peripheral oedema, paroxysmal nocturnal dyspnoea and orthopnoea. If present, AF may be symptomatic and if secondary to IHD, the patient may report anginal episodes. In cases of mitral valve prolapse, atypical chest pain, palpations and syncope may be reported.

EXAMINATION

- Pulse may be irregularly irregular in AF.
- High-pitched pansystolic murmur loudest at the apex, radiating to the axilla, louder on expiration. S1 is classically soft, but may be preserved in mixed rheumatic disease or MVP. Widely split S2 in severe MR, +/– audible S3 and S4.
- Displaced, volume-overloaded apex. Left parasternal heave from pressure overloaded RV.
- General features – mid-line sternotomy suggesting paravalvular MR, vein graft harvesting scars suggesting ischaemic heart disease, features of Marfan's or other connective tissue disorders, peripheral stigmata of infective endocarditis, bruising from anticoagulation for atrial fibrillation and features of cardiac failure.

INVESTIGATIONS

- *ECG* – p-mitrale (bifid broad p waves >40 ms), atrial fibrillation, features of LVH, RVH and strain.
- *CXR* – Cardiomegaly +/– pulmonary congestion.
- *Echo* – Dilated LA, quantification of the regurgitant jet on colour Doppler, LV function, EF, presence of pulmonary hypertension and evaluation of RV function.
- *Cardiac catheterization* – Evaluation of lesion-severity, ventricular function, presence and quantification of pulmonary hypertension, measurement of valve gradient, and assessment of concurrent CAD.

INTRAOPERATIVE MANAGEMENT

MONITORING

- *ECG* – Monitoring of at least two leads (II and V5) for early detection of ischaemia, although sensitivity may be reduced due to underlying LVH.
- *Invasive arterial blood pressure monitoring (preinduction)* – To allow rapid recognition of haemodynamic changes, where CCF is present.
- *TOE* – May be considered to monitor filling, although an experienced operator is required. Strongly recommended where possible for patients with mixed valvular disease where early detection of RWMAs is of greater importance.
- *Non-invasive cardiac output monitoring* – Not validated and values for stroke volume and cardiac output will be inaccurate.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a high-normal heart rate. Bradycardia should be avoided as this increases diastolic time and hence regurgitant volume.
- *Blood pressure* – Aim for a low-normal blood pressure with low-normal SVR ensuring adequate filling with IV fluid. Patients will be very sensitive to reduced preload.
- *Dysrhythmias* – Treat promptly particularly if associated with hypotension. A defibrillator should always be available in theatre for rapid DC cardioversion of tachyarrhythmias if required. Persistent bradycardia may

require treatment with anti-cholinergics or beta-agonists.

- *Other* – Avoid hypoxia, hypothermia, hypercarbia and acidosis which may increase PVR (see PH section)

ANAESTHETIC TECHNIQUE

Anaesthetic technique for patients with MR is similar to that for those with AR. Central neuraxial techniques are well tolerated. The aim of the anaesthetic should be to maintain a low-normal afterload. Good analgesia should be achieved to prevent catecholamine release and associated hypertension. A CVC may be considered for safe administration of vasoactive substances in symptomatic patients or patients undergoing high-risk surgery. PH commonly complicates severe disease, so precautions for this should be observed. Antiarrhythmic agents should be continued during the perioperative period.

POSTOPERATIVELY

Following high-risk procedures, or where large volume shifts have occurred, monitoring in a critical care area is indicated with invasive blood pressure monitoring, to detect early signs of CCF.

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CROSS-REFERENCES

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Heart failure, Chapter 2

MYOCARDIAL REPERFUSION INJURY

Modern treatment of myocardial infarction following coronary occlusion is early reperfusion therapy, using either primary percutaneous coronary intervention (PPCI) or thrombolytic therapy. This has been demonstrated to reduce the infarct size, although not to eliminate myocardial ischaemia completely (Figure 2.3). This is because paradoxically, the reperfusion essential to myocardial salvage can induce myocardial damage itself – this phenomenon is termed 'myocardial reperfusion injury'. It can be categorized clinically into myocardial stunning and reperfusion arrhythmias, microvascular obstruction (MVO) and lethal myocardial reperfusion injury.

In cardiac surgery, the ischaemic reperfusion-injury results from artificially induced stress, and whilst many protective measures are taken, clinically the syndrome remains similar. The heart will be exposed to the extremes of ischaemia and reperfusion on release of the cross clamp, when the myocardium is exposed to fully anticoagulated immunologically primed blood with a high partial pressure of O_2. Reperfusion injury is evident on autopsy examination of up to 45% of patients who die soon after CABG surgery.

PATHOPHYSIOLOGY

Interruption of blood flow to a cardiac myocyte causes physiological changes to normal energy production and utilization pathways. These changes cause reduced energy production and depletion of intracellular ATP, transition to anaerobic energy utilization and accumulation of anaerobic waste productions, reducing intracellular pH. Initially this damage is reversible for a period prior to cell death. The myocyte remains viable if restoration of normal blood flow and physiology occurs, but the cell
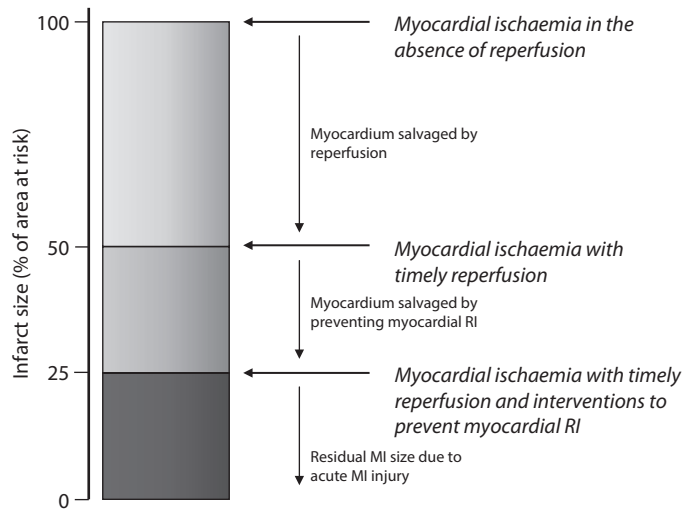
Figure 2.3 Effect of myocardial reperfusion injury on infarct area.

remains vulnerable to the changes that occur with reperfusion.

Following reperfusion of a myocyte, a rapid pH correction occurs with washout of lactic acid resulting in high levels of intracellular Na^+ and Ca^{2+} and oxidative stress. These factors lead to activation of the mitochondrial permeability transition pore (MPTP) and myocyte hypercontacture, and result in myocyte death. Opening of the MPTP results in mitochondrial oxidative uncoupling, resulting in loss of ability to produce ATP, and further ATP depletion, further ionic imbalance and ultimately necrotic cell death. Although the molecular entity of MPTP remains unknown, it appears to play a crucial role in animal models of reperfusion injury, inhibition with cyclosporin A or genetic inhibition of cyclophillin-D and results in a 40%–50% reduction in size of myocardial infarct.

The majority of evidence suggests myocardial injury occurs in the minutes immediately following reperfusion. Inhibition of myocyte contraction at time of reperfusion reduces infarct size in animal models, as does maintaining an acidic environment for the reperfused myocytes, and inhibition of calcium uptake. A small number of therapeutic interventions, however, have been reported to reduce MI size when administered up to 24 hours after reperfusion, suggesting the possibility of an early and a late phase.

CLINICAL CATEGORIZATION

MYOCARDIAL STUNNING

This refers to the transient postischaemic impairment of myocardial contractility that occurs on reperfusion, rather than to hibernating myocardium due to poor resting blood flow. Myocardial stunning is thought to be due to the high levels of intracellular calcium and the effects of oxidative stress.

REPERFUSION ARRHYTHMIAS

Reperfusion-induced arrhythmias are commonly seen in patients who have undergone PPCI, thrombolysis or cardiac surgery. Most commonly, accelerated idioventricular rhythms and ventricular premature beats are seen in the minutes after reperfusion. Rarely, sustained VT of VF is seen although this is thought to occur more commonly in the presence of MVO.

MICROVASCULAR OBSTRUCTION AND INTRAMYOCARDIAL HAEMORRHAGE

Microvascular obstruction is the inability to reperfuse distal small arterial circulation despite restoration of flow to the epicardial circulation. It is thought to be due to a combination of factors:

- Micro-embolization of thrombus or plaque to the distal circulation.
- Vasoconstriction, due to release of vasoactive substances in response to injury, effects of which may be attenuated by bronchodilators.
- New thrombus formation due to activation of platelets and increased circulating prothrombotic and proinflammatory substances.
- Structural insufficiencies of the capillary bed, direct compression of capillaries by oedema.

This often manifests at the time of PPCI with a postintervention TIMI risk score <2, known as 'no reflow syndrome'. However, it should be noted that in some patients with good flow (TIMI >3) following PPCI, up to 40% still have evidence of MVO. The presence of MVO in patients with reperfused MI is associated with larger infarct area, worse post-event ejection fraction, adverse LV remodeling and worsening of short- and long-term prognosis.

LETHAL MYOCARDIAL REPERFUSION INJURY

The most serious consequence of reperfusion is myocyte death. A large number of myocytes have already been lost following the initial insult and further loss worsens both short- and long-term outcomes. It has been difficult to demonstrate reperfusion-induced death of otherwise viable myocytes, and as a result, indirect evidence has been relied upon. Experimental studies have demonstrated up to a 50% reduction in the size of infarct following therapeutic intervention, making it a key target for cardioprotection.

THERAPEUTIC INTERVENTIONS

Many interventions demonstrating benefit in animal models or acute MI have failed to translate to benefits in clinical trials. This may be because in animal models, there is no preexisting coronary artery disease and no comorbid diseases such as diabetes. Also, many patients are pretreated with agents such as nitrates and statins, which could interfere with the cardioprotective effect.

With years of experience of cardiac surgery, many protective measures to reduce the impact of ischaemia intraoperatively have been developed. A reduction in myocardial metabolic activity and hence oxygen demand during the ischaemic period is achieved by inducing cardioplegia. This is maintained via hyperkalaemic, hypothermic arrest, with intermittent glucose and mannitol containing blood-mix cardioplegic solution. Despite this, the myocardium still remains susceptible to reperfusion injury.

ISCHAEMIC POSTCONDITIONING

Reocclusion of the vessel for a period of 30 or 60 seconds up to four times following reperfusion has demonstrated mixed results with Thibault et al. demonstrating reduced MI size and improved ejection fraction at 6 months and 1 year, respectively, whilst other studies have failed to demonstrate improved myocardial salvage. This technique has been used in cardiac surgery with positive results, where two 30 s cycles of aortic clamping were undertaken post-reperfusion following Tetralogy of Fallot repair in children, with reduced levels of tropnin I postprocedure. This was also demonstrated in adults undergoing valve surgery; however, the clamping and unclamping of the aorta increased the risk of embolic events.

REMOTE ISCHAEMIC PRECONDITIONING

The practice of remote ischaemic conditioning (RIC) allows the therapeutic intervention to be undertaken remotely prior to reperfusion. This has been demonstrated to have benefit with lower troponin I and improved myocardial salvage post-reperfusion when performed prior to PPCI for acute MI.

In cardiac surgery the evidence from animal models has been promising, although clinical trial data is conflicting. This may be because of variations

in anaesthetic technique, the heterogeneity of the populations studied, with multiple comorbidities and medications, and the fact that the studies were largely small, single-centre studies. The classical protocols involve inflation of a BP cuff on the upper limb for a period of 5 minutes and then a reperfusion period of 5 minutes, which is repeated for 3–5 cycles.

As with other areas, early proof of concept trials has been promising, demonstrating reduced troponins postoperatively and improved myocardial salvage; however, more clinically focused trials have yielded less promising results, although marginal gains can be seen across these, with the potential for development of preconditioning bundles.

VOLATILE PRECONDITIONING

Studies dating back to the 1980s have demonstrated cardioprotective effects of volatile anaesthetic agents. Extensive evidence exists in animal trials and a number of proof of concept trials have demonstrated protection against reperfusion injury with significant reductions in postoperative troponins. Unfortunately, data from recent meta-analyses in cardiac anaesthesia have proven inconclusive, with pragmatic large-scale multi-centre trials still required. Volatile agents have been hypothesized to have actions on myocytes via the reperfusion injury salvage kinase (RISK), and the survivor-activating factor enhancement (SAFE) pathways, as well as direct actions on the endothelium, mainly via the inhibition of TNF-alpha-induced adhesion molecules.

OTHER AGENTS

There is a large body of literature focused on prevention of reperfusion injury in the context of cardiac surgery and acute myocardial infarction. Whilst there is often benefit demonstrated in animal models or *in vitro* studies, this has yet to be translated into robust clinical trials for these agents.

Of note in anaesthesia, propofol has been suggested to scavenge peroxynitrite and up-regulate nitric oxide synthase activity. Morphine has been shown to enhance the preconditioning effects of isoflurane, and infusions of remifentanil have

been shown to protect the right atrium against reperfusion injury.

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PATIENTS WITH PERMANENT PACEMAKERS AND IMPLANTABLE DEFIBRILLATORS

The number of patients with cardiovascular implantable electronic devices (CIED) is continually increasing. These can be categorized into pacemakers (PM), cardiac resynchronization therapy (CRT), implantable defibrillators (ICD) or a combination of these. Rapid changes in the complexity and range of devices as well as responses to magnets and increasing potential sources of electromagnetic

interference (EMI) make the perioperative period a challenging time to ensure a high degree of patient safety.

PACEMAKER DEVICES

Pacemaker devices have advanced significantly in recent years. Classical implantable box lead transvenous pacemakers are still the standard seen in clinical practice. Leadless intracardiac devices have since been developed, but are not yet in routine use. Modern pacemaker boxes contain a lithium ion-battery, a pulse generator and a device controller contained in a hermetically sealed titanium shell. This shell has the dual effect of preventing diffusion of water vapour into the device, and shielding from EMI. Pacemaker leads are insulated to prevent escape of current, and conduction of stray electrical impulses to the myocardium. They are attached to the outside of the pacemaker box and are passed normally via the subclavian vein to attach to the epicardium via a screw or clasp mechanism at the tip of the lead. Leads may be exchanged independently of each other and the PM box.

Most modern pacemakers have multiple functions. They are able not just to pace at set rates to set leads, but also to detect transmitted impulses and respond to these by triggering or inhibiting an impulse. Many devices now have accelerometers allowing rate responsiveness to exercise. Pacing can take place either in a single chamber (atrium or ventricle), two chambers (dual, atria and ventricles), or multiple chambers (CRT/biatrial). Typically leads are placed in the right chambers of the heart, but in multi-chamber pacing such as CRT devices, a left-sided lead will be passed via the coronary sinus.

The British Pacing and Electrophysiology Group and the North American Society of Pacing and Electrophysiology have defined a generic coding for all CEIDs displayed in Table 2.14. The underlying pathophysiology in the patient will determine the number and placement of leads and the programming of the device *in situ*. Symptomatic sinus bradycardias may benefit from single lead AAI pacing, whereas patients with AF and AV block, or poor tolerance of rate control, with risk of symptomatic bradycardia, may benefit from DDDR devices. The addition of the –D symbol to the end of the device code denotes defibrillator function is present.

IMPLANTABLE CARDIAC DEFIBRILLATORS

Implantable cardiac defibrillators can be an isolated device or combined with pacemaker functionality. These devises are normally placed in patients with risk of VF or VT and hence sudden cardiac death. They can be recognized on CXR by a thickened coil at the end of the defibrillator lead. These devices have multiple therapeutic options. They can deliver a low energy synchronized shock, a high energy unsynchronized shock, overdrive pacing or back-up bradycardia pacing (Table 2.15).

PREOPERATIVE MANAGEMENT

Preoperative management of the patient with a CIED must be conducted in a timely and thorough manner. This should involve the anesthetic, surgical and cardiology teams. An understanding of the risk from EMI and how this can be minimised intraoperatively is essential. The CIED team should be

Table 2.14 Revised NASPE/BPEG generic code for pacemakers

I	II	III	IV	V
Chamber(s) paced	Chamber(s) sensed	Response to sensing	Rate modulation	Multisite pacing
O = None	O = None	O = None	O = None	O = None
A = Atrium	A = Atrium	T = Triggered	R = Rate modulation	A = Atrium
V = Ventricle	V = Ventricle	I = Inhibited		V = Ventricle
D = Dual (A+V)	D = Dual (A+V)	D = Dual (I+T)		D = Dual (A+V)

Table 2.15 Revised NAPSE/BPEG generic code for defibrillators

I	II	III	IV
Shock Chamber	**Anti-tachycardia pacing chamber**	**Tachycardia detection**	**Anti-bradycardia pacing chamber**
O = None	O = None	E = Electrogram	O = None
A = Atrium	A = Atrium		A = Atrium
V = Ventricle	V = Ventricle	H = Haemodynamic	V = Ventricle
D = Dual (A+V)	D = Dual (A+V)		D = Dual (A+V)

advised of any planned procedures, and the nature of these. Recommendations for management of the device will be based on information given. Essential information that should be considered and conveyed to the CIED team includes:

- Type, make and model of device if known and current programming.
- Date of last check and if there have been any problems with the device.
- Indication for the pacemaker – is the patient pacemaker dependent?
- Type of procedure, anatomical location of procedure, and patient positioning during procedure.
- Usage of diathermy (monopolar, bipolar or none), and if monopolar, placement of diathermy pad and path of EMI.
- Presence of other sources of EMI.
- Use or external defibrillator.
- Postprocedural arrangements.

The EHRA recommends that PMs should be checked every 3–12 months and ICDs every 3–6 months. All patients with PMs undergoing elective surgery should have had a device check within 12 months of the date of surgery and for ICDs or CRTs, within 6 months of the date of surgery. If this has not occurred, the operative team should arrange a check preprocedure. This should include information on the programming of the device including the response of the device to placement of a magnet and battery longevity. The CIED team may advise that the pacemaker should be reprogrammed to an asynchronous mode for the perioperative period, or that certain features should be deactivated, such as the tachyarrhythmia detection in an ICD. This

recommendation should also include recommendations for postoperative reprogramming, follow-up and assessment.

Preoperative work-up should include a 12-lead ECG to evaluate pacing function and baseline HR. Electrolyte levels should be determined and corrected as required. If no information is available about the device, or there are concerns about lead placement, a PA and lateral CXR may be helpful.

INTRAOPERATIVE MANAGEMENT

Monitoring should be carried out inline with AAGBI guidance suitable for the procedure being undertaken. Particular attention should be made to ensure the monitor shows the pacing spikes on the ECG trace as this may be suppressed by the default software settings. Due to the potential for interference with the ECG signal from a pacemaker device or misinterpretation by the monitoring software, it is vital a secondary source for heart rate is displayed and correlated with ECG rate. This would normally be the pulse oximetry contour analysis, although if an arterial line has been placed this would be a suitable alternative source. With the increased potential for arrhythmias, a defibrillator and alternative means of pacing must always be available in theatre.

Diathermy is the leading cause of EMI intraoperatively. Where possible, no or bipolar diathermy should be used to reduce the risk of EMI causing malfunction of the pacemaker. If monopolar diathermy has to be used, the time it is used for should be minimised and the plate should be placed as far away from the pacemaker as possible, e.g. for head and neck surgery on the opposite shoulder to the CIED.

Magnets classically had the effect of resetting devices to a default mode of asynchronous pacing, and in the case of an ICD, of inactivating tachyarrhythmia detection. However, in modern devices the programmed function of a magnet is diverse and can stimulate almost any function of the pacemaker, or may deactivate the device completely. Some devices may not respond at all to magnet placement. As a result, it is no longer recommended to place a magnet blindly on a device, except in emergency situations where discussion with a CIED team is not possible. In this situation, a magnet may be placed on an ICD device, provided alternative means of pacing and defibrillation are available, possibly even connected to the patient.

POSTOPERATIVE MANAGEMENT

If the device has been reprogrammed for the perioperative period, this should be reversed immediately postoperatively in the recovery area, and if there is any concern about the device's function, it should be checked by the CIED team prior to the patient leaving the recovery area. For certain procedures, even if no changes have been made to the device and/or no intraoperative concerns raised, the device should still be interrogated at an earlier than routine interval.

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CROSS-REFERENCE

PULMONARY HYPERTENSION

Pulmonary hypertension (PH) is a pathophysiological disorder, complicating many clinical conditions, and may arise as a sequelae of the majority of cardiovascular, respiratory and connective tissue diseases. PH is defined as a mean resting pulmonary artery pressure (PAP) >25 mmHg, assessed by right heart catheter studies. Normal resting PAP is <20 mmHg. A resting PAP between 21 and 24 mmHg is of unclear clinical significance, and should be carefully monitored due to the possibility of developing PH. Haemodynamic definitions are illustrated in Table 2.16. Pulmonary

Table 2.16 Haemodynamic definitions of pulmonary hypertension

Definition	Characteristics	Clinical group
PH	PAP > 25 mmHg	All
Precapillary PH	PAP > 25 mmHg and PAWP < 15 mmHg	1, 3, 4 and 5
Postcapillary PH	PAP > 25 mmHg and PAWP > 15 mmHg	2 and 5

arterial hypertension (PAH) or primary pulmonary hypertension is defined as precapillary PH and PVR >3 Wood Units in the absence of lung disease, thromboembolic disease or other multifactorial pathologies.

EPIDEMIOLOGY

Pulmonary hypertension has a UK prevalence of 97 cases per million, with a male to female ratio of 1:1.8. There is poor epidemiological data for the individual classes of PH, although it is clear that the most common cause of PH is left heart disease. Up to 60% of patients with severe LV systolic dysfunction have evidence of PH, and it is present in up to 70% of those with significant LV diastolic dysfunction. It is also evident in >80% of patients with symptomatic left sided valvular disease. PAH is the rarest of the groups accounting for 1.5 cases per million.

AETIOLOGY AND CLASSIFICATION

Numerous causes of PH have been identified. These have been grouped into comprehensive clinical classifications by Simonneau et al. and are widely adopted by the European Society of Cardiology and American College of Cardiology. This classification system, demonstrated in Table 2.17, groups the multiple aetiologies based on their clinical presentation into five groups. Current classifications are continually evolving as new mutations and clearer understanding of the pathophysiology develops.

PATHOPHYSIOLOGY

Increases in pulmonary vascular resistance are essential for the development of pulmonary hypertension.

In PAH there are various pathways implicated in the development of hypertrophy and fibrosis of pulmonary vessel intima, which is thought to be the key pathological change.

- Reduced expression and activity of eNOS resulting in impaired relaxation of the pulmonary artery smooth muscle.
- Increased production and reduced clearance of ET-1 a potent stimulator of vascular cell proliferation and moderate vasoconstrictor.

- Inhibition of prostacyclin synthase, which leads to reduced local prostacyclin levels, resulting in vasoconstriction and a prothrombotic tendency due to enhancement of platelet aggregation. The antiinflammatory and antiproliferative effects are also impaired due to enhanced leucocyte activation.

For non-PAH the mechanisms vary between the varying underlying disease pathologies, although grossly speaking, excessive pulmonary vascular cell proliferation, decreased cell apoptosis, thrombosis and reduced vasodilation play a significant role in disease development.

CLINICAL MANAGEMENT

Patients with PH have increased all-cause mortality and morbidity in the perioperative period, with up to a seven-fold increase in mortality following noncardiac surgery when compared to the normal population. The majority of patients presenting for noncardiac surgery with PH will have group two, three or four disease, and the underlying cause should be considered when planning an anaesthetic.

SPECIFIC TREATMENTS

Patients should be evaluated in specialist pulmonary hypertension clinics to allow optimisation of therapies and timely interventions. Treatment of underlying disease may slow the progression of PH although once established it is unlikely to be reversed by this alone.

Prostacyclin analogues – Can be used by intravenous infusion or nebulization. These reduce pulmonary vascular resistance, with potential additional antiinflammatory, antiproliferative and anticoagulant effects secondary to inhibition of platelet aggregation (epoprostenol, iloprost, beraprost).

Endothelin-1 receptor antagonists – Administration results in reduction of PAP by prevention of vasoconstriction. These agents may also slow disease progression through antiproliferative effects (bosentan, macitentan).

Phosphodiseterase type 5 inhibitors – Inhibit breakdown of cGMP, which can lead to reduction in PVR (sildenafil, tadalafil, vardenafil).

Table 2.17 Clinical classification of pulmonary hypertension

Group 1. Pulmonary arterial hypertension	Group 3. PH due to lung disease and/or hypoxia
1.1 Idiopathic	3.1 COPD
1.2 Heritable	3.2 ILD
1.2.1 BMPR2 mutation	3.3 Sleep-disordered breathing
1.2.2 Other mutations	3.4 Alveolar hypoventilation disorders
1.3 Drug or toxin induced	3.5 Chronic exposure to high-altitude
1.4 Associated with other conditions	
Group 1A. Pulmonary veno-occulsive disease and/or	**Group 4. Chronic thromboembolic PH and other**
** pulmonary capillary haemangiomatosis**	** pulmonary artery obstruction**
1A.1 Idiopathic	4.1 Chronic thromboembolic PH
1A.2 Heritable	4.2 Other pulmonary artery obstructions
1A.3 Drug, toxin or radiation induced	4.2.1 Angiosarcoma
1A.4 Associated with HIV or connective tissue disease	4.2.2 Other intravascular tumors
Group 1B. Persistent PH of the newborn.	4.2.3 Arteritis
	4.2.4 Parasites (hydatidosis)
Group 2. PH due to left-sided heart disease	**Group 5. PH with unclear and/or multifactorial**
2.1 LV systolic dysfunction	5.1 Haematological disorders: chronic haemolytic
2.2 LV diastolic dysfunction	anaemia, myleoproliferative disorders, splenectomy
2.3 Valvular disease	5.2 Systemic disorders: sarcoidosis pulmonary
2.4 Congenital/acquired left heart inflow/outflow tract	histiocytosis, neurofibromatosis
obstruction and congenital cardiomyopathies.	5.3 Metabolic disorders: glycogen storage disease,
2.5 Congenital/acquired pulse vein stenosis	Gaucher disease, thyroid disease

Lung transplant – In patients who fail mono- or combination therapy, consideration for lung transplant should be undertaken.

Balloon atrial septostomy – The creation of and right-to-left intra-atrial shunt can decompress the right heart chambers and increase LV preload and hence cardiac output. This procedure may be considered in patients awaiting lung transplant, but should be avoided in end stage patients or patients with mean RAP >20 mmHg.

PREOPERATIVE MANAGEMENT

Symptoms of PH are nonspecific and are usually related to progressive decline in RV function. Preoperative assessment should focus on eliciting the underlying cause of PH, evaluation of recent investigations (particularly echo, cardiac catheter studies, exercise tolerance tests, sleep studies, spirometry) and the current functional status of the patient, as well as any interventions attempted and medications used for treatment of PH and other comorbid diseases. Particular attention should be paid to the use of home oxygen therapy and anticoagulation, as this will require individualized preoperative management. If a diagnosis of HIV is present, recent CD4 titers should be reviewed and advice from a home ID team sought.

Symptoms of PH include:

- Features of RV dysfunction, shortness of breath, fatigue, weakness, angina, syncope, leg swelling, abdominal distension, fluid retention and weight gain.
- Mechanical complications of PH can result in haemoptysis, hoarse voice from compression of the laryngeal nerve, exertional angina from compression of left main coronary artery by the pulmonary artery.
- Symptoms relating to underlying disease, features of connective tissues disease, immunosuppression, or use of medications related to PH, e.g. appetite suppressants aminorex, fenfluramine or amphetamines.

EXAMINATION

- Cyanosis and clubbing may be seen.
- Continuous or burst oxygen therapy may be present.
- A right ventricular heave may be felt at the left parasternal edge.
- A loud or palpable P2 may be present, with fixed paradoxic splitting of S2 in severe RV dysfunction.
- Quiet pansystolic murmur of TR.
- Prominent JVP A waves may be seen with RVH, and prominent V waves with TR. Features of underlying disease may be present.
- Systemic sclerosis – Teleangectasia, digital ulceration and sclerodactyly.
- Interstitial lung disease – Fine inspiratory crackles and clubbing.
- Valvular heart disease – Typical murmurs of involved valves.
- Congenital heart disease – Sternotomy or thoracotomy scar, clubbing in the presence of cyanotic CHD, murmurs from residual surgical shunts, ASDs, VSDs or valvular heart disease.

INVESTIGATIONS

- *ECG* – May be normal or show features of right-sided disease, p-pulmonale (p-wave >2.5 mm tall) in the presence of right atrial enlargement, RBBB, RVH and strain, and QT prolongation. The presence of QT prolongation and a broad QRS is suggestive of severe disease. SVTs may occur, most commonly atrial flutter or atrial flutter-fibrillation. These are present in approximately 25% of patients 5 years after diagnosis.
- *Echo* – Evaluation of right ventricular function and valvular heart disease. Estimation of PAP is also possible, although if intervention is planned, cardiac catheterisation should be performed.
- *Pulmonary function tests* – In the presence of underlying lung pathology, PFTs allow quantitative evaluation.
- *Arterial blood gas* – Can allow evaluation of underlying lung disease and provide baseline values for postoperative targets. This may be of use in the context of home-oxygen therapy, severe COPD with CO_2 retention and interstitial lung disease.
- *Cardiac catheter studies* – Right heart catheter studies and vasoreactivity studies. These allow accurate pressure measurements for quantification of disease. In the context of coexisting CAD, a low threshold should be had for performing left-sided studies at the same time.
- *Sleep studies* – Nocturnal deoxygenation can occur even in the absence of obstructive sleep apnoea.
- *Exercise testing* – 6-minute walk tests are traditionally used to assess CO and peak oxygen consumption. Improved survival is seen in patients who can achieve 332 m. CPET may be of use although in advanced disease this may be difficult.

INTRAOPERATIVE MANAGEMENT

MONITORING

- *ECG* – Monitoring of at least two leads (II and V5) for early detection of ischaemia.
- *Invasive arterial blood pressure monitoring (from preinduction)* – To allow rapid recognition of haemodynamic changes.
- *TOE* – This allows accurate evaluation of ventricular filling and provides the most useful information where rapid volume shifts are expected, and can also identify RWMAs. However, this requires an experienced operator.
- *Pulmonary artery catheter* – Can provide accurate measurements of PAP, PVR and estimates of SVR. However, introduction may induce severe arrhythmias, and there is increased risk of RV thrombosis and embolic events.
- *Non-invasive cardiac output monitoring* – Can be useful to identify volume shifts, and estimates of filling, whilst avoiding the potential complications of PAC. However, does not allow right heart pressure measurements or measurement of PVR.
- *Central venous catheter* – Placement can be useful for administration of vasoactive drugs.

- *Continuous temperature monitoring* – Use of an oesophageal temperature probe allows rapid identification and correction of hypothermia to prevent rises in PVR.

PHYSIOLOGICAL TARGETS

- *Heart rate* – Aim for a low normal heart rate. Tachycardia can predispose to arrhythmias.
- *Blood pressure* – Aim for normal blood pressure and a high-normal SVR should be achieved using vasopressors. Meticulous attention to volume status to ensure adequate filling. Consider preloading with intravenous fluid boluses when fluid shifts are expected. Inotropic agents such as enoximone or milrinone should be used where required to support RV function. Low dose noradrenaline may be required to balance the fall in SVR.
- *Pulmonary vascular resistance (PVR)* – Prevent rises in PVR intraoperatively. Aggressive treatment of hypercarbia, hypothermia, hypoxia, pain and acidosis should occur. High PEEP, gas trapping, hyperinflation and high ventilation pressures should be avoided. Inhaled prostacyclin or NO can be considered.

ANAESTHETIC TECHNIQUE

Mild PH will have little consequences for anaesthetic technique; however, when severe PH is present, patients are at high risk of RV decompensation, pulmonary hypertensive crisis and cardiogenic shock.

A balanced approach to anaesthesia should be untaken with particular attention paid to the avoidance of hypoxia, hypercarbia, rapid changes in SVR and pain. Both general and regional techniques are considered safe. In general, avoidance of large volumes and rapid reductions in SVR should be achieved. To this end, consideration of an epidural enabling slow titration of agents may be preferential to spinal anaesthesia.

Selective pulmonary dilators can be used in the context of acute pulmonary hypertensive crisis, in-circuit prostacyclin nebulization or inhaled NO, although caution should be exercised as these may reduce SVR.

POSTOPERATIVE MANAGEMENT

Provision of monitoring and timely intervention is key to successful postoperative recovery. Meticulous fluid balance, maintenance of baseline parameters with supplemental oxygen and inotropic therapy as required. Patients may require prolonged ventilation and have potential difficulties with respiratory weaning. In these cases, advice from specialist centres should be sought. Elective admission to ICU should be considered for all high- and intermediate-risk patients, or those with slow progression to baseline function postoperatively.

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CROSS-REFERENCES

Heart failure, Chapter 2
Congenital heart disease in adult life, Chapter 2

TETRALOGY OF FALLOT

Tetralogy of Fallot (ToF) is one of the most common clusters of congenital cardiac abnormalities. It was first described in 1888 by Louis Arthur Fallot, classically comprising of a set of four malformations.

1. Large non-restrictive VSD
2. Valvular, subvalvular or supravavlular pulmonary stenosis, causing right ventricular outflow tract obstruction (RVOTO)

3. Right ventricular hypertrophy
4. An overriding aorta

Surgical correction in the first 6 months of life is now favoured over palliative shunt procedures with delayed correction, except in very small sick patients requiring urgent palliation.

EPIDEMIOLOGY

Approximately 10% of all CHD is accounted for by ToF, it is the most common cause of cyanotic CHD, with a prevalence reported between 3 and 5/10,000 live births and has remained relatively static since the 1980s. The prevalence is significantly higher in Asia than in Europe and North America, and in males. Mortality for untreated disease is approximately 60% by age 5.

AETIOLOGY

Most cases of ToF are sporadic with no clear cause identified. One identified association is DiGeorge syndrome where individuals have a 2q11.2 deletion, and conotruncal cardiac abnormalities are seen. Approximately 35% of cases will have ToF.

PATHOPHYSIOLOGY

The development of the phenotypic features of ToF results from under-development of the spiral septum, leading to anterior rightward misalignment of the muscle bundle, precluding correct fusion with the ventricular septum. This narrows the pathway from the RV to the PA, and enlarges the aortic root such that it overreaches the RV outflow tract.

Right-to-left intracardiac shunting, and hence cyanosis, results from the RVOTO and large VSD. Due to the unrestrictive nature of the VSD, the size of the shunt is governed solely by the degree of RVOTO. In severe RVOTO, pulmonary blood flow may be significantly reduced and dependent on a PDA, aortopulmonary collateral vessels or surgical shunt. The large nonrestrictive VSD equalises the pressures across the two ventricles in response to this, and compensatory RVH occurs. One of the key features is that the RVOTO can vary within the normal physiology of the patient and can result in

'Tet' spells where paradoxical cyanosis occurs due to exercise, feeding or crying because of an increased right-to left shunt.

HISTORY

Although most cases are now treated in childhood, the natural progression of ToF depends largely upon the degree of RVOTO, which is highly variable between individuals. If only minor RVOTO occurs, the patient will be relatively asymptomatic and not display cyanosis because the blood flow across both outflow tracts is relatively matched. In severe RVOTO, cyanosis will typically appear in the post-birth perinatal period. In moderate RVOTO, the patient will tend to present with paradoxical cyanosis, and exertional dyspnoea. Patients may have compensatory behaviors to raise their SVR and restore pulmonary blood flow, classically they report squatting during Tet cyanotic episodes, although this is rarely seen now due to antenatal and newborn screening. However, it is important to recognize cyanotic episodes because these can result in myocardial ischemia and CVAs.

EXAMINATION

A loud harsh ESM will be audible from day one of life at the left sternal border. There will often be a single second heart sound A_2 and as the RVOTO worsens the ESM will shorten. During hypercyanotic episodes it may be inaudible.

A right ventricular heave may be present and occasionally a systolic thrill along the left sternal border.

Older children and adults may show signs of chronic cyanosis such as finger clubbing.

Abnormal facies typical of DiGeorge syndrome may also be present.

In older children and adults who have undergone corrective surgery, there is a multitude of possible signs but the common ones are

- Sternotomy +/– thoracotomy scar
- Radio-radial pulse deficit ipsilateral to any surgical shunt
- Pectoral hypoplasia (Poland's syndrome) ipsilateral to shunt

- Elevated JVP
- Signs of heart failure (peripheral pitting oedema, end inspiratory crackles, hepatomegaly)
- Apex may be displaced if AR has developed
- A to-and-fro systolic-diastolic murmur in the pulmonary area if PR has developed

INVESTIGATIONS

CXR: Usually normal initially. In older children and adults, a boot-shaped heart may be seen due to RVH. A right-sided aorta may be seen or more typically a convexity of the left border of the heart where the main pulmonary artery and RVOT normally sit. Pulmonary vascular markings will appear normal or decreased.

ECG – Initially normal although will show signs of RVH/strain in later life. RBBB may occur following operative correction even in the absence of ventriculotomy.

ECHO – This will demonstrate the number and size of VSDs, and gross anatomy and degree of RVOTO, and presence of associated anomalies such as ASDs seen in pentad of Fallot. Cardiac catherisation may be required for detailed anatomy and functional studies.

Diagnostic cardiac catheter studies – These will provide detailed information on the anatomy of the RVOTO level and function, VSD number and size, ventricular filling pressures, presence and anatomy of aortopulmoary collaterals, anatomy of pulmonary arteries, and allows balloon valvuloplasty of the pulmonary valve to be performed.

PALLIATIVE SURGERY – BLALOCK-TAUSSIG SHUNT

For many years, palliative shunts were the treatment of choice. In current practice they are rarely performed, due to advances in repair surgery. A small Gore-Tex tube is used to connect the subclavian artery to one of the pulmonary arteries bypassing the RVOTO. Following a BT shunt, saturations of 80%–90% are targeted on air to allow the child to grow prior to definitive surgery being undertaken. Circumstances where this may be considered are generally in the first weeks of life in small and very sick children with the following:

- Marked RVOTO with cyanosis
- Pulmonary atresia and duct dependent circulation
- Anomalous coronary arteries
- Unsuccessful pulmonary balloon valvuloplasty

ANAESTHESIA FOR BLALOCK-TAUSSIG SHUNT

A full and thorough history and review of investigations should be undertaken prior to surgery, and one should proceed cautiously in children with uncorrected ToF. If a duct-dependent circulation is present, its patency should be maintained pre- and intraoperatively with a prostacyclin infusion. Awareness, early detection of, and management of Tet spells is also crucial. The majority of cases are performed via a thoracotomy although in technically difficult cases or where pulmonary blood flow is critical, bypass may be used.

The child should undergo intubation and ventilation with careful consideration of induction agents to maintain adequate SVR. Gas induction or ketamine are considered safe.

Adequate analgesia should be ensured to avoid catecholamine surges.

Full invasive monitoring is recommended, including central venous and arterial access. Femoral arterial access is considered preferable because the subclavian artery will be clamped prior to the shunt being attached.

Inotropic support with adrenaline or dopamine is contraindicated until the shunt has been opened due to risk of infundibular spasm, although this may be required once the shunt has been opened.

For non-bypass cases, a heparin bolus is required prior to the clamp being placed.

Once the shunt has been established, inspired oxygen should be reduced to 30% prior to opening the shunt. Adequacy of the shunt can then be assessed by an increase in saturations on pulse oximetry and ABG following unclamping.

POSTOPERATIVE MANAGEMENT

Elective admission to ICU postoperatively should be considered. A low-dose heparin infusion should be started, and if no or minimal postop bleeding, converted to aspirin after 3–5 days. Patency

should initially be checked on echo and confirmed regularly with monitoring of oxygen saturations and auscultation of a continuous murmur over the shunt.

CORRECTIVE SURGERY

Surgical repair for ToF involves closure of the VSD and relief of the RVOTO. Surgery is usually carried out in the first year following birth, at age 3–6 months. More recently, earlier repairs have been completed with good outcomes in the first few weeks of life. A trans-atrial approach is favoured for bovine patch repair of the VSD. Approach to reduction of the RVOTO will depend on the level of the obstruction, and where possible the pulmonary annulus should be maintained. In patients with borderline annular size, the residual RVOTO needs to be traded off against the obligate insufficiency seen with a transannular patch. In patients with pulmonary atresia or coronary artery abnormalities, a pulmonary conduit may be required.

Transoesophageal echo is the intraoperative assessment of choice for adequacy of repair, as this allows evaluation of the VSD closure and RVOT.

ANAESTHESIA FOR CORRECTIVE SURGERY

A full and thorough history and review of investigations should be undertaken prior to surgery, and one should proceed cautiously in children with uncorrected ToF. In patients with a preexisting BT shunt, patency should be checked preoperatively with close attention to fluid status, as reduced filling may result in cyanosis. Awareness, early detection of and management of Tet spells is also crucial. If corrective surgery is performed on bypass, consideration should be given to use of cardiopulmonary bypass in infants and on-pump cooling.

Children with a history of Tet spells may benefit from premedication with 0.5 mg/kg midazolam 30 minutes preoperatively.

A balanced induction of anaesthesia by inhalation using sevoflurane, or intravenous using ketamine is considered safe as these avoid large reductions in SVR relative to PVR.

Full invasive monitoring including central venous and arterial access is recommended.

An experienced TOE operator should be available.

Ensure blood products and antifibrinolytics are available, generally two units of cross-matched red cells, platelets and cryoprecipitate.

Adrenaline and dopamine are contraindicated prior to bypass being established. Inotropic support may be required for coming off-pump, in which case milrinone plus noradrenaline or adrenaline is commonly used.

POSTOPERATIVE MANAGEMENT

Following repair of ToF, elective admission to a paediatric critical unit should be undertaken.

Invasive monitoring should continue with careful intravenous filling to minimize the effect of residual RVOTO.

Iontropic support is often required following bypass and a phosphodiesterase inhibitor such as enoximone or milrinone is ideal. The addition of noradrenaline may be required to counteract the reduction in SVR these cases.

Ventilation should continue until the child is warm, with minimal bleeding and adequate peripheral perfusion.

Bleeding should be controlled surgically, although administration of blood products and antifibrinolytics may be required. The use of these should be guided by TEG or ROTEM assays.

Avoidance of electrolyte disturbances with potassium maintained in range 4.5–5.0 mmol/L, and magnesium in the range 1.5–2.0 mmol/L to reduce the risk of cardiac arrhythmias is recommended. Junctional escape tachycardias are commonly seen following cardiac surgery. These should be treated promptly with cooling to 34°, maintenance of serum electrolytes and amiodarone loading.

MANAGEMENT OF 'TET' SPELLS

General measures to increase oxygenation, improve cardiac output and reduce infundibular spasm and right-to-left shunting should be undertaken.

- Give 100% O_2, and check ETT position.
- Deepen anaesthesia and give an opiate bolus.

- Fluid bolus if indicated.
- Phenylepherine bolus to increase SVR, if repeated administration is required consider noradrenaline infusion.
- Consider beta-blocker infusion.

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NONCARDIAC SURGERY IN THE TRANSPLANTED HEART PATIENT

Cardiac transplant is now the treatment of choice for many patients with severe cardiac failure who have failed medical therapy, with figures estimating in excess of 5000 transplants per year worldwide. Survival to 1 year for transplant recipients is 85%–90% with 3-year survival reported to be as high as 75%. As a result, increasing numbers of cardiac transplant recipients will present for noncardiac surgery to nonspecialist centres. Knowledge of the physiology of the denervated heart is crucial to provide safe and effective anaesthesia for these patients.

THE DENERVATED HEART

A transplanted heart has an absence of sensory, sympathetic and parasympathetic innervation. Due to lack of parasympathetic innervation, vagal tone is lost and the resting heart rate will be in the region of 90–110 bpm. Cardiac output of the transplanted heart is relatively dependent upon preload, and in response to hypovolemia the cardiac output is increased by increasing the stroke volume rather than the heart rate and contractility, as in the normal heart, due to insensitivity to neurohormonal stimulation.

A degree of sympathetic and parasympathetic innervation develops with time, and may be seen as early as 3 years' post-transplant. Neurohormonal influences will therefore be seen to have an increasing effect, as this innervation progresses. Whilst this should not be relied upon for a response to hypervolemia, it should be considered when administering medications that profound bradycardia and asystole may occur with partial reinnervation, in response to anticholinesterases.

The ECG in 80% of recipients will demonstrate two p-waves due to the posterior portion of the atrial walls being retained from the original heart. The p-waves from the recipient's SA node cannot be conducted across the suture line and so have no chronotropic effect on the donor heart.

CLINICAL MANAGEMENT

Preoperative assessment should focus on the functional status of the patient and heart. The patient should be assessed for evidence of rejection (Table 2.18) and infection, with evaluation of other organ involvement. Immunosuppressive therapy should be continued and any PPM or ICD checked preoperatively. Any concerns should be discussed with the transplant team.

HISTORY

A thorough history should be taken with regards to the transplant procedure (including whether an isolated cardiac or heart-lung transplant was performed), and any complications should be sought. Particular

Table 2.18 Symptoms and signs of organ rejection

Pyrexia
Anuria/oliguria/new renal impairment
Fatigue
Allograft vasculopathy – evident on biopsy
Signs of CCF – swollen ankles, weight gain, pulmonary oedema
Palpitations
Dry cough

attention should be paid to the patient's current functional status, and review of recent investigations of cardiac function. These may include echo, stress testing, cardiac catheterization and biopsy results. Details of immunosuppressive therapy should be obtained as well as any other medication the patent is taking. If the patient has a PPM or ICD, this should be checked and, if required, reprogramming arranged. If a new history of fever, shortness of breath, ankle swelling, fatigue or palpations is volunteered, graft rejection should be considered and investigated thoroughly.

EXAMINATION

- A midline sternotomy scar.
- Pacemaker/ICD box may be present – normally in the infra-clavicular region.
- Phrenic or recurrent laryngeal nerve palsies may be present. These may lead to sputum retention and chest infection postoperatively.
- Features of CCF may be seen in rejection.
- Consequences of immunosuppressive therapy
  - *Tacrolimus* – HTN, diabetes, neurotoxicity, renal insufficiency and post-transplant lymphoproliferative disorder (PTLD)
  - *MMF* – Petechial rash of thrombocytopaenia or clinical features of anaemia

INVESTIGATIONS

- *FBC* – Anaemia, leukopaenia, thromocytopaemia or pancytopaenia may be present in the context of long-term immunosuppressive therapy usage.
- *U&Es* – Evidence of renal impairment secondary to rejection or immunosuppressive therapy.
- *LFTs* – Evidence of hepatic damage from immunosuppressive therapy.

- *ECG* – Two p-waves may be observed and RBBB/LAHB is not uncommon in transplant patients although this rarely progresses to complete heart block. Chronic ST-segment abnormalities may be observed; heart transplant recipients undergo accelerated atheromatous disease even if there is no CAD disease at time of transplant.
- *Echo* – evaluation of graft function, particularly LVEF.
- *Exercise testing* – if no recent data available, consider exercise testing to evaluate for functional capacity and evidence of accelerated atheromatous disease.
- *PPM/ICD interrogation* – ensure correctly functioning and reprogram for the perioperative period if required.

INTRAOPERATIVE MANAGEMENT

MONITORING

Standard monitoring as per AAGBI guidance for the type of surgery should be observed.

- *ECG* – Monitoring of at least two leads (II and V5) for early detection of ischaemia.
- *Invasive arterial blood pressure monitoring* – If large volume shifts are anticipated. Strict asepsis should be observed due to the immune suppression.
- *Noninvasive cardiac output monitoring* – Can be useful if large volume shifts are expected to monitor stroke volume and avoid reduction in preload.

PHYSIOLOGICAL TARGETS

- *Heart rate* – The decreased/absent vagal tone will result in a increased resting heart rate, fluctuations to this should be minimized in the intraoperative period and bradycardias treated with direct-acting drugs such as isoprenaline.
- *Blood pressure* – A normal blood pressure should be targeted perioperatively, and large falls in SVR should be avoided. Meticulous attention to volume status should be observed to ensure

adequate filling. Consider preloading with intravenous fluid boluses when fluid shifts are expected.

ANAESTHETIC CONSIDERATIONS

The choice of anaesthetic agents and technique should largely be based on the type of surgery the patient is undergoing, regional techniques and general anaesthesia, with ETT, or LMA are considered equally safe, although a slow induction is generally recommended. Caution should be exercised to avoid large or rapid falls in SVR, and adequate filling should be ensured, as the denervated heart will be unable to increase cardiac output in response to precipitous falls in SVR via increases in HR and contractility, as discussed previously.

Bradycardia and hypotension should be treated with direct acting drugs such as isoprenaline, adrenaline, phenylephrine and noradrenaline. Indirect therapies may have no response or an unpredictable response. Beta and alpha blockers will also act as expected.

Due to partial reinnervation of the heart, patients may have profound bradycardic and even asystolic events in response to neostigmine and other reversal agents. These should be avoided where possible and if used, caution should be taken because the bradycardia will not respond to glycopyronium or atropine. Ideally, if muscle relaxation is required, steroid-based agents should be used which can be reversed with sugammadex.

For patients in whom calcium channel antagonists are used to reduce the required dose of ciclosporin, the dose of relaxant may need to be reduced due to the potential for prolongation of action, and monitoring of the neuromuscular block should always be undertaken.

Since liver and renal function may be compromised, caution should be exercised with drugs metabolised and excreted via these pathways, as accumulation may occur.

Consideration should be given to positioning and careful moving and handling of these patients, as immunosuppressive therapy will increase the risk of skin damage, and there may be a degree of skeletal frailty.

POSTOPERATIVE CARE

Admission to critical care should be considered on a case-by-case basis. Although this decision should not differ significantly from normal practice for any given procedure, preoperative organ impairment may be influential. Careful fluid balance should be maintained in all patients due to the relative preload-dependence. Patients should also be monitored for signs of renal impairment.

Immunosuppressive drugs should be continued in the postoperative period, and if there are any queries these should be discussed with the home transplant team. General principles apply, including avoidance of medications which interfere with cytochrome p450 enzymes. If these are unavoidable or if hepatic/renal failure occurs, levels of immunosuppressive agents should be checked, with continuation of the normal regimen pending advice from the transplant team.

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Central nervous system

ELEANOR CHAPMAN

3

AUTONOMIC DYSREFLEXIA

Autonomic dysreflexia is a medical emergency seen in patients with spinal cord injuries at or above T6. It is characterised by profound hypertension, headaches, sweating and nasal congestion caused by unopposed sympathetic stimulation below the spinal lesion.

PATHOPHYSIOLOGY

Noxious stimuli – classically bladder distension (80%) but also bowel distension, bowel pathology, urinary infections, skeletal fractures, painful stimuli, sexual activity and uterine contractions stimulate the sympathetic chain causing vasoconstriction and hypertension. Lesions at T6 become relevant as this is the point at which splanchnic circulation (the major blood reservoir) becomes vasoconstricted leading to severe hypertension. The vasoconstriction response is further exaggerated by an increased sensitivity to adrenaline and noradrenaline seen in spinal injury patients. Baroreceptors detect the hypertension and send signals via cranial nerve IX and X. The parasympathetic inhibitory response takes effect above the level of the injury leading to bradycardia, nasal congestion, flushing and sweating but the inhibitory pathways are terminated at the level of injury. The hypertension can lead to intracranial and retinal haemorrhage, pulmonary oedema and myocardial ischaemia.

MANAGEMENT

Patients and carers should be educated in the importance of avoiding the most common causes – urinary
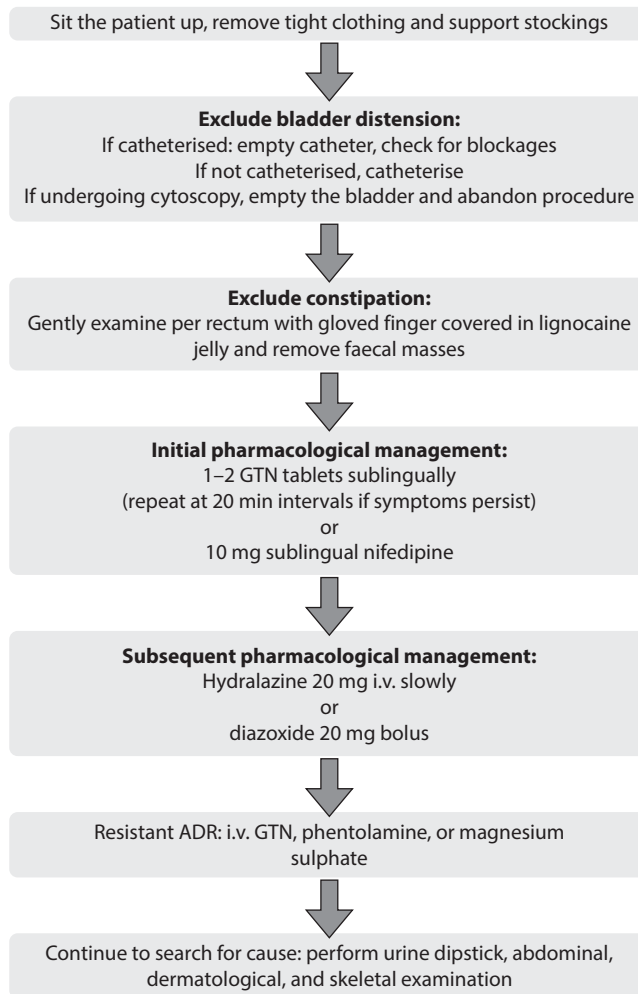
Figure 3.1 Management of an acute attack of autonomic dysreflexia.

retention, blocked catheters and constipation. In an acute attack, the management is detailed in Figure 3.1.

ANAESTHETIC IMPLICATIONS

PREOPERATIVE ASSESSMENT

Patients with spinal cord injuries have multiple comorbidities associated with their injury as well as being at risk of autonomic dysreflexia and often present late with acute pathology. Each patient should be assessed with this in mind. Patients who frequently suffer from autonomic dysreflexia should be identified establishing triggers, frequency and severity. Where appropriate use preoperative antihypertensive agents.

MONITORING

High-risk patients and those undergoing high-risk procedures (cystoscopy) should have invasive BP monitoring in addition to standard AAGBI monitoring. Anaesthetists should have a low threshold for urinary catheterisation to avoid the possibility for urinary retention.

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ANAESTHETIC TECHNIQUE

Spinal anaesthesia is an established technique in spinal injury patients, effectively abolishing autonomic dysreflexia. Epidural anaesthesia is proven in reducing autonomic dysreflexia in labouring women and is recommended for up to 48 hours after birth. Epidurals are less effective in general and urological procedures. General anaesthesia provides some protection from autonomic dysreflexia. Patients often require less induction agents due to altered pharmacokinetics and lack the sympathetic response to hypotension. Extensive fluid resuscitation and the use of vasopressors is frequently required.

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BRAIN-STEM DEATH

Brain-stem death is a clinical state where irreversible cessation of the integral function of the brain-stem can be equated to the death of the individual and allow a medical practitioner to diagnose death.

DIAGNOSIS OF DEATH BY NEUROLOGICAL CRITERIA

There are three essential components:

- Fulfilment of essential preconditions
- Exclusion of potentially reversible contributions
- Formal demonstration of coma, apnoea and absence of brainstem activity

PRECONDITIONS

They should have an irreversible brain injury of known aetiology, be apnoeic, on a ventilator and be unresponsive.

EXCLUSION CRITERIA

There should be no evidence that this state is due to depressant drugs. Detailed history of sedative drugs ingested or administered is reviewed and any altered metabolism which could be causing the comatose state excluded. Before performing brain-stem tests:

- Allow four times the half life of sedative drugs.
- If drug assays are available, check plasma concentrations (e.g. thiopentone < 5 mg/L, midazolam < 10 μg/L).
- Use reversal agents (e.g. naloxone, flumazenil) where appropriate.

Table 3.1 provides guidance for circulatory, metabolic and endocrine values. However, it is recognised that many disturbances such as diabetes insipidus are the result of brain-stem death rather than the cause of the coma and should not delay brain-stem testing.

FORMAL DEMONSTRATION OF COMA, APNOEA AND ABSENCE OF BRAINSTEM ACTIVITY

The tests should be performed by two medical practitioners:

- Each registered for more than 5 years
- One a consultant
- Neither a member of the transplant team

Table 3.1 Potentially reversible circulatory, metabolic and endocrine disturbances

Parameter	Acceptable value at brain-stem testing
Temperature	>34°C
Mean arterial pressure	Consistently >60 mmHg
$PaCO_2$	<6.0 KPa
PaO_2	>10 KPa
pH	7.35–7.45
Na^+	115–160 mmol/L
K^+	>2 mmol/L
PO_4	0.5–3.0 mmol/L
Mg^{2+}	0.5–3.0 mmol/L
Glucose	3.0–20 mmol/L

One doctor performs a first test with the second observing. If there is no evidence of brain-stem function, the patient is reconnected to the ventilator to allow arterial blood gases to return to baseline parameters. The tests are then repeated by the second doctor with the first observing. If again there is no evidence of brainstem activity, then brain-stem death is confirmed. The time of death is documented as the end of the first test.

EXAMINATION OF BRAIN-STEM AREFLEXIA

- Pupils are fixed and do not respond to sharp changes in intensity of light (mesencephalon, cranial nerves II and III).
- No corneal reflex when gently brushed with a swab (pons, cranial nerve V and VI).
- No oculo-vestibular reflexes – No eye movements when 50 mL of ice cold saline is put into each external auditory meatus. Meatus must be clear of ear wax (pons, cranial nerves VIII, III, IV and VI).
- No response to painful stimulus when applied to supra-orbital ridge (pons, V and VII).
- No gag reflex when pharynx is stimulated with a spatula (medulla, cranial nerves IX and X).
- No cough reflex when stimulated by bronchial suction catheter (medulla, cranial nerve X).

APNOEA TEST

The apnoea test should be performed last and only if no brainstem reflexes are shown to be present thereby avoiding any hypoxia or metabolic damage to potentially recoverable brain tissue. It is performed as follows:

- Increase FiO_2 to 1.0.
- Check ABG and confirm $PaCO_2$ and SaO_2 correlate with monitored values.
- With $SaO_2 > 95\%$, reduce respiratory rate to allow a slow rise in $ETCO_2$.
- Once $ETCO_2$ rises above 6.0 KPa check ABG and confirm that $PaCO_2$ is at least 6.0 KPa and

pH < 7.40. These values should be adjusted for those with chronic CO_2 retention and those who have received bicarbonate to a $PaCO_2 > 6.5$ KPa.
- Blood pressure should remain stable throughout the apnoea test.
- Disconnect the ventilator and entrain oxygen at a rate of 5 L/min to ensure oxygenation by mass action transfer.
- If maintenance of oxygenation is difficult, a Waters circuit may be used and CPAP applied and possibly recruitment manoeuvres prior to apnoea.
- After 5 minutes if there is no respiratory response a further ABG should be taken to ensure there has been a minimum increase in $PaCO_2$ of 0.5 KPa.
- The patient should be reconnected to the ventilator.

ANCILLARY INVESTIGATIONS

In the UK, ancillary investigations are not required to support the confirmation of brain-stem death. However, in complicated cases such as extensive maxillofacial injuries precluding reliable brain-stem reflex testing or in high cervical cord injury it is at our professional discretion to use further investigations to demonstrate inadequate cerebral blood flow or function. The most popular and validated technique is EEG testing although this is less useful with any residual sedation. Four vessel angiography can be helpful but may be technically difficult to arrange. CT angiography is more practical and shown to be highly specific but not sensitive at diagnosing brain-stem death.

PATHOPHYSIOLOGY OF BRAIN-STEM DEATH

CARDIOVASCULAR

- An initial sympathetic storm of tachycardia and hypertension is shortly followed by more prolonged hypotension, myocardial impairment, pulmonary oedema and arrhythmias.

ENDOCRINE

- Lack of ADH secretion causes diabetes insipidus, hypovolaemia, hypernatraemia, hyperosmolality and hypokalaemia.
- Variable loss of function of the anterior pituitary results in reduced cortisol and thyroid hormones.
- Hyperglycaemia may occur secondary to reduced insulin secretion, treatment of hypernatraemia with 5% dextrose or high levels of circulating catecholamines.

COAGULATION

- Tissue thromboplastin release causing disseminated intravascular coagulation.

TEMPERATURE REGULATION

- Loss of hypothalamic temperature control
- Fall in metabolic rate/muscle activity
- Peripheral vasodilatation

METABOLISM

- Reduced myocardial energy stores
- Increased anaerobic metabolism
- Increased lactate and free fatty acids
- Metabolic acidosis

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DISEASE OF THE NEUROMUSCULAR JUNCTION

The neuromuscular junction (NMJ) can be affected by a number of rare but diverse disease processes (Table 3.2), leading to severe weakness, bulbar and respiratory failure.

Table 3.2 Disease affecting the neuromuscular junction

- Myasthenia gravis
- Lambert–Eaton myasthenic syndrome
- Autoimmune neuromyotonia
- Toxins and drugs
  - Botulism
  - Tetanus
  - Organophosphates

MYASTHENIA GRAVIS

AETIOLOGY AND PATHOLOGY

Myasthenia gravis (MG) is the most common disease of the neuromuscular junction, affecting 2 in 100,000 of the population. There is autoimmune mediated destruction of the post-synaptic acetylcholine (ACh) receptor. A strong link between MG and disorders of the thymus gland exists. The thymus is thought to be responsible for production of the antibodies.

CLINICAL PRESENTATION AND DIAGNOSIS

Initially patients may complain of double vision and demonstrate ptosis. This progresses to bulbar and proximal limb weakness and fatigue. The weakness is often fluctuant. Approximately 20% of patients have respiratory muscle involvement and, if severe, require intubation and ventilation. Diagnosis can be difficult and a high index of suspicion is required. Patients with MG show a rapid improvement in muscle strength lasting about 5 minutes after IV injection of edrophonium (the Tensilon® test). Unfortunately, this test has low sensitivity and specificity. Electromyography (EMG) shows a diminishing response of the muscle action potential on repeated stimulation, but again has low specificity. The presence of anti-acetylcholine receptor antibodies is diagnostic.

Rarer forms of MG include a seronegative variant with antibodies directed against muscle specific kinase (anti-MuSK) or drug induced (e.g. penicillamine). MG is associated with other autoimmune disease.

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MANAGEMENT

The management of MG involves:

- Enhancing neuromuscular transmission with long-acting anti-cholinesterases.
- Immunosuppression with steroids, azathioprine or cyclophosphamide.
- Thymectomy for those with evidence of an enlarged thymus on CT scan.
- Intravenous immunoglobulin or plasma exchange. Usually reserved for those with myasthenic crisis or resistance to standard therapy (e.g. anti-MuSK myasthenia).
- Withdrawal of precipitating drugs.

ANAESTHETIC IMPLICATIONS

Preoperative

MG patients may present for incidental surgery or for thymectomy. History of symptoms and functional ability, with particular reference to respiratory and bulbar function, is important to predict postoperative course. Respiratory failure or consistently low FVC may indicate the need for postoperative ventilation. A soft voice, a history of dysphagia and choking indicates bulbar dysfunction. Close cooperation with a neurologist is vital to optimize patients prior to surgery.

Omit anticholinesterases on the morning of surgery as they interfere with all neuromuscular blocking agents. In the absence of respiratory or bulbar involvement, sedative premedication is acceptable. MG is not a contraindication to peripheral nerve or neuraxial blockade.

Induction

Due to the reduced number of ACh receptors, patients with MG are relatively resistant to normal doses of suxamethonium but large or repeated doses may produce dual block. They are sensitive to nondepolarising neuromuscular blockers requiring as little as 10% of the usual dose. The muscle relaxation provided by volatile agents is often sufficient to permit intubation. If muscle relaxants are needed, rocuronium followed by reversal with sugammadex has been shown to give good recovery. Patients should be ventilated

through the procedure and neuromuscular function monitored with a nerve stimulator.

Postoperative

Most patients can be extubated at the end of the procedure but those with bulbar dysfunction should be extubated when fully awake. Patients with respiratory or severe bulbar failure may benefit from a period of postoperative ventilation. Reintroduce anticholinesterases as soon as possible, starting at a lower dose. Adequate analgesia is essential and epidural analgesia may be considered.

BOTULISM

AETIOLOGY

The clinical manifestation of infection with *Clostridium botulinium*, a Gram positive, spore forming organism capable of producing a number of neurotoxins (A-G). Several of these target proteins in the presynaptic region of the human neuromuscular junction, preventing ACh release. Infection usually results from contaminated foods or wounds (increasingly common in intravenous/subcutaneous drug users).

CLINICAL FEATURES

Classically botulism presents with bilateral cranial nerve palsies, which may be preceded by GI symptoms if the toxin is ingested. Common symptoms and signs include:

- Blurred vision, diplopia, dilated/fixed pupils and ptosis
- Expressionless face
- Bulbar involvement: dysarthria, dysphagia and aspiration
- Autonomic dysfunction: postural hypotension, dry mouth, urine retention and constipation

The disease can progress rapidly (hours to days) to a descending flaccid paralysis with loss of tendon reflexes. Ventilatory support may be required if there is respiratory muscle involvement. The sensory system is unaffected and the patient remains fully lucid. Patients are often apyrexial. CNS imaging and CSF values are normal, whilst EMG demonstrates NMJ

blockade. Diagnosis requires the identification of the toxin in stool or plasma.

TREATMENT

The only effective treatment is botulism antitoxin and, as it only neutralizes unbound toxin, it should be given as early as possible. Wounds require surgical debridement. Antibiotics may accelerate toxin release and are not indicated in foodborne botulism but do have a role in wound botulism. Patients should be managed in the ICU and intubation should occur early. Patients may require ventilation for months before recovery.

TETANUS

AETIOLOGY AND PATHOLOGY

Tetanus though rare in the UK (10–15 cases per year) is still prevalent in parts of the developing world. It is caused by the Gram positive bacillus *Clostridium tetani*, which is ubiquitous within the environment. Inoculation is usually through a deep wound but tetanus can complicate surgery, snakebites or childbirth. The clinical effects result from the exotoxin, tetanospasmin, which is taken up by local nerve terminals and transported to the inhibitory interneurones in the brain stem and spinal cord where it interferes with the release of GABA and glycine. Motor neurones, preganglionic sympathetic neurones and parasympathetic centres are all affected.

CLINICAL FEATURES

Muscle spasms, rigidity and autonomic disturbance occur. Masseter and facial muscle spasm give rise to the characteristic 'lockjaw' and 'risus sadonicus'. Limb spasms may be severe enough to fracture bones and avulse tendons. Spasm of the laryngeal/pharyngeal muscles can occlude the airway while spasm and rigidity of the chest wall may result in respiratory failure. Spasms may occur spontaneously or in response to the slightest of visual, auditory or touch stimulation. Autonomic dysfunction is common. Resting tachycardia and hypertension are frequent but during autonomic

storms can rapidly convert to severe hypotension and bradycardia.

MANAGEMENT

Effective vaccination programmes have almost eradicated tetanus from the developed world. In established disease the aims of treatment are to:

- Neutralise free toxin with human tetanus immune globulin and enhance short-term immunity with tetanus toxoid.
- Eradicate the infection – metronidazole is the antibiotic of choice. Thoroughly debride all wounds.
- Control the spasms – nurse in a quiet environment keeping stimulation to a minimum. Midazolam is helpful initially but if spasms continue a propofol infusion may be required, necessitating intubation and ventilation. Neuromuscular blockade is used for those with refractory spasms. Other agents include magnesium, phenobarbitone, dantrolene and intra-thecal baclofen.
- Control autonomic disturbance – beta blockers, vagolytics, clonidine and magnesium have all been used.

These measures, combined with fastidious supportive care, have significantly reduced mortality. Functional recovery is good in survivors.

ORGANOPHOSPHATE POISONING

Organophosphates are found in insecticides but are also used as chemical weapons. Organophosphates can be absorbed by inhalation, ingestion or through the skin and mucous membranes. They irreversibly inhibit acetylcholinesterase resulting in the overstimulation of muscarininc and nicotinic receptors. This produces a 'cholinergic crisis' characterized by:

- *Central effects* – agitation, convulsions, loss of consciousness, respiratory depression.
- *Parasympathetic effects* – bronchoconstriction, bradycardia, salivation, miosis, vomiting, abdominal cramps, diarrhoea.
- *Neuromuscular effects* – muscle fasciculations progressing to weakness.

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A delayed polyneuropathy may develop weeks after the acute event. Diagnosis is initially clinical but may be supported by red blood cell anticholinesterase activity. Organophosphates can be detected in blood and urine.

MANAGEMENT

First consideration is decontamination of the patient and protection of healthcare workers. There is little evidence to guide treatment and initial supportive measures include:

- Intubation and ventilation for respiratory failure or loss of consciousness
- Haemodynamic support including fluid, vasoactive agents, treatment of dysrhythmias
- Control of seizures, initially with benzodiazepines

Specific management includes:

- Atropine – to reverse the muscarinic effects
- Pralidoxime (low dose 1–2 g slow IV) – capable of reactivating AchE if given early but is ineffective against certain organophosphates
- Magnesium has shown benefit in recent trials

If the patient is able to access healthcare services before respiratory compromise, outcome is usually good.

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EPILEPSY

A chronic illness occurring in approximately 1 in 200 of the general population and characterised by recurrent (2 or more) seizures. Common associations include genetic, congenital and developmental conditions in the young, tumours in the over 40s and trauma or infection at any age. Approximately 20%–30% of patients remain refractory to drug therapy or develop intolerable side effects. Many become candidates for epilepsy surgery. Epilepsy is also the direct cause of approximately 1000 deaths per year in the UK as a result of status epilepticus, trauma from seizures or sudden unexpected death in epilepsy.

CAUSES

Most cases are idiopathic and a definite cause is only found in 25%–35%. Specific causes include:

- Genetic, e.g. juvenile myoclonic epilepsy
- Trauma – depressed skull fractures or intracranial haemorrhage
- Tumours – particularly slow-growing frontal tumours
- Infection – meningitis or encephalitis
- Cerebrovascular disease – 6%–15% of stroke patients
- Alcohol – lowers seizure threshold
- Others, e.g. dementia, multiple sclerosis, metabolic disorders

PATHOPHYSIOLOGY

Brain electrical activity is normally well controlled but in epileptogenic disorders normal regulatory functions are altered. Sudden and disordered

neuronal activity occurs resulting in the clinical manifestations of epilepsy, in particular:

- Appearance of pacemaker neurones
- Introduction of significant excitatory synaptic connections
- Loss of postsynaptic inhibition

Pacemaker neurones appear to be the centre of the epileptic focus and have the capacity to produce spontaneous burst discharges seen as inter-ictal spikes on the EEG. Increased cellular activity and loss of normal inhibitory tone allows spread to surrounding areas, resulting in uncontrolled neuronal firing and seizure activity. Changes in membrane flux, impaired GABA-mediated synaptic inhibition and alterations in local neurotransmitter levels are implicated in this process.

CLASSIFICATION

Epilepsy may be generalized or partial (Table 3.3) but there are over 40 different types recognized. Generalized epilepsies occur in 20% of cases, involve both hemispheres and are associated with initial impairment of consciousness. Simple partial seizures are caused by localised discharge with no impairment of consciousness. In complex partial seizures (CPS) the initial focal discharge spreads widely and secondary loss of consciousness occurs. CPS is the most common seizure disorder in adults and includes temporal lobe epilepsy (TLE). In many patients, high

Table 3.3 Classification of the epilepsies

| **Generalised Epilepsy** |
| • Generalised absence – petit mal |
| • Generalised tonic-clonic – grand mal |
| • Myoclonic |
| • Tonic-clonic |
| • Atonic |
| **Partial Epilepsy** |
| • Simple |
| • Complex partial – temporal lobe epilepsy |
| • Partial onset with secondary generalization |
| Also, pseudoseizures and non-epileptic seizures |

quality MR imaging demonstrates hippocampal sclerosis in patients with TLE and extended temporal lobectomy may offer a reduction of seizure frequency and severity.

ANTI-EPILEPTIC DRUGS

The aim is to achieve a seizure-free patient with minimal drug-related side-effects. Correct choice of anticonvulsant (Table 3.4) involves consideration of seizure type and history, patient age and side-effects. Therapy is initiated with a single agent given to produce therapeutic plasma levels according to NICE guidelines (Table 3.5). If seizures continue, or if unacceptable side-effects develop, another agent is substituted. Monotherapy will control seizures in many patients but some require addition of second- or third-line agents.

Table 3.4 Anticonvulsants: Commonly prescribed drugs, and side-effects

Drug	Side-effects
Phenytoin	Skin rash, drowsiness, ataxia, slurred speech, gingival hypertrophy, excess hair growth, anaemias, neuropathy. Measure blood concentrations.
Sodium Valproate	Tremor, drowsiness, weight gain, alopecia, raised hepatic transaminase, thrombocytopaenia. Avoid in pregnancy.
Carbamazepine	Rash, double vision, ataxia, hyponatraemia, thrombocytopaenia.
Phenobarbitone	Drowsiness, rash, osteomalacia, anaemia, folate deficiency.
Ethosuximide	Nausea, drowsiness, anorexia, photophobia.
Lamotrigine	Rash, drowsiness, double vision, headache, insomnia, tremor, flu-like symptoms.
Levetiracetam	Dizziness, drowsiness, insomnia, ataxia, tremor, headache, behavioural problems
Primidone	Nausea, nystagmus, sedation, anaemias, ataxia
Vigabatrin	Visual field defects, drowsiness, psychotic reactions
Gabapentin	Drowsiness, dizziness, headache
Clobazam	Drowsiness, tolerance

Table 3.5 NICE guidelines for pharmaceutical management of epilepsy

Seizure type	First-line agent	Adjunctive agents	Other agents in tertiary care	Do not offer (may worsen seizures)
Generalised tonic–clonic	Carbamazepine Lamotrigine Oxcarbazepinea Sodium valproate	Clobazam Lamotrigine Levetiracetam Sodium valproate Topiramate		(If there are absence or myoclonic seizures, or if JME suspected) Carbamazepine Gabapentin Oxcarbazepine Phenytoin Pregabalin Tiagabine Vigabatrin
Tonic or atonic	Sodium valproate	Lamotrigine	Rufinamide Topiramate	Carbamazepine Gabapentin Oxcarbazepine Pregabalin Tiagabine Vigabatrin
Absence	Ethosuximide Lamotrigine Sodium valproate	Ethosuximide Lamotrigine Sodium valproate	Clobazam Clonazepam Levetiracetam Topiramate Zonisamide	Carbamazepine Gabapentin Oxcarbazepine Phenytoin Pregabalin Tiagabine Vigabatrin
Myoclonic	Levetiracetam Sodium valproate Topiramate	Levetiracetam Sodium valproate Topiramate	Clobazam Clonazepam Piracetam Zonisamide	Carbamazepine Gabapentin Oxcarbazepine Phenytoin Pregabalin Tiagabine Vigabatrin
Focal	Carbamazepine Lamotrigine Levetiracetam Oxcarbazepine Sodium valproate	Carbamazepine Clobazam Gabapentin Lamotrigine Levetiracetam Oxcarbazepine Sodium valproate Topiramate	Eslicarbazepine acetate Lacosamide Phenobarbital Phenytoin Pregabalin Tiagabine Vigabatrin Zonisamide	

(*Continued*)

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Table 3.5 (Continued) NICE guidelines for pharmaceutical management of epilepsy

Seizure type	First-line agent	Adjunctive agents	Other agents in tertiary care	Do not offer (may worsen seizures)
Prolonged or repeated seizures and convulsive status epilepticus in the community	Buccal midazolam Rectal diazepam Intravenous lorazepam			
Convulsive status epilepticus in hospital	Intravenous lorazepam Intravenous diazepam Buccal midazolam	Intravenous phenobarbital Phenytoin		
Refractory convulsive status epilepticus	Intravenous midazolam Propofol (not in children) Thiopental sodium			

PREOPERATIVE ASSESSMENT

Pay particular attention to:

- Seizure frequency, type and pattern
- Current anticonvulsant therapy (including plasma levels)
- Complications of anticonvulsant therapy (Table 3.4)
- IQ – poorly controlled, chronic epilepsy is associated with low IQ and this may make informed consent problematic

Continue anticonvulsant therapy up to and including the day of surgery. If premedication is needed use a benzodiazepine. If the patient is likely to remain nil by mouth postoperatively, consider placing a nasogastric tube to facilitate enteral administration of anticonvulsant drugs. Only phenytoin, sodium valproate and lamotrigine are available in injectable form.

ANAESTHETIC AGENTS AND THE EEG

The action of anaesthetic agents on the EEG is complex and usually dose related. Some may be proconvulsant in low doses whilst having anticonvulsant action at higher doses.

IV INDUCTION AGENTS

Propofol has a profound dose-dependent effect on the EEG causing activation at small doses and burst suppression at higher (clinical) doses. It activates the EEG in TLE and may produce seizures and opisthotonos in nonepileptic patients, possibly via a glutaminergic mechanism. Propofol has been widely used in the treatment of status epilepticus resistant to other therapies.

Most barbiturates are anticonvulsant at normal clinical doses and thiopental may be used to control seizures. Thiopental infusion is beneficial in status epilepticus.

Etomidate increases frequency of postoperative seizures and will prolong seizures when used in ECT. Avoid where possible.

Ketamine is proconvulsant at analgesic doses but anticonvulsant at anaesthetic doses.

Midazolam and other benzodiazepines have anticonvulsant effects and are widely used in the treatment of seizures.

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INHALATIONAL AGENTS AND NITROUS OXIDE

The EEG effects are dose dependent. EEG activity is maintained with sevoflurane, isoflurane and desflurane concentrations <1 MAC, although background epileptiform activity may be suppressed. At concentrations >2 MAC the EEG becomes isoelectric. Enflurane has a proconvulsant action, exaggerated in the presence of elevated $PaCO_2$. With nitrous oxide, anticonvulsant effects predominate at higher inspired concentrations.

OPIOIDS

Opioids have minimal effect on the EEG at normal clinical doses although marked EEG slowing occurs at high doses. Fentanyl causes modest activation of the EEG, whereas high-dose opioids result in EEG slowing. Alfentanil increases epileptiform activity and has been used to provoke seizure activity during electrocorticography. Use with caution. Remifentanil is safe and may be used during epilepsy surgery with minimal impact on intraoperative EEG recording. Tramadol lowers seizure threshold and should be avoided.

NEUROMUSCULAR BLOCKERS

Use these drugs with caution as they may mask seizure activity. Succinylcholine increases EEG activity but not seizure activity. It is not advised in cases of prolonged status due to potentially fatal rises in potassium levels. Antiepileptic agents are enzyme inducers which may cause resistance to the aminosteroids – neuromuscular monitoring is advisable. Atracurium's metabolite laudanosine has epileptogenic potential in animals but not in humans.

LOCAL ANAESTHETICS

At low plasma levels lidocaine has anticonvulsant-like actions, whilst at high levels it causes CNS excitation including the provocation of seizures.

ANAESTHETIC TECHNIQUE

General or local anaesthesia are suitable taking care to avoid factors known to precipitate seizures (Table 3.6). Avoid agents that are proconvulsant.

Table 3.6 Causes of seizures in the perioperative period

- Preexisting epilepsy
- Subtherapeutic anticonvulsant levels
- Hypoxia
- Hypercarbia
- Proconvulsant drugs/anaesthetic agents
- Electrolyte disturbances
  - Hyponatraemia
  - Hypoglycaemia
  - Uraemia
- Related disorders
  - Head injury
  - Eclampsia

Intraoperative seizures are rare but may be masked by neuromuscular blockade. Unexpected tachycardia, hypertension and increase in end-tidal CO_2 are warning signs. Particular vigilance needs to be taken in poorly controlled epileptics and those undergoing high risk surgery such as craniotomies. Intraoperative cerebral electrical activity monitoring may be indicated. An intravenous bolus of propofol or thiopental, followed by deepening of anaesthesia, is usually sufficient to bring seizures under control.

POSTOPERATIVE CARE

Continue anticonvulsant therapy under the guidance of a neurologist. Recurrent seizures, leading to status epilepticus, are more common in the postoperative period in patients with preexisting epilepsy (Table 3.6). Treatment of postoperative seizures must be rapid and aggressive and precipitating factors corrected. Plasma levels of antiepileptic agents should be checked and doses adjusted if necessary. If seizures occur post-neurosurgery a CT head scan is obligatory to rule out any potentially surgically treatable cause, e.g. extradural or subdural haematoma or pneumocephalus.

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Table 3.7 The Glasgow Coma Scale

Best motor response (observed in the upper limb)	Score
Obeys commands	6
Withdraws from painful stimuli	5
Localizes to painful stimuli	4
Flexes to painful stimuli	3
Extends to painful stimuli	2
No response	1
Best verbal response	
Orientated	5
Confused speech	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Eye-opening response	
Spontaneously	4
To speech	3
To pain	2
None	1

HEAD INJURY

1.4 million people a year suffer head injuries in the UK. Most are mild but 10% are moderate or severe resulting in significant mortality and permanent disability. All age groups are affected. It is the leading cause of death and disability in young adults.

Traumatic brain injuries (TBI) are classified according to the Glasgow Coma Scale (GCS – Table 3.7), mild 13–15, moderate 9–13 and severe 13–18.

PRIMARY AND SECONDARY BRAIN INJURIES

TBIs are often referred to as primary or secondary. The primary injury is the damage done immediately after impact causing haemorrhage (extradural, subdural, subarachnoid or intraventricular) or shearing of the white matter tract (diffuse axonal injury). Little can be done to reverse this damage. As the injury evolves, the brain swells, ICP increases and cerebral perfusion drops resulting in cerebral ischaemia. This is the secondary injury and is preventable. It is worsened by hypotension, hypoxaemia, hypercarbia, fever, hyperglycaemia and seizures. All are associated with trauma and can be prevented and modified. The identification, prevention and treatment of secondary brain injury is the principle focus of care for patients with severe head injury.

RESUSCITATION

Secure the airway and maintain adequate oxygenation and blood pressure in all patients with severe head injury because secondary brain injury begins and continues from the moment of impact.

AIRWAY AND BREATHING

Severely head-injured patients are unlikely to be able to protect their airway and often have impaired gas exchange – hypoxaemia occurs in up to 65%. Intubation is recommended in anyone with GCS <8 or who are unable to maintain their respiratory goals.

Intubation can be hazardous. Cervical spine control with collar and blocks may need to be removed and replaced by manual inline stabilisation. A full stomach should be assumed and a rapid sequence induction undertaken. Increasingly rocuronium (1 mg/kg) rather than suxamethonium

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(1.5 mg/kg) is being used as the patient remains paralysed for longer, increases in ICP are minimised and it can be rapidly reversed with sugammadex. Preoxygenation is mandatory and oropharyngeal suction may be necessary with maxillofacial injuries.

Thiopentone and propofol may cause hypotension that will need rapid correction. Ketamine was thought to be contraindicated in suspected head injury patients, despite being haemodynamically stable and being a potent analgesic, due to concerns of a rise in ICP. This has now largely been debunked and it may be neuroprotective through free-radical scavenging. A short-acting opiate is advised to obtund the response to laryngoscopy. As the patient is likely to remain intubated and ventilated, an endotracheal tube with a subglottic suction port should be used. Excessively tight tube ties should be avoided.

Once the airway is secured, control ventilation aiming for a $PaCO_2$ of 4.5–5.0. Hyperventilation may precipitate cerebral ischaemia. The target PaO_2 is given in the management goals in Table 3.8; values greater than normal (13 KPa) are unnecessary and FiO_2 should be adjusted accordingly.

Commence sedation (propofol is recommended) as the patient is likely to need transfer for CT scan. Adequate sedation reduces the cerebral metabolic rate but additional boluses of neuromuscular blocking agents will be needed to prevent coughing and unnecessary surges in ICP.

CIRCULATION

Hypotension results in reduced cerebral perfusion; maintenance of systemic blood pressure is a prerequisite for good neurological outcome after head injury. NICE guidelines recommend mean arterial pressure to be maintained at 80 mmHg or higher by infusion of fluid and vasopressors. Hypotension in an adult should always trigger a search for other causes of blood loss. Volume replacement may be achieved with an isotonic crystalloid (Ringer's lactate or 0.9% saline). Severe dilutional anaemia should be avoided with appropriate use of blood products. Studies of hypertonic saline have not proved definitively beneficial although future studies may warrant their introduction. An arterial line is recommended.

Table 3.8 Summary of management goals in TBI

Systems	Management goals
Airway	Early tracheal intubation if GCS ≤ 8 or unable to maintain respiratory goals
Respiratory	Avoid hypoxia, maintain, SaO_2 > 97% PaO_2 > 11 kPa
	Maintain $PaCO_2$ 4.5–5.0 kPa
	Hyperventilation, $PaCO_2$ value of 4.0–4.5 kPa reserved for impending herniation
CVS	Avoid hypotension, maintain MAP > 80 mm Hg
	Replace intravascular volume; avoid hypotonic and glucose-containing solutions
	Use blood as necessary, reverse coagulopathy
	Vasopressor agents as necessary to maintain CPP
Brain	Monitor ICP; avoid ICP > 20 mm Hg
	Maintain CPP > 60 mm Hg
	Adequate sedation and analgesia
	Hyperosmolar therapy, keep Na^+ < 155 mmol L^{-1}, P_{osm} < 320 mosm L^{-1}
	CSF drainage
	Treat seizures
	Barbiturate coma, decompressive craniectomy, hypothermia, all reserved for elevated ICP refractory to standard medical care
Metabolic	Monitor blood glucose, aim for 6–10 mmol L^{-1}
	Avoid hyperthermia
	DVT thromboprophylaxis

DISABILITY AND DYSFUNCTION (NEUROLOGICAL)

Assess conscious level using GCS but localizing signs and pupillary reaction must also be recorded.

EXAMINATION

About 40% of head-injured patients have associated injuries which may affect outcome. These should be sought during a full trauma survey and appropriate investigations. Severe extracranial injuries should

be dealt with immediately to avoid hypotension or ventilatory failure. Active bleeding and chest and abdominal injuries must be treated aggressively, but it is sufficient to stabilise non-life-threatening injuries.

TRANSFER

Severe head-injured patients will need immediate transfer (Table 3.9) for CT head and c-spine and often a full trauma series. Most moderate and severe injuries require intra-hospital transfer for neurosurgical management at short notice and outside normal working hours. Transfer is a hazardous procedure if poorly managed. For keys to successful transfer see Table 3.10.

NEUROSURGICAL INTERVENTION

Treatment of an expanding intracranial haematoma requires urgent surgical intervention within 4 hours. Additional procedures include insertion of external ventricular drains, insertion of ICP bolts and occasionally decompressive craniectomies.

Table 3.9 NICE recommendation of indication for CT head scan following injury

- GCS less than 13 on initial assessment
- GCS less than 15 at 2 hours after the injury
- Suspected open or depressed skull fracture
- Any sign of basal skull fracture[a]
- Post-traumatic seizure
- Focal neurological deficit
- More than one episode of vomiting
- Amnesia for events more than 30 minutes before impact

CT should also be performed immediately for the following risk factors if loss of consciousness or amnesia since the injury:

- Age 65 years or older
- Coagulopathy[b]
- Dangerous mechanism of injury[c]

[a] Haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign
[b] History of bleeding, clotting disorder, current treatment with warfarin
[c] A pedestrian or cyclist struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or 5 stairs

Table 3.10 Guidelines for transfer of head-injured patients

- The patient must be stabilized prior to transfer
- The doctor escorting the patient should be of appropriate seniority and have sufficient skill to recognize and treat deteriorations that may occur during transfer
- All equipment and drugs, including oxygen, should be checked prior to departure
- Patients with GCS 8 or less require intubation
- All intubated patients require:
  - Sedation (propofol), to prevent increases in ICP
  - Paralysis (vecuronium or atracurium), to prevent coughing and to facilitate mechanical ventilation
  - Analgesia (e.g. fentanyl), if indicated

Monitoring should be of the same standard as expected in an operating theatre

  - ECG, invasive BP, SpO_2 and temperature are the minimum acceptable
  - End tidal CO_2 should be controlled
  - Urinary catheter, volume recorded

OTHER MANAGEMENT

- Hyperosmolar therapy, Mannitol 20% ($0.25–1g\ kg^{-1}$) or hypertonic saline (2 mL/kg 5% solution) may be used as a temporary means of reducing ICP following consultation with the neurosurgical centre.
- Anticonvulsants are used after post-traumatic seizures but should not be used prophylactically.
- Both hypothermia and hyperthermia are associated with worsened outcome – maintain normothermia.
- Avoid glucose containing fluids; maintain at $6–10\ mmol^{-1}$.
- Patients on warfarin with a strong suspicion of TBI should be reversed immediately with prothrombin complex.
- Thiopental infusion is used for those with refractory raised ICP. It should be titrated using EEG to cause burst suppression. There is no evidence that it improves outcome.

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CROSS-REFERENCES

INFLAMMATORY BRAIN DISEASE

The blood–brain barrier is effective in reducing the exposure of the brain to many chemicals but it is not able to prevent the chronic effects of neuro-inflammation. Increased oxidative stress has been implicated in many neurological conditions. The inflammatory state may produce structural change, disruption of neuronal communication and accumulation of intra- and extracellular material. These changes may have a global or regional distribution and some functional structures may be more susceptible than others.

DEMENTIA

AETIOLOGY AND PATHOPHYSIOLOGY

The most common cause of dementia is Alzheimer's disease (AD) – Table 3.11. There are currently about 750,000 people in the UK with a form of dementia

Table 3.11 Types of dementia

Type of dementia	Proportion of total cases (%)
Alzheimer's disease (AD)	62
Vascular dementia (VaD)	17
Mixed dementia (AD and VaD)	10
Dementia with Lewy bodies	4
Fronto-temporal dementia	2
Parkinson's dementia	2
Other dementias	3

and it is estimated that this will rise to 940,000 by 2021.The incidence increases with age:

- 40–64 years: 1 in 1400
- 65–69 years: 1 in 100
- 70–79 years: 1 in 25
- >80 years: 1 in 6

What is common between the various forms of dementia is the presence of a chronic neuroinflammatory state with reactive microglia triggered release of inflammatory cytokines and increased oxidative stress, ultimately resulting in neurodegeneration and neuronal death. Accumulation of beta-amyloid protein in AD, plaques and neurofibrillary tangles may represent effects of ongoing inflammation. Similar inflammatory processes are thought to occur in multiple sclerosis and Parkinson's disease. Research is examining the use of NSAIDs and antibiotics, such as minocycline, in the hope that neuronal dysfunction in dementia can be minimised with anti-inflammatory strategies.

CLINICAL FEATURES

Dementia is a complex of related signs and symptoms in which diverse areas of cognition, including memory, language and problem solving, deteriorate. Dementia syndromes are chronic and progress slowly. They may have been present for at least six months before diagnosis, manifest as increasing impairment of activities of daily living. Delirium, in contrast, is an acute confusional state.

DRUG TREATMENT

Reversible anticholinesterase medications are the mainstay of the pharmacological management of dementia – they do not treat the disease but may slow the deterioration in some patients. Currently, donepezil, galantamine and rivastigmine are approved in the UK for the treatment of mild to moderate AD. They counteract the deficiency in cholinergic neurones and increase the concentration of neuronal acetylcholine. The glutamatergic agent memantine may also have a useful role in dementias. Frontotemporal dementia, in contrast, shows deficiencies in the serotonin and dopamine neurotransmitter systems.

ANAESTHETIC IMPLICATIONS

Preoperative assessment includes a thorough physical examination of all systems since many patients with dementia are elderly, with unreported symptoms or comorbidities. Assessment of the degree of mental impairment should be made using the Abbreviated Mental Test Score or Mini Mental State Examination – scoring under 6 or 23, respectively, is suggestive of dementia or delirium.

Consent

Patients with dementia may present difficulties with informed consent. The Mental Capacity Act, 2005 states that there should be a default presumption of capacity – 'every adult has the right to make his or her own decisions and must be assumed to have capacity to do so unless it is proved otherwise'. Therefore, attempts to support the patient to make his or her own decisions should be made and any actions or interventions must be in their best interests. It is advisable wherever possible to involve relatives in discussions and decisions.

Drug interactions

Sensitivity to, recovery from, sedative or anaesthetic agents may be unpredictable. The failure in the cholinergic pathways in the brain renders patients suffering with AD very susceptible to the effects of anticholinergic drugs, in particular atropine which can cross the blood–brain barrier. The administration of atropine may worsen a patient's neurological symptoms and be undesirably sedating.

Anticholinesterase medication can antagonise the effects of nondepolarising neuromuscular blocking drugs and potentiate succinylcholine.

POSTOPERATIVE CARE

Pain assessment can be particularly difficult and often requires assessment using nonverbal cues or charting of visual analogue scales. Inadequate pain management is associated with delayed ambulation, loss of appetite, increased postoperative morbidity and worsening of the preexisting cognitive impairment.

Chronic alcohol abuse can be a cause of dementia and is associated with the development of Wernicke's encephalopathy and Korsakoff's psychosis. In such patients, alcohol withdrawal needs to be anticipated in the perioperative period and managed accordingly.

CREUTZFELDT–JAKOB DISEASE

In contrast to the slowly progressing forms of dementia, Creutzfeldt–Jakob disease (CJD) develops over a period of a few months. Various forms of CJD are recognised. Sporadic CJD usually affects those over the age of 60, familial CJD displays autosomal dominant inheritance and variant CJD (vCJD) can have an incubation period of many years and was until recently attributed to the ingestion of contaminated beef products. Between 1996 and 2006 there were 161 recorded cases of vCJD in the UK. Iatrogenic causes of CJD include transmission from the use of human-derived growth hormone, cadaveric dura mater grafts, EEG and brain stereotactic needles.

AETIOLOGY AND PATHOPHYSIOLOGY

Although the exact detail is still poorly understood, CJD is caused by accumulation of a highly stable and resistant protein called the prion (PrP). The abnormal form has the designation PrP^{TSE} for Transmissible Spongiform Encephalopathy. PrP^{TSE} accumulates in the brain and initiates an inflammatory cascade which eventually results in the formation of neuronal vacuoles and neuronal cell death. Testing for PrP^{TSE} is unreliable and the absence of the prion does not indicate absence of infectivity. PrP^{TSE} is resistant to standard chemical and physical methods of

inactivation and even autoclaving is unreliable in removing all traces of the protein. Only incineration at 850°C or higher can guarantee PrP elimination.

ANAESTHETIC IMPLICATIONS

For anaesthesia of known or suspected cases of CJD, use single-use equipment, which is incinerated following use. If single use laryngoscope handles are not available, they should be protected by a disposable sheath. Standard universal precautions should be taken including the use of disposable gloves, waterproof apron, gown, mask and eye protection.

Samples should be clearly labelled 'Biohazard' and the laboratory informed of the nature of the material before its arrival. Clinical waste from high- or medium-risk tissue (the central nervous system, posterior eye and pituitary gland) in a patient at 'increased risk' of vCJD should be incinerated. Where possible, procedures should be performed last on the operating list to allow adequate time for appropriate decontamination.

As PrP^{TSE} has been found to be concentrated in lymphoid tissue as well as the CNS, concerns have been raised regarding the anaesthetic management of patients for tonsillectomy. However, in 2008, the AAGBI published guidance recommending that single use laryngoscopes were not mandatory for such cases.

BLOOD TRANSFUSION

Since 2003, there have been three documented cases of vCJD transmission via blood transfusion, resulting in tighter restriction of blood products usage. UK blood donors who received blood transfusion in the UK were removed from the donor pool and all plasma products are now imported into the UK (mainly from Germany and the United States).

NOTIFICATION

Although not a statutory notifiable disease, any new cases of CJD (variant or otherwise) should be referred by the neurologist to the National Creutzfeldt–Jakob Disease Surveillance Unit.

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MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is an autoimmune mediated chronic inflammatory disease of the central nervous system. It is the most common cause of nontraumatic neurological disability in young adults in North America and Europe.

AETIOLOGY

The exact aetiology is unknown but is thought to be autoimmune mediated by complex interactions between genetic susceptibility and environmental insults. The inheritance is unclear but there is increased concordance in monozygotic twins. It is

more common in countries further from the equator and some evidence for viral triggers exists as patients with MS report being affected by common viruses later than age-matched controls.

EPIDEMIOLOGY

The prevalence is roughly 200 per 100,000 population with regional variation. It is more common in the northern hemisphere and migrants who move before age 15 take on the risk of the native population. Like many other autoimmune diseases, it is more common in women than men, although in older people the gender distribution is 50:50. First presentation is usually in the third and fourth decade of life, although it can present at any age.

PATHOGENESIS

MS is characterised by the formation of sclerotic plaques that represent the end stage of a process of inflammation, demyelination and remyelination. Autoreactive lymphocytes cross the blood–brain barrier and deplete oligodendrocytes (myelin forming cells). Remyelination can occur initially but repeated attacks lead to plaques building up over the axon. Characteristically, the plaques are disseminated in space and time especially in the periventricular regions, optic nerve, brain stem and spinal cord white matter. Initially axons are preserved but later demyelination is associated with irreversible axonal and neuronal degeneration.

CLINICAL FEATURES

Clinical manifestations reflect plaques in motor, sensory, visual and autonomic systems. Many varied symptoms and signs can occur (Table 3.12). Characteristic are Lhermittes sign (an electrical sensation in the legs on neck flexion) and Uhthoff phenomenon (transient worsening of symptoms on raising the body temperature, e.g. after exercise).

RELAPSING/REMITTING MS

Eighty-five percent of patients initially present with acute episodes affecting one or several sites. Recovery from this and subsequent episodes is often complete. New episodes occur erratically but rarely

Table 3.12 Signs and symptoms of multiple sclerosis

Cerebrum	Motor weakness
	Sensory changes
	Cognitive impairment
	Affective disorders
Optic Nerve	Unilateral loss of vision
Cerebellum	Diplopia
	Vertigo
	Ataxia
	Impaired speech and swallowing
Spinal Cord	Weakness
	Spasticity
	Bladder dysfunction
Other	Pain
	Fatigue
	Temperature insensitivity

more than 1.5 per year. Relapses can be triggered by systemic infection and stress and are more common in the postpartum period. Relapses are not usually triggered by surgery, anaesthesia or vaccinations.

SECONDARY PROGRESSIVE MS

Fifty percent of relapsing remitting MS sufferers will progress to secondary progressive disease within 15 years where recovery from each episode is incomplete and there is progressive neurological deterioration.

PRIMARY PROGRESSIVE MS

In around 20% the disease is progressive from the onset.

DIAGNOSIS

Diagnosis is based on clinical, radiological (MR) and laboratory criteria. Typically there should 2 or more clinical episodes over 30 days apart with a characteristic MR imaging to confirm the diagnosis.

CLINICAL

- History of exacerbation and remission
- Clinical manifestations suggesting multiple lesions in different sites
- Optic neuritis is the presenting sign in 20%–30% patients

LABORATORY

Abnormal CSF examination:

- Increased white cell count
- Raised protein levels
- IgG oligoclonal bands on electrophoresis (found in 75%–80%)

RADIOLOGICAL

Multifocal lesions on MR scan, especially involving periventricular, cerebellum and spinal cord white matter. Gadolinium enhanced scans demonstrate active MS lesions.

TREATMENT

There is no known treatment that predictably slows the course of MS. The aim of treatment is to return function after an attack and either prevent, or increase the interval between, further attacks.

ACUTE TREATMENTS

- *Corticosteroids* – High dose methylprednisolone (oral or IV) is used for acute relapses. Prednisolone is usually introduced after 5 days. Steroids will shorten the duration of relapses but not affect long-term outcome.
- *Plasmapheresis* – For steroid resistant severe relapses.

DISEASE MODIFYING DRUGS

These drugs are used in relapsing-remitting MS. They have no value in the other types.

- β Interferon – reduces the frequency of relapses by about 30% over 2–3 years, decreases the incidence of fixed disabilities but does not delay entry into the secondary progressive phase. It can be used early in disease progression because of limited side effects. Local injection site reactions and flu-like symptoms are relatively common.
- Glatiramer acetate – induces tolerance to myelin-reactive lymphocytes. It decreases the frequency of relapses in the early stages of the disease. There may be some be some benefit in combination therapy with β interferon.
- Teriflunomide is a pyrimidine synthesis inhibitor that inhibits T cells involved in the formation of plaques. Not as effective as β Interferon.
- Natalizumab – given by infusion as monotherapy in severe disease. It reduces the relapse rate and the chance of acquiring fixed disability.

OTHER THERAPIES

- Baclofen and tizanadine are used to treat muscle spasm.
- Gabapentin is useful for painful symptoms.
- Amantidine is used to treat fatigue.
- Oxybutinin may help bladder function.
- Neuro-rehabilitation – including physiotherapy and occupational therapy.

ANAESTHETIC MANAGEMENT

Anaesthetic implications of MS are unclear and it is difficult to separate the effects of anaesthesia and surgery from spontaneous new lesion formation. Stress can exacerbate MS symptoms and measures should be taken to relieve anxiety. The majority of patients undergoing surgery will be young and otherwise well.

PREOPERATIVE ASSESSMENT

Pay particular attention to:

- Disease progression and history of relapses. Document current abnormal neurology.
- Evidence of infection – a rise in temperature of 0.5–1°C increases conduction block in demyelinated neurones.
- Respiratory reserve – respiratory function tests and blood gas analysis may be useful.
- Bulbar function.
- Autonomic dysreflexia in patients with extensive spinal cord demyelination.
- Steroid medication.

- Mental state – depression and fatigue are common.
- The presence of any pressure sores or contractures which may affect surgical positioning.

PREMEDICATION

- Continue antispasmodic medication.
- Prescribe H_2 receptor antagonists if there is any history of aspiration.
- Avoid sedative premedication.

INTRAOPERATIVE MANAGEMENT

- There is no evidence for the benefits of one induction agent or maintenance technique over another.
- Tracheal intubation is recommended if there is poor bulbar function.
- Avoid suxamethonium if there is extensive demyelination as it may cause hyperkalaemia.
- Nondepolarising muscle relaxants are safe and response is normal although monitoring of block is recommended.
- Monitor temperature – avoid hypo- and hyperthermia.
- Give deep vein thrombosis prophylaxis as there is an increased tendency to platelet aggregation.
- Pay careful attention to positioning especially in patients with contractures and pressure sores.

POSTOPERATIVE MANAGEMENT

- Postoperative analgesia should take into account the patient's respiratory function.
- Respiratory physiotherapy is recommended.
- Maintain normothermia.
- Encourage early mobilisation and return to normal function.
- The stress of surgery may provoke a relapse.

GENERAL VERSUS REGIONAL ANAESTHESIA

There is no contraindication to the use of regional anaesthesia although it is sometimes avoided in

patients with neurological signs and symptoms. Epidural and spinal anaesthesia can both be used in obstetric analgesia and anaesthesia. There is no evidence that a regional technique leads to a symptomatic relapse but the decision to proceed with either a general or regional technique should be based on careful discussion with the patient. Before performing any regional block, carefully document preexisting signs and symptoms and repeat the neurological assessment after the block has worn off.

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CROSS-REFERENCE

Autonomic dysreflexia, Chapter 30

PARKINSON'S DISEASE

Parkinson's disease (PD) is an idiopathic neurodegenerative condition characterised by tremor, rigidity and bradykinesia. Parkinsonism is the most common movement disorder affecting approximately 0.3% of the UK population overall, increasing to 3% in those aged over 65 years. It is a worldwide disease, with a very slight male preponderance and occurs in all ethnic groups.

AETIOLOGY

The aetiology is unknown, although the neurodegeneration may be induced by genetic, environmental or infectious factors. Increasing age is the most

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Table 3.13 Causes of Parkinsonism

- Genetic
  - Dominant, e.g. alpha-synuclein mutations (PARK 1) or gene duplications/triplications (PARK 4)
  - Recessive, e.g. parkin mutations (PARK 2) or DJ1 mutations (PARK 7)
- Drugs
  - Phenothiazines
  - Butyrophenones
  - Metoclopramide
- Toxins
  - Carbon monoxide
- Encephalitis
- Cerebrovascular disease
- Heavy metal poisoning and metabolic defects, e.g. Wilson's disease
- Other degenerative CNS diseases
  - Supranuclear palsy
  - Multi-system atrophy

Table 3.14 Organ systems affected by Parkinson's disease

- Autonomic
  - Postural hypotension
  - Hyperhidrosis
  - Difficulty with micturition
  - Impotence
- Respiratory
  - Upper airway dysfunction
  - Excess bronchial secretions
  - Poor cough/retained secretions
- Cardiovascular
  - Postural hypotension
  - Dysrrhythmias
- Gastrointestinal
  - Excessive salivation
  - Gastro-oesophageal reflux
- Neurological
  - Speech impairment
  - Dementia
  - Depression
  - Sleep disturbance

consistent risk factor. PD is distinguished from Parkinsonism which has several specific causes (Table 3.13).

PATHOPHYSIOLOGY

PD results from cellular degeneration of dopaminergic cells in the substantia nigra and subsequent lack of dopamine. In addition, tyrosine ß-hydroxylase, the rate-limiting step in dopamine synthesis, also diminishes. The remaining cells contain the pathological hallmark of eosinophilic Lewy bodies.

CLINICAL FEATURES

There are no specific diagnostic tests for PD and the diagnosis is made on clinical grounds. Patients exhibit the classic 'triad' of symptoms (usually asymmetrical):

- *Tremor at rest* – 'Pill rolling' at 4–6 Hz cycles per second, improved by voluntary movement
- *Rigidity* – 'Cogwheel' in nature, juddering on passive extension
- *Bradykinesia* – Paucity of movement, monotonous speech, expressionless face, shuffling gait and abnormal posture

Therapeutic trials offer confirmatory evidence, with over 90% of patients showing a good early improvement with L-DOPA.

PD is associated with a multitude of clinical features because of its multi-system effects (Table 3.14).

MANAGEMENT

Treatment is usually pharmacological, although surgical methods are useful for some patients.

DRUG TREATMENT

L-DOPA and dopamine agonists form the mainstay. L-DOPA crosses the blood–brain barrier and is converted to dopamine in the CNS. The addition of peripheral decarboxylase inhibitors (e.g. benserazide, carbidopa), which do not cross the blood–brain barrier, reduces systemic side effects. Ergot derivatives (e.g. bromocriptine, pergolide, lisuride, cabergoline, ropinirole) are dopamine receptor agonists and are reserved for adjuvant therapy or for those with severe side effects from L-DOPA. Nonergot derivatives such as pramipexole or rotigotine have a

more favourable side effect profile. Apomorphine is the only available parenteral dopaminergic drug and should be given with the anti-emetic domperidone.

Monoamine oxide inhibitors (e.g. selegiline) reduce the central breakdown of dopamine.

Anticholinergic drugs (procyclidine, benztropine, biperiden, benzhexol, orphenadrine) have limited efficacy and side effects include confusion, urinary retention and glaucoma.

Catechol-O-methyl transferase inhibitors (e.g. entacapone) improve the dopamine concentration profile, resulting in fewer on-off symptom fluctuations.

SURGICAL TREATMENT

Stereotactic pallidotomy, thalamotomy and deep brain stimulation are increasingly used as treatment for PD.

ANAESTHETIC IMPLICATIONS OF PARKINSON'S DISEASE

People with Parkinson's disease undergoing surgery have increased mortality and length of stay. Missing dopaminergic medication can result in life threatening complications such as neuroleptic malignant syndrome.

PREOPERATIVE ASSESSMENT

Focus on identifying and optimising the systemic complications (Table 3.14). Record the severity of symptoms as well as the potential for cooperation with a regional technique.

Give the usual Parkinson's medication despite being nil by mouth for surgery. If subsequent doses are likely to be missed, consider inserting a nasogastric tube to give the medication. If an extended period of gastroparesis is anticipated, discuss with a neurologist.

Preoperative investigations include ECG, CXR, spirometry (restrictive pattern often identified) and vital capacity measurement. Adequate preoperative hydration is essential to minimize the risk of cardiovascular complications. Patients with PD have a high incidence of gastro-oesphageal reflux. A urinary catheter may be required, particularly for longer procedures.

CONDUCT OF ANAESTHESIA

Regional anaesthesia may avoid some of the complications of general anaesthesia but tremor, confusion and exaggerated hypotension may cause difficulties in awake patients. Potential drug interactions should be considered (Tables 3.15 and 3.16). These are common but can be unexpected.

Table 3.15 Interaction of drug therapy for Parkinson's disease with other therapy

- Amantadine – anticholinergics, levodopa, CNS depressants and stimulants, diuretics
- Biperiden – antihistamines, CNS depressants, quinidine, metoclopramide
- Levodopa/benserazide – MAOIs, potent opioids, antihypertensives, sympathomimetics, ferrous Sulphate
- Benzhexol – MAOIs, phenothiazines, antihistamines, antidepressants, disopyramide, amantadine
- Benztropine – MAOIs, phenothiazines, antihistamines, antidepressants, disopyramide, amantadine
- Bromocriptine – erythromycin, metoclopramide, alcohol
- Cabergoline – neuroleptics, ergot alkaloids, dopamine antagonists, macrolides, hypotensive agents
- Entacapone – MAOIs, rimiterole, sympathomimetics, methyldopa, antidepressants, iron, bromocriptine
- Levodopa/carbidopa – MAOIs, antihypertensives, sympathomimetics
- Orphenadrine – phenothiazines, antihistamines, antidepressants, amantadine, disopyramide, terodiline
- Pergolide – dopamine antagonists, antihypertensives, anticoagulants, competition for protein binding
- Pramipexole – amantadine, cimetidine, ranitidine, diltiazem, quinidine, quinine, verapamil, digoxin, procainamide, triamterene, sedatives, alcohol
- Procyclidine – phenothiazines, antihistamines, antidepressants, amantadine, disopyramide, ketoconazole, quinidine, MAOIs
- Ropinirole – antihypertensives, antiarrhythmics, neuroleptics, oestrogens, alcohol, drugs affecting P450
- Selegiline – fluoxetine, sertraline, paroxetine, pethidine, non-selective MAOIs, tricyclic antidepressants, anticoagulants, digitalis

Table 3.16 Interaction of commonly used drugs in patients taking treatment for Parkinson's disease

Metoclopramide	Extrapyramidal s/e or parkinsonian symptoms	Use Domperidone
Droperidol		5H-T3 (eg Ondansetron etc) or antihistamine (e.g. Cyclizine)
Prochlorperazine		
Analgesics		
Pethidine	↑BP and rigidity with Selegiline	?resemble Malignant Hyperpyrexia
Synthetic opiates	↑rigidity	dose related
Volatiles	↑l-dopa induced dysrhhythmias	Avoid Halothane if ECG abnormal
Antihypertensives	Exaggerated drug effects	
	↑BP with Indirect antihypertensives and Selegiline	
Antipsychotics	Extrapyramidal s/e or parkinsonian symptoms	Use 'atypical' antipsychotics e.g. Clozapine or Olazapine
Tricyclics	↑l-dopa induced dysrhhythmias	
SSRI's	↑BP and cerebral exitation with Selegiline	

Thiopental, etomidate and ketamine have been used although there has been concern regarding a hypertensive reaction to ketamine in some patients. Despite reports of propofol causing exacerbation of dyskinesia, it is safe to use. Volatile agents may potentiate hypotension and dysrrhythmias. NSAIDS and simple analgesics may be used. Fentanyl, alfentanil and morphine may cause rigidity in some cases. The use of local and regional techniques minimises opioid requirements. Depolarising and nondepolarizing neuromuscular agents can be used safely as can neostigmine and glycopyrrolate.

Chlorpromazine, droperidol and metoclopramide may exacerbate Parkinsonian symptoms; ondansetron and cyclizine are safer options.

POSTOPERATIVE CARE

Mobilizing may be delayed because of Parkinsonian symptoms and drug regimes should be reinstituted early. Pay attention to pressure areas and thromboprophylaxis. Postoperative confusion can be treated with a benzodiazipine or 'atypical' antipsychotic whilst the cause is sought. PCA may be difficult to manage and regional techniques should be continued as appropriate. Physiotherapy for chest (impaired cough, laryngeal function and increased secretions) and mobility facilitates early mobilisation and minimises postoperative complications.

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CROSS-REFERENCES

PERIPHERAL NEUROPATHIES

Peripheral neuropathies may be either acute or chronic and may be related to demyelination or destruction of the nerve axons (Table 3.17). They are rare in but may pose a significant challenge to the anaesthetist, including:

- Respiratory muscle weakness
- Bulbar weakness
- Sensitivity to neuromuscular blocking agents
- Hyperkalaemia following suxamethonium

Table 3.17 Causes of peripheral neuropathy

Acute
- Guillain–Barré syndrome
- Porphyria
- Toxins
- Vasculitis

Chronic
- Chronic inflammatory demyelinating polyneuropathy
- Paraneoplastic
- Myeloma
- Toxins/drugs, e.g. heavy metal intoxication, fluoroquinonlones

GUILLAIN-BARRÉ SYNDROME (GBS)

AETIOLOGY

GBS is the most common form of acute peripheral neuropathy in the UK with an incidence of 2 per 100,000 population. Approximately two-thirds of patients have suffered from a preceding upper respiratory or gastrointestinal tract infection. GBS may also be triggered by other events including surgery but there is little evidence to link it to current vaccines. There are several well-recognized variants of GBS (Table 3.18).

PATHOPHYSIOLOGY

GBS is thought to be an autoimmune disease of the peripheral nervous system in which there is immune mediated destruction of the peripheral nerve myelin sheaths or occasionally the axons.

CLINICAL FEATURES

GBS presents with an ascending, symmetrical, weakness typically starting in the lower limbs. It progresses over hours to days reaching a nadir by 3–4 weeks. The weakness may ascend to affect muscles of respiration and bulbar function. In extreme circumstances, all cranial and peripheral motor nerves may be affected, mimicking brain stem death. Examination demonstrates a flaccid paralysis

Table 3.18 Variants of Guillain-Barré syndrome

Variant	Nerve pathology	Relative frequency in UK	Symptoms and signs
Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)	Demyelination	90%	Predominantly motor involvement with, pain & autonomic dysfunction.
Fisher syndrome		5%	Ophthalmoplegia, ataxia, areflexia
Acute sensory and motor axonal neuropathy	Axonal degeneration	5% (65% in China)	Motor and sensory involvement. Autonomic dysfunction less severe than AIDP
Acute motor axonal neuropathy			Purely motor involvement
Acute sensory neuropathy			Primarily sensory, evidence of motor involvement on nerve conduction studies

with loss of deep tendon reflexes. There are often associated sensory symptoms (e.g. pain, parasthesia), though few (if any) demonstrable deficits. The majority of patients are severely affected and unable to walk without help, and 25% require ventilation for respiratory or bulbar failure.

Autonomic dysfunction is frequent with marked fluctuations in blood pressure, tachycardia, bradycardia (including asystole), urinary retention, ileus (up to 15% of patients) and sweating. ECG changes are also common.

INVESTIGATIONS

Lumbar puncture demonstrates a raised CSF protein (may be normal in the first week) with normal CSF white cells. Nerve conduction studies demonstrate slow conduction velocities with either demyelination or axonal loss. Antiganglioside antibodies may be present (useful in the diagnosis of Miller Fisher Syndrome).

TREATMENT

Immunotherapy

Plasma exchange and intravenous immunoglobulin (IG) both reduce time to recovery, autonomic dysfunction and the requirement for mechanical ventilation. IGg is now a standard of care unless there is a contraindication. Steroids have no place in the treatment of GBS and may be deleterious.

Respiratory support

Twenty-five percent of patients require intubation for respiratory failure. Regular forced vital capacities (FVC) are vital to detect deterioration in respiratory function. FVC <20 mL/kg indicates an imminent need for ventilation but earlier intubation may be necessary if bulbar weakness coexists. Tracheostomy is indicated if the period of ventilation is prolonged.

Pain

Most patients suffer nociceptive and neuropathic pain. Management combines regular analgesics including opioid agents and adjuncts such as gabapentin or amitriptyline.

Thromboembolic prophylaxis

Graduated compression stockings and low molecular weight heparins should be used where possible.

Autonomic complications

Hypertension and tachycardia can be managed with short-acting beta-blockers, e.g. labetolol or esmolol, whereas hypotension may require a norepinephrine infusion. Bradycardias can usually be managed with vagolytics but may be severe enough to warrant temporary pacing. Ileus can be supported by nasogastric and rectal tubes and, if required, TPN.

ANAESTHETIC IMPLICATIONS

Preoperative

Many patients will be ventilated on the ICU. If not, assessment of bulbar and respiratory function will predict the need for postoperative ventilation. Ileus increases the risk of aspiration during induction. A rapid sequence induction is advisable.

Induction of anaesthesia

Suxmethonium is contraindicated because of potentially fatal hyperkalaemia, following excessive potassium release from denervated muscles. Rocuronium is a suitable alternative. Autonomic dysfunction can make induction and intubation hazardous, resulting in labile pulse and blood pressure.

There is no contraindication to peripheral nerve or neuraxial block. Very high blocks can impair respiratory muscle function.

Intraoperative

Use controlled ventilation if respiratory function is impaired. GBS patients are sensitive to nondepolarizing neuromuscular blocking agents and their use should be kept to a minimum. Consider extubating when fully awake once bulbar reflexes have returned.

Postoperative

Postoperative ventilation is often required. If patients are extubated, careful monitoring of respiratory function is vital, ideally in the ICU. Pain can be problematic since many patients are often not opiate naïve but need rapid assessment and control.

DIPHTHERIA

AETIOLOGY

Diphtheria is caused by *Cornyebacterium diphteriae*, a Gram positive bacillus. Due to immunisation it is extremely rare in the UK but still endemic in many parts of the world. It is easily spread through droplet transmission or touch. Its pathogenecity is related to the expression of an exotoxin.

CLINICAL FEATURES

Diphtheria usually presents as respiratory (pharyngitis, tonsillitis, laryngitis) or cutaneous (skin sores or shallow ulcers). Symptoms include low grade fever, malaise, anorexia, hoarse voice and sore throat. Examination demonstrates a grey/green membrane that may cover the soft palate or tonsils but can progress to cause airway obstruction. If the disease progresses, patients develop marked cervical lymphadenopathy and oedema giving a characteristic bull-neck appearance. Absorption of the toxin leads to the systemic manifestations of diphtheria, including:

- Myocarditis, dysrhythmias and heart failure.
- Demyelinating polyneuropathy principally affecting bulbar and respiratory muscles, but can involve limbs and eyes. It occurs once the acute illness is resolving. The response is often biphasic with patients deteriorating after an initial improvement. Recovery is variable but patients are often left severely disabled.

Diagnosis is clinical but can be confirmed by isolating *Corynebacterium diphteriae* from the pharynx. Diphtheria carries a mortality of 5%–10%.

MANAGEMENT

Antitoxin and benzylpenicillin (or erythromycin) should be started once diphtheria is clinically suspected. Diphtheria is a notifiable disease.

The progressive neuropathy can result in respiratory failure and approximately 20% of patients will require ventilation. Bulbar failure may require nasogastric feeding and, if severe, tracheostomy.

ANAESTHETIC IMPLICATIONS

Patients with diphtheria may present with acute airway obstruction. Endotracheal intubation is hazardous as the pharynx and larynx are oedematous and the adherent membrane may obscure the view of the larynx entirely. Early recognition of airway compromise and close cooperation with ENT surgeons is vital.

The neuropathy usually develops after the acute infection but puts the patient at risk of respiratory failure requiring postop ventilation. The chronically denervated muscles expose the patient to fatal hyperkalaemia if suxamethonium is used.

PORPHYRIAS

AETIOLOGY

A group of diseases caused by enzyme defects within the haem synthetic pathway resulting in the overproduction of porphyrins. Porphyrias can be classified in several ways, e.g. site of defect (hepatic or erythropoietic), acute or nonacute or pattern of enzyme defect. It is the acute porphyrias (acute intermittent porphyria [AIP], variegate porphyria [VP] and hereditary coproporpyria [HCP]) that are of particular relevance to the anaesthetist as these can precipitate a neurovisceral crisis. The nonacute ones are erythropoietic porphyria and porphyria cutanea tarda.

ACUTE PORPHYRIAS

AIP affects about 1 in 20,000 Europeans but is more common in Northern Scandinavia. VP is less common except in the Afrikaaner community (1:250). Acute attacks are precipitated by events that decrease haem concentrations and thus stimulate the activity of δ-amino levulinic acid (ALA) synthetase activity and accumulation of porphyrins within the tissues. These events include starvation, stress, dehydration, infection and certain drugs (Table 3.19).

Table 3.19 Drugs that precipitate porphyria

- Barbiturates
- Etomidate
- Phenytoin
- Sulphonamides
- Hydralazine
- Oral contraceptive pill

CLINICAL FEATURES

Acute attacks often present with severe abdominal pain (though often little on clinical examination), vomiting, electrolyte and autonomic disturbances (hypertension, tachycardia) and neurological involvement. Acute attacks often result in a severe neuropathy (predominantly motor) with the involvement of peripheral nerves, anterior horn cells, autonomic ganglia, brain stem and cerebellar pathways. Weakness commonly presents in proximal limb muscles but may progress, leading to tetraplegia, respiratory and bulbar failure. There are often associated sensory losses. Other manifestations include altered mental state, cranial nerve palsies, seizures and coma. Neurological recovery is often slow or incomplete. VP is also associated with bullous skin lesions on exposure to light during the acute episode. HCP is also associated with photosensitivity but symptoms are usually less severe than in AIP and VP.

MANAGEMENT

Management of an acute attack involves managing the underlying precipitant, correcting electrolyte abnormalities, and treating pain, agitation and autonomic dysfunction. Intubation and ventilation will be required if there is bulbar or respiratory failure, or a significant decrease in level of consciousness.

Specific treatment is targeted at suppressing haem synthesis and includes carbohydrate and the administration of haematin, a haem compound that produces negative feedback on ALA synthetase.

ANAESTHETIC CONSIDERATIONS

Anaesthesia is safe providing trigger factors are avoided. Adequate hydration and correction of abnormal electrolytes is crucial, and preoperative starvation should be kept to a minimum. If starvation is unavoidable, then IV glucose should be administered, although care needs to be taken to avoid hyponatraemia. Many modern anaesthetic drugs are definite or potential precipitants, but a simple and safe regimen includes:

- *Induction* – Propofol or possibly ketamine
- *Maintenance* – Propofol, nitrous oxide, halothane, isoflurane
- *Neuromuscular blockade* – Suxamethonium, vecuronium
- *Analgesia* – Morphine, fentanyl, paracetamol

Porphyria is not a contraindication to neuraxial or peripheral nerve blocks and both bupivacaine and fentanyl are acceptable.

Postoperative care depends on accurate pain management and the avoidance of infection, dehydration and starvation.

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CROSS-REFERENCE

Autonomic dysfunction, Chapter 3

PRIMARY NEUROMUSCULAR DISEASE

Destruction of upper motor neurones (UMN) or lower motor neurones (LMN) leads to paralysis of associated muscle groups. The process can be limited, producing minimal weakness and disability, or widespread and progressive leading to paralysis, bulbar involvement, respiratory failure and death. This group can present significant clinical and ethical challenges to the anaesthetist.

MOTOR NEURONE DISEASE

AETIOLOGY

Motor neurone disease (MND) describes a number of neurological disorders resulting from the degeneration of the anterior horn cells and motor cranial nuclei (Table 3.20). There is considerable overlap between the different types of MND making distinction difficult. The most common form is amytrophic lateral sclerosis (ALS), characterised by UMN and LMN lesions. Affecting 1–2 per 100,000 population, its incidence peaks between 55 and 75 years. Survival is 2–5 years from diagnosis, with death usually resulting from respiratory failure.

CLINICAL PRESENTATION

The diagnosis is clinical with investigations being used to exclude other causes of weakness. Patients usually present with progressive distal limb weakness initially in the upper followed by lower limbs. Weakness is often asymmetric and patients notice wasting of affected muscle groups. Bulbar onset is less common but presents with slurred speech progressing to dysphagia, nasal regurgitation and pulmonary aspiration. Respiratory muscle involvement rarely occurs at presentation and leads to nocturnal hypoventilation with early morning headaches and hypersomnolence progressing to respiratory failure. As the disease progresses, patients become paralysed, unable to swallow or communicate and eventually succumb to respiratory failure. Cognition remains intact in most patients.

On examination there are both upper (spasticity and hyper-reflexia) and lower motor neurone (wasting and fasciculation) signs. There are no sensory signs and autonomic dysfunction occurs late.

MANAGEMENT

Management is multidisciplinary and involves medical, symptomatic and nonpharmacological support. Riluzole is the only disease-modifying drug available and prolongs life by a median 3 months. It is generally well tolerated but may cause liver dysfunction or neutropaenia.

Respiratory

The majority of patients die from respiratory failure. The use of noninvasive ventilation (in those without severe bulbar failure) improves both length and quality of life. Ventilation via tracheostomy, though rare in the UK, also prolongs life, particularly in patients with bulbar failure. Carbocysteine is helpful in the management of thick secretions, whilst anticholinergic drugs are useful to control secretions.

Table 3.20 Motor neurone disease

	Motor neurone affected	Life expectancy	Proportion of MND
Amytrophic lateral sclerosis	UMN & LMN	2–5 years	80%
Progressive bulbar palsy	UMN & LMN	6 months–3 years	15%
Progressive muscular atrophy	Pure LMN	5–10 years	5%
Primary lateral sclerosis	Pure UMN	May be normal	0.5%

Abbreviations: LMN, lower motor neurone; MND, motor neurone disease; UMN, upper motor neurone.

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Pain

Pain is caused by immobility, cramps and spasticity and is managed with analgesics, quinine sulphate, baclofen or injection of botulinum toxin into specific muscle groups. Physiotherapy may also be helpful.

Nutrition

Malnutrition is common and associated with a decreased survival. Early speech and language therapy and the insertion of a gastrotomy tube is vital.

Psychiatric

Patients often suffer from emotional lability, anxiety and depression, and a combination of behavioural and pharmacological therapies can be helpful. A small proportion of patients develop a frontal dementia.

ANAESTHETIC CONSIDERATIONS

Preoperative

A preoperative history of disease progress, symptoms and functional ability is important to predict postoperative course. Assessment of respiratory function, including details of recent chest infection or evidence of hypoventilation, is crucial. A 30% fall in vital capacity from standing to lying suggests significant diaphragm involvement and postoperative ventilation may be necessary. A history of drooling, choking and a nasal voice suggests bulbar impairment. Severe bulbar or respiratory impairment precludes sedation for procedures such as endoscopic gastrostomy insertion.

If the disease is advanced, information about advanced directives and limitations of care must be sought.

Induction of anaesthesia

Suxamethonium may induce a fatal hyperkalaemia from the chronically denervated muscle and is contraindicated. Rocuronium is a suitable alternative.

Intraoperative

Patients with ventilatory impairment require ventilation through the procedure. Patients are sensitive to the effects of nondepolarising neuromuscular blockers and use of these agents should be minimised and their effects monitored. MND is not a contraindication to neuraxial block although high blocks may further impair respiratory function.

Postoperative

Extubate only when fully awake and laryngeal reflexes have returned. Humidified oxygen, physiotherapy and early mobilisation reduce postoperative respiratory complications. However, a period of postoperative ventilation may be required in those with respiratory failure. Adequate postoperative analgesia must be provided and peripheral nerve blocks and epidurals reduce the need for opiates.

RABIES

AETIOLOGY

Rabies is an acute encephalomyelitis following infection by a virus belonging to the lyssavirus genus. Though eradicated in the UK, rabies is endemic in many parts of the developing world. Travellers returning from overseas may have been exposed to rabies either through an infected animal bite or, rarely, cuts contaminated with infected saliva.

PATHOLOGY

The rabies virus initially replicates in skeletal muscle before ascending, via the peripheral nerves, into the CNS. Once in the CNS it replicates rapidly and disseminates around the body.

CLINICAL PRESENTATION

Symptoms are initially nonspecific and include itching or pain at the wound site. Fever, myalgia and headache follow. The investigation of choice is PCR of skin biopsies to demonstrate viral RNA. The disease may progress in two distinct patterns.

Furious rabies

This accounts for 80% of cases and is associated with periods of agitation, delirium, autonomic disturbances, and upper motor neurone/cranial nerve lesions. It is associated with the classic hydrophobia

where drinking or even the thought of water causes intense laryngeal spasm. Patients may die of acute cardio/respiratory arrest or progress to paralysis, coma and respiratory failure.

Paralytic rabies

Patients develop a progressive flaccid paralysis eventually resulting in bulbar and respiratory failure. Hydrophobia is rare.

TREATMENT AND ANAESTHETIC IMPLICATIONS

As rabies is almost invariably fatal, management is aimed at prophylaxis or postexposure prophylaxis. Advice should always be taken from the virology department.

By the time the patient starts to show symptoms, death is almost inevitable. Most patients die of multiorgan failure. If a patient survives, they show significant neurological impairment. There is no evidence base to guide management but expert consensus recommends:

- Barrier nursing
- ICU care to manage the respiratory, bulbar and autonomic dysfunction and the ensuing multiorgan failure
- Combination anti-viral therapy including:
  - Rabies vaccine
  - Human rabies immunoglobulin
  - Intravenous and intraventricular ribavirin and interferon-α
  - Intravenous ketamine (reduces rabies replication and blocks access of virus to NMDA receptor)

Sedatives may also help to induce burst suppression on the EEG. Death is to be expected and arguably palliative care would be more appropriate, particularly for those presenting late in the disease.

POLIOMYELITIS

AETIOLOGY

Since the introduction of effective vaccination programmes, polio has been eradicated from the developed world but is still endemic in parts of the Indian sub-continent and sub-Saharan Africa. It is the result of the highly infectious poliovirus and the usual route of infection is via the gastrointestinal tract.

CLINICAL FEATURES

The majority of infections are either asymptomatic or present as a 'flu-like' illness. Less than 1% of patients develop 'classical' paralytic polio. Paralytic polio follows the viral destruction of the motor neurons within the anterior horn cells, producing a flaccid paralysis and loss of associated reflexes. It usually affects the lower limbs and the weakness is often asymmetric, with proximal muscle groups being more affected than distal.

Poliovirus may invade the brain stem destroying the lower cranial nerves and give rise to bulbar polio. These patients have difficulty swallowing, speaking and suffer from recurrent aspiration. A combination of bulbar and cervical cord involvement produces bulbospinal polio where a combination of bulbar weakness and diaphragmatic involvement can severely impair respiratory function rendering the patient ventilator-dependent.

Following a period of stability some patients notice a further deterioration in function, associated with fatigue, joint and muscle pains. This is the post-polio syndrome, is poorly understood and not due to 'reactivation' of the polio virus.

TREATMENT

There is no treatment and management is supportive. Initially this involves analgesics, treating intercurrent infections and nutritional and ventilatory support. Most paralytic polio patients recover so these measures are often temporary. A few are left with long-standing weaknesses requiring on-going rehabilitation, callipers, braces and recurrent surgery for progressive deformity. Ongoing domiciliary ventilatory support may also be required.

ANAESTHETIC IMPLICATIONS

Key points in the management include:

- Avoid sedation in those with respiratory or bulbar failure.
- Avoid suxamethonium because of the risk of hyperkalaemia.

- Controlled ventilation in patients with respiratory failure.
- Awake extubation in patients with bulbar failure.
- Postoperative management in ICU for those with significant respiratory/bulbar failure.

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SPINAL CORD INJURY

AETIOLOGY

The incidence of spinal trauma is approximately 13 per million of the population per year. The cervical spine is most susceptible to injury being the least supported, although thoracolumbar junction injuries are also relatively common. Injuries to the vertebral bodies, discs and soft tissue/ligaments may all occur. Up to 50% of spinal cord injuries (SCI) are considered incomplete and appropriate early management will prevent further disability from secondary cord injury.

PATHOPHYSIOLOGY

In acute SCI, primary injury is often caused by stretching or tearing of the cord as a result of hyperflexion or hyperextension of the spinal column. The cord may also be damaged by direct, penetrating trauma or by disrupted bony structures. There is disruption of neural tissue and ischaemia. Ongoing or secondary ischaemic injury may follow due to hypotension, hypoxaemia and progressive cord oedema. This ultimately leads to an inflammatory cascade with free radical formation, cellular oedema, apoptosis and further destruction of neural tissue.

The hallmarks of chronic spinal cord injury, namely the return of reflex activity resulting in hyperreflexia and hypertonia, appear 6–8 weeks following the initial insult. Spinal cord injury not only results in neurological dysfunction but also affects other organ systems.

MANAGEMENT

AIRWAY AND BREATHING

To prevent secondary injury, ensure adequate oxygenation with intubation and ventilation as needed (Table 3.21). The degree of airway and respiratory compromise is governed by the level of the cord lesion and the presence or absence of any systemic injuries such as head or chest trauma. In cervical cord injuries, cord oedema may ascend requiring respiratory support. Lesions above C4 result in loss of both diaphragmatic and intercostal function with the possible additional loss of accessory muscle function. Remove any hard collar and immediately replace by manual inline stabilisation. Use a rapid sequence induction technique because of gastroparesis and lack of lower oesophageal sphincter tone. In the first 72 hours, suxamethonium is safe but must subsequently be avoided. The use of cricoid pressure in unstable cervical spine fractures

Table 3.21 Indications for tracheal intubation after spinal cord injury

$PaO_2 < 10.0$ kPa
$PaCO_2 > 6.5$ kPa
Vital capacity < 20 mL/kg
Aspiration risk
Associated chest/lung or facial injuries
Associated traumatic brain injury

remains controversial. The assistant should support the neck with the other hand beneath the neck or collar. There is no preferred method for laryngoscopy so use a technique that is familiar and that needs minimal flexion and particularly extension of the neck. If an awake fibreoptic intubation is planned, the collar and blocks should remain in place.

Diaphragmatic function is often spared in lesions below C5 so spontaneous ventilation may be possible but an ineffective cough is often present. Where SCI does affect breathing, remember that respiration is most effective when supine where the pressure of abdominal contents aids inspiration.

Patients with chronic SCI frequently suffer from retained secretions, recurrent chest infections and chronic respiratory failure resulting in the need for whole time, or night-time ventilatory support.

CARDIOVASCULAR

Hypertension is often present immediately after injury but this is swiftly replaced by hypotension due to loss of sympathetic pathways. Compensatory reflexes are lost, particularly if the lesion is above T1. Bradycardia may ensue as a result of unopposed parasympathetic activity. These symptoms are unresponsive to fluid therapy and should be treated with vagolytics and vasopressors.

In chronic SCI, the most common cardiovascular manifestation is autonomic dysreflexia. Cardiac arrhythmias, including vagally mediated bradycardia, and orthostatic hypotension may also continue.

DISABILITY

The degree of sensory, motor and reflex function should be documented using the American Spinal Injury Association (ASIA) chart as soon as possible. Patients should be transferred for CT head and neck or a full trauma series. An MR scan may be required to identify ligamentous injuries, establish cord integrity and aid operative planning. In patients with a depressed level of consciousness other signs suggestive of SCI include diaphragmatic breathing, priapism, bradycardia,

hypotension, areflexia and no pain response below a certain level.

GASTROINTESTINAL AND GENITOURINARY SYSTEMS

SCI produces bowel and bladder atony, gastric dilatation, paralytic ileus and urinary retention. There is an increased risk of regurgitation and pulmonary aspiration, constipation, recurrent urinary tract infections and renal calculi.

OTHER

A nasogastric tube and urinary catheter should be inserted. Venous thromboprophylaxsis should be considered although potential exacerbation of haemorrhage from systemic injuries and future surgical interventions must be considered. SCI patients have a very high risk for pressure sores so spinal boards should be removed within 30 minutes.

MANAGEMENT OF THE SPINAL INJURY

In conservative treatment the cervical spine can be immobilised using traction applied with skull tongs. This often entails prolonged bed rest although application of a halo body jacket allows some mobilisation. Surgical treatment involves open reduction and stabilization of spinal fractures along with spinal decompression. High dose steroids, vasopressor support (MAP > 85 mmHg) and therapeutic hypothermia have been explored but none are now routinely recommended.

ANAESTHETIC IMPLICATIONS

In both acute and chronic SCI, careful assessment of both the respiratory and cardiovascular systems must be carried out. Lung function tests and arterial blood gas analysis are important. Avoid respiratory depressant drugs in patients who are self-ventilating. Because this population of patients undergoes multiple procedures, they are at increased risk of latex allergy. Patients with injuries requiring non-spinal

surgery may require no anaesthesia (depending upon operation and sensory level), or with regional anaesthesia. General and regional techniques both diminish autonomic dysreflexia. Avoid suxamethonium beyond 3 days post-injury.

Obstetric patients with chronic spinal cord lesions in labour provide a particular challenge. They are highly susceptible to autonomic dysreflexia which can be induced by uterine contractions, instrumental delivery and perineal distension. Life-threatening episodes of dysreflexia may continue for up to 48 hours after delivery and epidurals should be continued during this period. If an epidural is technically impossible, then the dysreflexia may be controlled pharmacologically with hydralazine or nifedipine.

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SUBARACHNOID HAEMORRHAGE

Subarachnoid haemorrhage (SAH) is a devastating neurological disease with multisystem sequelae accounting for 5% of all strokes. SAH can be either spontaneous or traumatic (tSAH). The majority of spontaneous SAH are due to intracranial aneurysms (Table 3.22).

Table 3.22 Causes of subarachnoid haemorrhage

1. Intracerebral saccular aneurysms (85%)
2. Arteriovenous malformations (5%)
3. Carotid dissection
4. Vasculitis
5. Bleeding diatheses
6. Recreational drug use (cocaine/ecstasy)
7. Bleeding into meningeal tumours
8. Amyloid angiopathy
9. Unknown

SPONTANEOUS SUBARACHNOID HAEMORRHAGE

- The incidence is 6–12 per 100,000 population per year with the highest rates in the Finnish and Japanese populations. It is most common between ages 45 and 60.
- More common in women than men (3:2).
- Risk factors include hypertension, smoking, atherosclerosis, alcohol, cocaine abuse, polycystic kidneys, Ehler's Danlos Type 4 and familial intracerebral aneurysms.
- 1%–2% of patients who present to A&E departments with headache have SAH, although 25% of those with 'the worst headache of their lives' and abnormal neurology have SAH.
- SAH is a notable cause of maternal mortality contributing to a number of deaths in the Confidential Enquiry into Maternal Deaths triennial reports.

TRAUMATIC SUBARACHNOID HAEMORRHAGE

- The incidence is around 100 per 100,000 population per year.
- Up to 40% of patients with moderate to severe head injury have evidence of subarachnoid blood on CT scan.
- tSAH is associated with an increased incidence of hypoxia, hypotension, skull fractures, cerebral contusions and raised ICP.

- Associated vasospasm is less common than in spontaneous SAH.
- If history preceding the head injury is unclear, cerebral angiography should be performed to exclude spontaneous SAH and secondary trauma.

INTRACRANIAL ANEURYSMS

The natural history of intracranial aneurysms is incompletely understood, but the annual rupture rate is 0.5%–2%. Aneurysms >10 mm in diameter are at particular risk of haemorrhage. Aneurysms typically develop in the Circle of Willis at bifurcations because of turbulent blood flow. Common sites for rupture are the posterior communicating artery/internal carotid take-off and the anterior cerebral artery.

PRESENTATION

- Sudden, severe, 'thunder clap' headache often described as 'worst in my life'
- Signs of meningism – photophobia, neck stiffness, nausea and vomiting
- Focal neurology
- Deteriorating consciousness
- 15%–30% of patients die on aneurysm rupture, secondary to sustained rise in ICP

The SAH may be preceded by a prodromal headache days or weeks prior to the ictus. This is caused by a 'sentinel bleed' and probably represents extravasation of blood into the aneurysmal wall or subarachnoid space.

There are over 40 clinical grading scales of SAH. The recommended one is from the World

Table 3.23 WFNS classification of subarachnoid haemorrhage and associated morbidity and mortality

Grade	GCS	Motor Deficit
I	15	Absent
II	14–13	Absent
III	14–13	Present
IV	12–7	Absent or present
V	6–3	Absent or present

Table 3.24 Fisher grading of CT appearance of subarachnoid haemorrhage

Grade	Finding on CT
1	No subarachnoid blood detected
2	Diffuse or vertical layers <1 mm
3	Localised clot and/or vertical layer >1 mm
4	Intracerebral or intraventricular clot with diffuse or no SAH

Federation of Neurosurgeons (Table 3.23). This allows standardisation, aids clinical assessment and estimates prognosis. The Fisher grading, as described in Table 3.24, is often given in addition to the WFNS grading system.

DIAGNOSIS

Computed tomography

- Non-contrast CT scans are highly sensitive (>95%) for blood in the subarachnoid space within 24 hours of acute haemorrhage.
- CT scans also detect associated intracranial haematoma (ICH) and hydrocephalus.
- The location and distribution of the blood may suggest the site of bleeding.
- Blood is rapidly cleared from the subarachnoid space and the diagnostic sensitivity of CT decreases to 30% at 2 weeks.
- The increased density of blood on CT scan is a function of haemoglobin concentration and SAH may not be apparent in anaemic patients.

Angiography

- Intra-arterial catheter digital subtraction angiography (DSA) remains the gold standard for confirming the presence of an aneurysm or arteriovenous malformation.
- CT angiography (CTA) is the primary investigation to identify an aneurysm.
- MR imaging with haemosiderin-sensitive sequences is also highly sensitive but rarely used due to the logistical problems it presents.

Lumbar Puncture

- A lumbar puncture (LP) is indicated in patients with a suspected SAH in the presence of a normal CT scan.
- Blood-stained CSF that does not clear or the presence of xanthochromia suggests SAH.

MANAGEMENT

The main aims of treatment are to:

- Maintain perfusion and oxygenation in the area of the aneurysm.
- Reduce risk of rebleeding prior to definitive treatment.
- Secure the aneurysm.
- Reduce the risk of delayed neurological deficit (DND).

Airway assessment and management

Tracheal intubation and mechanical ventilation is indicated:

- In unconscious patients (GCS <8)
- In agitated patients unable to tolerate interventions
- To optimise oxygenation and normocarbia (PaO_2 >13 KPa and $PaCO_2$ 4.5–5.0 KPa)
- For control of seizures

Blood pressure control

BP is often elevated secondary to pain, anxiety and sympathetic activation and should be treated promptly if >160 mmHg to minimise the risk of further bleeding. Analgesia may be sufficient but if not labetolol infusion, β-blockers and calcium channel antagonists are all commonly used. Hypotension (systolic pressure <100 mmHg) should be avoided.

Serial assessment of neurological function

A fall in GCS may signify an extension of the bleed or development of hydrocephalus and indicate further imaging, ICP control or insertion of external ventricular drain.

Clipping versus coiling

The initial findings of the International Subarachnoid Aneurysm Trial (ISAT) favoured endovascular coiling as the treatment for ruptured intracranial aneurysm, giving an absolute risk reduction of 6%–9%. Clipping is still used when coiling is unsuitable such as in middle cerebral artery aneurysms and aneurysms with a wide neck.

GENERAL MEASURES

- Normothermia
- Glycaemic control (4.5–11 mmol/L)
- Avoid rises in ICP – bed at 30°, avoid neck ties, good analgesia, avoid coughing
- Once diagnosis is confirmed refer the patient to the local neuroscience centre

COMPLICATIONS

REBLEEDING

- The rate of rebleeding is 5%–10% in the first 72 hours. Without definitive treatment, 50% of patients will rebleed within 6 months. The mortality from a rebleed is 50%–60%, mainly due to vasospasm.
- Rebleeding is related to variations and changes in blood pressure, rather than the absolute pressure. Bed rest, analgesia and stabilisation of blood pressure are recommended to minimise the risk of rebleeding. Aggressive BP reduction should not be attempted as it may lead to reduced cerebral perfusion in the presence of raised ICP.

DELAYED NEUROLOGICAL DEFICIT

DND is any clinically detectable neurological deficit after initial stabilisation excluding rebleeding. After securing the aneurysm, avoiding DND is the major focus of treatment. DND could include

- Vasospasm and delayed cerebral ischaemia
- Hydrocephalus
- Cerebral oedema
- Fevers
- Seizures
- Electrolyte abnormalities

VASOSPASM AND DELAYED CEREBRAL ISCHAEMIA

Vasospasm is arterial narrowing demonstrated on angiography or on Doppler ultrasonography with corresponding symptoms and signs. It was originally assumed to be caused by vasoconstriction and endothelial vessel hypertrophy. However, it is now clear that the pathophysiology is multifactorial and ischaemia without vasospasm is possible leading to the term delayed cerebral ischaemia (DCI). DCI occurs in 60% of SAH patients. Its peak incidence is between day 4 and 10 and is associated with a worse outcome. Risk factors include poor grade, large subarachnoid blood load, intraventricular extension and smoking.

Diagnosis

- Angiography
- Trans-cranial Doppler ultrasonography (TCD)
- Both methods have limitations and a diagnosis is often made on clinical grounds

Treatment

Nimodipine (60 mg 4 hourly oral or 30 mg 2 hourly IV) is the only treatment shown to decrease the incidence of DCI and poor outcomes. It has minimal side effects and so is used prophylactically in all patients with SAH and continued for 21 days. Hypotension is rarely a problem in well-hydrated patients. Nimodipine appears to provide benefit by inhibiting calcium entry into smooth muscle cells and vasoactive substance release from platelets and endothelial cells. It does not reduce the incidence of angiographic vasospasm so its beneficial effects may also occur because of more general brain protective mechanisms.

Hypertension

In the past, clinicians would treat DCI and vasospasm with 'triple H' therapy: hypertension, hypervolaemia and haemodilution. This has been abandoned although it is accepted that hypovolaemia and hypotension should be avoided. There is some evidence that hypertension alone may be of benefit and consensus guidance suggests using hypertension in the treatment of vasospasm and DCI and observing for neurological improvement.

Angioplasty

- Balloon angioplasty is effective for the treatment of discreet lesions in large proximal arteries. The optimal timing is uncertain and major complications include vessel rupture, dissection, occlusion and haemorrhagic infarction.
- Chemical angioplasty using intra-arterial papaverine has an immediate but short-lived effect and has been abandoned by most centres. Other agents used include intra-arterial nicardipine, nimodipine and milrinone.

HYDROCEPHALUS

- Acute obstructive hydrocephalus (within 72 hours) is common, particularly after poor grade SAH. It is caused by subarachnoid clot in the basal cisterns and is managed with external ventricular drainage.
- Any signs of a falling GCS are an indication for immediate CT scan since it may represent impending hydrocephalus.
- Late communicating hydrocephalus (after 30 days) occurs in 25% of patients and may require a permanent ventricular shunt.

SEIZURES

- Occur in up to 25% of patients after SAH.
- Prophylactic anticonvulsant treatment is not usually recommended.

CARDIAC DYSFUNCTION

- ECG abnormalities (e.g. QT prolongation, ST segment changes) are common and may be associated with a small troponin rise. They are usually transient but may persist for up to 8 weeks.
- Neurogenic stunned myocardium is a reversible neurologically mediated syndrome characterised by ECG changes and left ventricular dysfunction. It may be asymptomatic but in severe cases can result in cardiogenic shock and pulmonary oedema.

- Cardiac abnormalities are probably the result of excessive local release of norepinephrine from myocardial sympathetic nerve terminals causing physiological myocardial denervation in the presence of normal myocardial perfusion.
- Cardiac arrhythmias occur in 35% of patients of which 5% are life threatening. These include sinus tachycardia, atrial fibrillation, torsades de pointe and ventricular fibrillation.
- Myocardial dysfunction, ECG changes and biomarker rises correlate with the degree of neurological injury but do not require change in management.
- If there is any doubt as to whether these changes are a result of SAH or primary myocardial injury, coronary angiography is diagnostic.

ELECTROLYTE DISTURBANCES

- Hyponatraemia complicates up to 30% of cases of SAH usually due to the syndrome of inappropriate secretion of antidiuretic hormone. Cerebral salt wasting syndrome may occur due to secretion of ANP causing excessive natriuresis, hyponatraemia and hypovolaemia.
- Other electrolyte disturbances including hypokalaemia, hypomagnesaemia and hypocalcaemia are also common.

RESPIRATORY COMPLICATIONS

Respiratory dysfunction or failure accounts for 23% of non-neurological causes of death after SAH. Common complications include:

- Pneumonia
- Pulmonary oedema – neurogenic or cardiogenic
- Pulmonary embolus

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CROSS-REFERENCES

Gastrointestinal tract

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MATTHEW JAMES JACKSON

CHRONIC LIVER DISEASE

Chronic liver disease is an increasingly common condition in patients presenting for elective and emergency surgery. Common causes include alcohol, hepatitis B and C, autoimmune conditions and fatty infiltration. Disorders of iron and copper storage represent less common causes. The final common pathway is cirrhosis. Histologically this is seen as severe fibrosis and nodular regeneration of the liver tissue. These patients present a significant risk for surgery; successful perioperative management requires a careful assessment of the multisystem effects of the liver disease.

MULTISYSTEM EFFECTS OF CHRONIC LIVER DISEASE

GASTROINTESTINAL TRACT

- Portal hypertension and associated oesophageal varices
- Ascites
- Delayed gastric emptying and hyperacidity
- Poor hepatocellular function
- Gastrointestinal haemorrhage

CARDIOVASCULAR SYSTEM

- Decreased peripheral vascular resistance
- Increased cardiac output
- Increased circulating volume
- Cardiomyopathy is associated with excess alcohol intake and haemochromatosis

RESPIRATORY

- Intrapulmonary shunts (not corrected with supplementary oxygen)
- Ventilation perfusion mismatch (correctable with supplementary oxygen)
- Restrictive defects due to pleural effusions and ascites

NERVOUS SYSTEM

- Encephalopathy
- Peripheral neuropathy particularly in alcoholic cirrhosis due to B vitamin deficiencies

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RENAL AND METABOLIC

- Increased sodium and water retention due to hypoaldosteronism
- Metabolic alkalosis with potassium loss
- Susceptible to renal failure: acute tubular necrosis and hepatorenal failure

BLEEDING AND CLOTTING

- Decreased production clotting factors
- Thrombocytopaenia
- Abnormal platelet function
- Hyperfibrinolysis

PREOPERATIVE ASSESSMENT

HISTORY AND EXAMINATION

- Ascertain the cause of the liver disease
- Assess for the effects of chronic liver disease on other organ systems (see above)

INVESTIGATIONS

- ECG
- Baseline arterial blood gas to assess acid base status and lactate clearance
- Urea and electrolytes
  - Hyponatraemia is common
  - Renal dysfunction
- Full blood count
  - Hb
  - Platelet count
- Coagulation
  - PT
  - APTT
  - Bleeding time
- LFTs
- Chest X-ray (look for pleural effusions, heart size)
- Echocardiogram, low threshold for request to assess ventricular function
- Lung function: restrictive or obstructive defects
- Cardiopulmonary exercise testing may be helpful in certain patients to help quantify risk.

PREOPERATIVE OPTIMISATION

- Optimise nutritional status and coexisting disease
- Ascites should be controlled in consultation with a hepatologist
- Give vitamin K for several days prior to surgery if possible
- Have adequate blood cross-matched, and anticipate the need for additional products.
- Start an intravenous infusion of glucose from the point of starvation

PREMEDICATION

Avoid premedication where possible. Where essential, oral lorazepam is an effective anxiolytic.

PERIOPERATIVE MANAGEMENT

MONITORING

Routine AAGBI monitoring. For anything other than minor surgery include urine output, temperature and arterial line. Also hourly samples for: haemoglobin, electrolytes, glucose, calcium, lactate and clotting studies are indicated. Cardiac output monitoring may be useful; however, transoesophageal echo and Doppler are relatively contraindicated in the presence of ascites.

INDUCTION AND MAINTENANCE

The pharmacokinetics of many drugs is altered in severe liver disease to a variable and often unpredictable degree. In general, the volume of distribution of water soluble drugs is increased, protein binding and hepatic metabolism are reduced. Opiates and benzodiazepines may exacerbate encephalopathy. There is relative resistance to nondepolarising neuromuscular blocking drugs: atracurium is preferred as this is not metabolised in the liver.

The choice of agents for induction and maintenance is less important than the anaesthetic goal: maintenance of end organ perfusion pressure and avoidance of hypoxia. Drugs should be given slowly and titrated to effect. Short acting agents such as desflurane and remifentanil are ideal.

Antibiotic prophylaxis should be administered prior to surgery.

ADDITIONAL GOALS

Maintenance of urine output (greater than 0.5 mL/kg/hr) is of paramount importance and this can be helped by meticulous attention to fluid balance. Avoid correcting hyponatremia rapidly intraoperatively. Intravenous glucose will be needed as hepatic stores are low.

During major surgery with marked blood loss, regular assessment of clotting should guide replacement therapy. Laboratory tests such as PT, PTT and platelet count may be used, but thromboelastography has also been found to be a reliable and effective guide to blood product requirements.

Avoid hypothermia. Wrap patient in reflective blanketing. The use of a warming mattress, warm air overblanket and the warming of all fluids are vital.

POSTOPERATIVE MANAGEMENT

Intraoperative monitoring should be continued in the postoperative period. This is best carried out in HDU. Elective ventilation should be considered following prolonged surgery, severe blood loss, continuing haemorrhage and hypothermia.

ANALGESIA

Opiates are best given via a patient-controlled system. Regional analgesia is worth considering, provided there is no coagulopathy. NSAIDs are not recommended.

OUTCOME

The leading causes of death in the surgical patient are

- Infection
- Liver failure
- Renal failure
- Haemorrhage

Other factors associated with high mortality include:

- Respiratory failure
- Cardiac failure
- Infection, particularly intra-abdominal
- Emergency surgery

Both the Child-Pugh and MELD score have been evaluated in cohorts of patients with liver disease undergoing specific procedures.

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CROSS-REFERENCES

DISORDERS OF THE OESOPHAGUS AND OF SWALLOWING

Disorders of the oesophagus and the swallowing mechanism present hazards to the patient undergoing anaesthesia because mechanisms to clear the pharynx of foreign material and keep it clear may be compromised.

PATHOPHYSIOLOGY

ANATOMICAL ABNORMALITIES

Hiatus hernia results in compromise to the functional integrity of the lower oesophageal sphincter.

Pharyngeal pouch and other diverticulae in the oesophagus may contain solid food particles or fluid for many hours after ingestion. They may also contain partially putrefied food. Discharge of

the contents of the pouch may occur with changes in posture, or unexpectedly, and present an aspiration risk.

A tracheo-oesophageal fistula is a direct communication between the trachea and oesophagus.

Tumours of the oesophagus are usually present with dysphagia which may be partial or complete at the time of surgery. Residual food particles may remain in the oesophagus as may liquid in the case of complete aphagia. Where obstruction is complete the patient will not be able to clear saliva.

Achalasia of the cardia is characterized by a hypertrophy of the muscular layer at the lower end of the oesophagus resulting in increasing obstruction to the passage of material into the stomach. Whilst the risk of regurgitation is very low in these patients, the oesophagus may be greatly dilated above the obstruction and may contain significant volumes of swallowed material. This may be demonstrated on a preoperative barium swallow. The dilated oesophagus does not contain stomach acid.

PHYSIOLOGICAL

Oesophageal motility is reduced in scleroderma. The lower oesophageal sphincter is functionally incompetent in these patients and this may result in reflux of gastric contents into the oesophagus.

A history of heartburn can often be elicited, but is absent in patients taking omeprazole.

PREOPERATIVE ASSESSMENT

The history should determine whether obstruction of the oesophagus is present and whether the patient can swallow liquid without regurgitation. A history of regurgitation of solid material hours after food suggests the presence of either a diverticulum or a dilatation above an obstruction. In the case of a pharyngeal pouch, the patient may be able to prevent filling of the pouch or empty it by pressure on the neck. Prolonged avoidance of solid food allowing free fluids may help to clear solid material. A nasogastric tube placed in the oesophagus may be useful in achalasia of the cardia. A CT or MR scan of the chest may be available.

PREMEDICATION

Antacids are of no value in oesophageal obstruction. However, where surgery relieves an obstruction, reflux of stomach acid may occur postoperatively.

Drying agents are beneficial if the patient is unable to swallow saliva.

ANAESTHETIC MANAGEMENT

Rapid sequence induction of anaesthesia is required where the oesophagus may not be empty at the time of induction.

In the case of pharyngeal pouches, the source of the risk is above the cricoid cartilage and cricoid pressure is of no value. Induction of anaesthesia in the lateral position should be considered with the pouch dependent.

Intubation of the trachea with a cuffed tube is required for protection of the airway. For oesophageal tumour resections, a double-lumen tube may be required.

If the patient is unable to swallow saliva, induction in the lateral position should be considered.

POSTOPERATIVE MANAGEMENT

Extubate the trachea with the patient awake and in the lateral position, since the risk to the airway may persist into the postoperative period.

After intubation of an oesophageal tumour, reflux of stomach acid may occur through the tube.

Surgery for achalasia of the cardia may render the lower oesophageal sphincter incompetent and be followed by acid reflux in the postoperative period.

Full competence of the protective laryngeal reflexes may take several hours to return.

CROSS-REFERENCES

The full stomach, Chapter 4
Airway and aspiration risk, Chapter 26
Hiatus hernia, Chapter 4

HIATUS HERNIA

Hiatus hernia is a common condition caused by the migration of a portion of the gastrointestinal tract

through the oesophageal hiatus in the diaphragm. It is of concern to the anaesthetist as it is associated with regurgitation and airway soiling when the patient is obtunded. Airway management is based on the balance between perceived risks and benefits; this calculation is hindered as many of the specific risks are unknown. Therefore, caution is advised.

LOWER OESOPHAGEAL SPHINCTER

The lower oesophageal sphincter (LOS) is an anatomically indistinct area of the oesophagus found around the diaphragmatic hiatus, 3–5 cm long. In health the LOS is reinforced by the diaphragmatic crura. The difference between gastric pressure and LOS pressure is known as the barrier pressure. Reflux occurs when gastric pressure is higher than that of the LOS.

The normal response to an increase in gastric pressure is an increase in LOS pressure to maintain the barrier pressure. Numerous forms of hernia are described. The sliding type occurs in 95% of cases; here the gastro-oesophageal junction passes into the thorax. This leads to impaired barrier function of the lower oesophageal sphincter and increased irritation of the lower oesophagus by gastric acid.

INVESTIGATION

Hiatus hernia may be discovered on investigation of reflux symptoms with endoscopy or incidentally on radiological studies. Its appearance radiologically is a mediastinal gas bubble lying behind the heart.

MANAGEMENT

Management focusses on symptom control. Medical management includes lifestyle changes, proton pump inhibitors, H_2 receptor antagonists and rarely prokinetics. Patients may self-medicate with antacids. The preferred surgical management is with a laparoscopic fundoplication.

PREOPERATIVE ASSESSMENT

HISTORY

Although often associated with symptoms of reflux oesophagitis, a hiatus hernia may be symptomless;

equally, reflux oesophagitis may occur in the absence of hiatus hernia. There are no symptoms specific to hiatus hernia.

A higher incidence of hiatus hernia is associated with the following:

- Increasing age
- Obesity
- Pregnancy
- Previous gastro-oesophageal surgery
- Skeletal deformities: kyphosis, scoliosis, pectus excavatum

Key features in the history are

- Epigastric or retrosternal pain and heartburn, promoted by bending or lying down, pregnancy or obesity, relieved by antacids
- Discomfort or 'crushing' chest pain due to distension of the stomach with food
- Waterbrash and reflux of bitter fluid into the pharynx and mouth
- Dysphagia is rare, and usually denotes oesophageal stenosis
- A persistent cough refractory to usual treatment may suggest regurgitation and aspiration

INVESTIGATIONS

- *Full blood count* – Chronic blood loss is common, resulting in iron-deficiency anaemia
- *Chest X-ray* – To exclude aspiration pneumonia

PERIOPERATIVE MANAGEMENT

PREMEDICATION

Consideration should be given to the preoperative use of antacids, proton pump inhibitors or H_2 receptor antagonists, if not already prescribed. Sodium citrate is preferred as it is residue free; it should be taken immediately prior to induction due to a short period of action. Metoclopramide or domperidone will increase LOS pressure and reduce gastric transit time.

AIRWAY

If the patient is asymptomatic, there are no factors present likely to increase the risk of regurgitation

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and the surgery itself is short and does not require tracheal intubation, then spontaneous ventilation using a laryngeal mask following antacid premedication may be considered.

However, if there is any doubt about the patient's safety, then full precautions to prevent regurgitation and aspiration of stomach contents must be employed, i.e. preoxygenation, rapid sequence induction with cricoid pressure, and tracheal intubation. This is mandatory if the hiatus hernia is symptomatic.

EMERGENCE

- Ensure extubation with the patient in the lateral position and as awake as possible
- Repeat administration of sodium citrate via nasogastric tube
- Semi-recumbent or sitting position, when practical

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CROSS-REFERENCES

Obesity, Chapter 4
Airway and aspiration risk, Chapter 26

MALNUTRITION

Malnutrition occurs when there is a negative balance between nutritional supply and demand. Reduced supply states are caused by mechanical obstructions, poor absorption and psychogenic eating disorders; increased demand occurs in hypermetabolic states such as sepsis, trauma and cancer. Malnutrition is a common problem and occurs in in approximately half of surgical patients, resulting in an increased incidence of postoperative complications.

PATHOPHYSIOLOGY

Patients have a reduced muscle mass and fatigue easily. Their tissues are thin and physiological reserve is limited. This results in delayed postoperative ambulation, respiratory complications, bed sores, wound infections and in extremis multi-organ failure.

PULMONARY FUNCTION

Diaphragmatic muscle mass falls in a linear fashion with body weight. There is a fall in vital capacity and maximal ventilatory volume, an increased incidence of postoperative respiratory failure and difficulty in weaning from mechanical ventilation. Decreased surfactant production and emphysematous changes in the lung cause alveolar atelectasis.

CARDIOVASCULAR FUNCTION

Myocardial muscle loss occurs alongside skeletal muscle in malnutrition. At rest patients are frequently bradycardic and hypotensive and have ECG abnormalities. The cardiovascular system lacks the reserve to increase cardiac output at times of stress.

ADDITIONAL FEATURES

- Depletion of serum proteins
- Hypoalbuminaemia results in interstitial and pulmonary oedema and reduced binding of metabolites, drugs and toxins
- Anaemia, due to folate and/or iron deficiency
- Low serum transferrin
- Low T-lymphocyte count and immune function
- Impaired antibody response
- Low serum IgA
- Pseudocholinesterase deficiency in severe malnutrition (serum albumin <2 g dL^{-1})

PREOPERATIVE ASSESSMENT

NUTRITIONAL ASSESSMENT

Many of the indices of malnutrition lack specificity and are poor predictors of perioperative morbidity and mortality.

Malnutrition is defined as:

- *Moderate* – 15% loss of ideal body weight
- *Severe* – 30% loss of ideal body weight

CLINICAL ASSESSMENT

Assess each organ system for symptoms and signs of dysfunction; investigate any attempts to optimise nutrition preoperatively

- General inspection – Evidence of fat and muscle loss, quality of skin, hair and fingernails
- Reduced pulmonary function – Complaints of shortness of breath on minimal exertion
- Heart failure – Peripheral oedema, orthopnoea
- Recent history of multiple infections
- Presence of a feeding tube

INVESTIGATIONS

- *ECG* – AV block, prolonged Q–T interval
- *Echocardiogram* – Reduced myocardial contractility
- *Pulmonary function tests* – FVC 50 mL kg^{-1} in the absence of obvious lung disease; reduced maximal ventilatory volume
- *Routine bloods* – Electrolyte abnormalities, low serum proteins, anaemia

PREOPERATIVE OPTIMISATION

Preoperative nutrition should be discussed with surgeons and dieticians. Where possible, surgery should be delayed to allow for nutritional assessment and implementation of a feeding plan. In intestinal failure, total parenteral nutrition (TPN) is used; otherwise, the enteral route is used. Ideally feeding should occur for at least seven days and the appropriate precautions should be taken to prevent and monitor for refeeding syndrome.

Where TPN has been used, examine patient for potential complications which include improper central line placement, line infection, fluid overload and metabolic disturbances (hyperglycaemia, hypercarbia, hypokalaemia, hypomagnesaemia, hypophosphataemia). TPN must not be stopped suddenly,

as rebound hypoglycaemia may occur. It should be either:

- Continued at the same rate, controlling hyperglycaemia perioperatively as in the diabetic patient
- Weaned to half the maintenance rate over 12 h preoperatively
- Replaced by 10% glucose infused at the same rate (in unstable patients)

Blood transfusion for anaemia and correction of clotting abnormalities may be required. Controversy exists over the routine use of albumen solutions.

PERIOPERATIVE MANAGEMENT

Most drugs should be dosed on actual body weight. There is an increased sensitivity to:

- Intravenous induction agents
- Suxamethonium in severe malnutrition (albumin <2.0 g dL^{-1}), since pseudocholinesterase deficiency may exist
- Nondepolarising neuromuscular blockers in the presence of hypocalcaemia, hypophosphataemia and hypomagnesaemia
- Drugs bound to albumin, e.g. diazepam
- Drugs bound to skeletal muscle, e.g. digoxin

Routine monitoring is used and a low threshold for invasive monitoring is adopted. Blood sugar monitoring should occur hourly.

The malnourished heart functions at the peak of the Starling curve, and so cardiac output may fall with increased diastolic filling, precipitating cardiac failure. Judicious fluid management with cardiac output monitoring may be required.

POSTOPERATIVE MANAGEMENT

- Transfer to ITU/HDU for patients with severely reduced cardiorespiratory reserve
- Mechanical ventilation in the case of
  - Fatigue
  - Increased CO_2 production due to glucose feed
  - Impaired response to hypoxaemia

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- Supplemental oxygen on the ward
- Physiotherapy
- Analgesia to allow an effective cough
- Restart nutritional support slowly over 12–24 h postoperatively
- Monitor blood glucose and serum potassium and avoid hypophosphataemia
- Early dietetic involvement

OUTCOME

Several studies have shown an increased incidence of postoperative complications in the malnourished patient. The role of pre- and postoperative nutritional support in the malnourished patient is poorly defined. Both reduce postoperative complications and improve pulmonary function in patients with fistulae, short bowel syndrome, burns and acute renal failure, and may have a place in patients who have lost more than 20% of their usual body weight. For the maximum nutritional benefit, feeding (whether enteral or parenteral) should be started 1 week to 10 days preoperatively.

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OBESITY

Obese patients frequently present for urgent and elective surgery. Obesity is a chronic nutritional disorder associated with hypertension, cardiovascular and respiratory disease, diabetes, liver cirrhosis and hiatus hernia. In addition to these associated medical conditions, obese patients present multiple logistical challenges. While medical morbidity is correlated with weight, few prospective studies have been performed relating obesity and anaesthetic outcome.

Obesity may be defined on the basis of ideal body weight, or body mass index (see Table 4.1).

PATHOPHYSIOLOGY

CARDIOVASCULAR SYSTEM

In the absence of ischaemic heart disease, obesity raises end-diastolic ventricular volume, increases stroke volume, and thereby increases cardiac output. Left ventricular work rises, in part due to the increased systemic vascular resistance, and is compensated for by biventricular hypertrophy. Filling pressure and cardiac output rise promptly with exercise, which includes moving body position in

Table 4.1 Calculation of ideal body weight and body mass index

Calculation of ideal body weight (IBW, Broca's index, kg)	Calculation of Body Mass Index (BMI, kg M^{-2})
For men: IBW (kg) – Height (cm) – 100	BMI = weight (kg)/(height in metres)^2
For women: IBW (kg) – Height (cm) – 105	
	Underweight: BMI <18.5 kg M^{-2}
Obese: Weight greater than 30% of ideal	Normal: BMI 18.5–24.9 kg M^{-2}
Morbidly obese: Weight greater than 100% of ideal	Overweight: BMI 25–29.9 kg M^{-2}
	Obese 1: BMI 30–34.9 kg M^{-2}
	Obese 2: BMI 35–39.9 kg M^{-2}
	Obese 3: BMI >40 kg M^{-2}

the morbidly obese. Right-sided failure occurs with sleep apnoea.

RESPIRATORY SYSTEM

Obesity acts to impose a load on the chest wall, such that the work of breathing is raised, although lung compliance, in the absence of coexisting disease, is normal. Lung volumes are reduced and fall further when supine. In the morbidly obese, tidal breathing falls within the closing volume range. Airway closure can cause wheeze which is frequently attributed to asthma. Arterial hypoxaemia is common, and increases pulmonary vascular resistance. Eight percent of morbidly obese patients developed a blunted response to carbon dioxide; this is obesity hypoventilation syndrome.

GUT

Both gastric acid and fasting gastric volume are raised in the obese. Abdominal pressure increases linearly with weight gain, and the incidence of hiatus hernia is high.

ENDOCRINE SYSTEM

Glucose tolerance in the obese is impaired; diabetes is common.

COAGULATION

Laboratory evidence of hypercoagulability is slight, although some clinical reports suggest that the incidence of deep venous thrombosis (DVT) is raised.

PRACTICAL CONSIDERATIONS

The obese patient will require special equipment which can accommodate the altered body dimensions and width. Manual handling requires more staff. Noninvasive blood pressure cuffs often do not work. Vascular access, invasive monitoring, neuraxial and regional techniques are all challenging when the usual landmarks are neither visible nor palpable.

Operative times are often prolonged and procedures technically challenging as the surgical team face similar challenges.

PREOPERATIVE ASSESSMENT

MORPHOLOGICAL

- Assessment of veins and arteries
- Airway – mouth, jaw, dentition, neck
- Back for neuraxial technique if considered: identification of midline, Tuffier's line, spaces
- Transportation to theatre

HISTORY

Take a systematic review of associated cardiovascular, respiratory and endocrine disease; focus upon functional capacity. Specifically ask about obesity hypoventilation syndrome, which by day is characterised with somnolence and poor concentration, and by night, by respiratory obstruction, nightmares and restlessness.

INVESTIGATIONS

- *ECG* – Ventricular hypertrophy, ischaemia, pulmonary hypertension
- *Echocardiography* – Right or left chamber enlargement, regional wall abnormalities
- *Spirometry* – Flow limitation, reduced vital capacity, reduced FRC
- *Blood gas analysis* – Hypoxaemia when standing

PERIOPERATIVE MANAGEMENT

Inform the theatre team that the anaesthetic time will take over twice the usual time. Ensure additional help is available.

PREMEDICATION

Sedative premedication should be avoided where possible. Acid aspiration prophylaxis should be considered.

MONITORING

This should commence in the anaesthetic room with the patient awake. Standard monitoring should be used, with the following caveats.

- 5-lead ECG should be used to detect ischemia; waves will be small.
- Noninvasive blood pressure performs poorly – if an automated reading fails while the patient is awake, there should be a low threshold to place intra-arterial monitoring.

VASCULAR ACCESS

Peripheral veins are often difficult to see or palpate. There should be a low threshold to use ultrasound. Similarly, intra-osseous devices and central lines are challenging to place.

AIRWAY

Obese patients deoxygenate three to five times quicker than nonobese controls due to high oxygen consumption and a reduced functional residual capacity (FRC). Both face-mask ventilation and intubation may be difficult. There is an increased incidence of difficult intubation in obese patients. Controlled ventilation through a tracheal tube should be used for all procedures where general anaesthesia is planned due to the increased risk of aspiration.

The airway management plan should be based upon the airway assessment. Prior to induction of anaesthesia, the patient should be carefully positioned in a ramped position: this will increase FRC and provide optimal conditions for airway management.

CHOICE OF ANAESTHETIC AGENTS

Drug dosing should in general be based on lean body weight. In general short acting, water soluble drugs should be chosen over long acting, lipid soluble agents to allow for a quick wake up. While nitrous oxide offers a theoretical advantage for some surgeries, its practical utility may be limited by the need for high inspired oxygen tensions.

MECHANICAL VENTILATION

Oxygenation may be impaired despite high inspired oxygen tensions and PEEP. A head-up tilt will reduce the abdominal pressure on the diaphragm.

CARDIOVASCULAR SEQUELA

Cardiovascular depression is likely during general anaesthesia, leading to rises in filling pressure and declining cardiac output. Aortocaval compression may occur in the supine posture, increasing systemic vascular resistance and decreasing venous return. Left ventricular function has been shown to continue to decline in the postoperative phase as compared with the nonobese.

REGIONAL ANAESTHESIA

Although this may be technically difficult if the bony landmarks are obscured, subarachnoid and epidural anaesthesia have been advocated in the morbidly obese. Standard length needles may be too short. Dose requirements are generally reduced compared to those in the nonobese (75%–80%). Cardiovascular decompensation has been reported in cases where the sympathetic block was variably higher than the somatic blockade. Motor blockade of the respiratory muscles limits the height of blockade to T7. Peripheral nerve blockade is likewise hampered by poor sono-anatomy.

POSTOPERATIVE CARE

- Wake in the sitting position; remove the endotracheal tube only when the patient is fully awake.
- Do not leave morbidly obese patients lying supine.
- Continuous oxygen therapy with a low threshold to use CPAP.
- Humidification.
- Physiotherapy and early mobilization.
- Analgesia avoiding respiratory depression.
- Deep venous thrombosis therapy.
- Critical care is often required for the first few days postoperatively.

OUTCOME

Although epidemiological studies have shown obesity to correlate with mortality, this is not the case with anaesthetic mortality. From the limited literature available, similar mortality has been recorded for hysterectomy (1%) and gastric bypass (1.2%) when these results have been matched to nonobese controls. This is attributable to improvements in anaesthetic technique.

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CROSS-REFERENCES

PREVIOUS LIVER TRANSPLANT

Orthotopic liver transplant (OLT) has become recognised as a therapeutic procedure for end-stage liver disease. Improvements in organ preservation, surgical and anaesthetic techniques and immunosuppression have led to an increase in the number of centres and in the number of transplants and patient survival. As a result, recipients will increasingly present to nontransplant centers for nontransplant related surgery. The late complications of transplantation, such as transplant biliary leak and duct, will usually be dealt with at a primary transplant centre and are not further considered here.

PREOPERATIVE ASSESSMENT

The majority of recipients is 30–60 years old and enjoy good health. The cardiopulmonary effects of end-stage liver disease reverse shortly after transplantation. Most have normal liver function unless there is rejection, sepsis or recurrence of original disease. There is a high incidence of hypertension reported after OLT. Immunosuppression increases susceptibility to infection.

At the preoperative consultation:

- Systemic review for intercurrent or chronic problems and any evidence of infection.
- Reason for transplantation, timing of surgery and health following transplant.
- Formal clinical and biochemical assessment of liver function (chronic rejection, recurrent disease, biliary obstruction).
- Enquire which immunosuppressive agents are used and assess for common side effects; all cause an increased risk of infection:
  - *Corticosteroids* – Hypertension, hypokalaemia, hyperglycaemia, adrenal suppression
  - *Tacrolimus* – Hypertension, renal dysfunction, electrolyte abnormalities and hyperglycaemia
  - *Cyclosporin* – Nephrotoxicity with hyperkalaemia, hepatotoxicity, neurotoxicity, hypertension
  - *Azathioprine* – Bone marrow suppression
- Liaise with transplant centre, ask for advice regarding management of immunosuppression.

INVESTIGATIONS

- FBC: Hb, white blood cells
- U&E: electrolyte abnormalities common with most immunosuppressants
- Liver function tests
- Coagulation: PT, APTT, platelets ± bleeding time
- Chest X-ray
- ECG

PERIOPERATIVE MANAGEMENT

Management is similar to that of a patient with liver impairment. The aim is to avoid any deterioration or compromise in liver function, by optimizing hepatic oxygenation and blood flow. Maintain normoxia, normocarbia and pH and normovolaemia at all times. Direct arterial monitoring is helpful for all but the shortest of procedures.

Be aware of increased infection risk due to immunosuppression: all invasive monitoring must be inserted with 'no touch' aseptic technique.

ANAESTHESIA

- Isoflurane or desflurane are the agents of choice: minimal metabolism and best preservation of hepatic arterial and mesenteric flow
- Atracurium for neuromuscular relaxation.
- Maintain normocapnia and normal acid–base status to minimize effects on liver blood flow.
- Analgesia: epidural ideal for postoperative analgesia (N.B. check coagulation). If liver function deranged, fentanyl is the safest choice intraoperatively.

POSTOPERATIVE MANAGEMENT

For major procedures, these patients will require HDU care for a minimum of 24 h.

- Postoperative analgesia:
  - Epidural opiates/low-dose bupivicaine infusion
  - Intravenous opiate infusion/patient-controlled analgesia pumps
- Continue to ensure good oxygenation and optimize haemodynamics and volume status
- Renal-dose dopamine for 24 h postoperatively
- Antibiotic prophylaxis
- Continue immunosuppression and steroid cover

OUTCOME

Increasing numbers of patients have successful liver transplants and may present months to years later with unrelated surgical problems. Careful preoperative assessment is essential, especially in relation to liver and kidney function. Perioperative management is directed to avoiding any factors that might compromise hepatic and renal function and minimizing the infection risk with antibiotic prophylaxis and careful aseptic techniques.

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CROSS-REFERENCE

THE FULL STOMACH

General anaesthesia abolishes the upper airway reflexes that prevent pulmonary aspiration of active or passively regurgitated gastric contents in health. Aspiration of solid material can cause a mechanical obstruction, with subsequent lung collapse and ensuing pneumonia or abscess formation. Aspiration of over 25 mL of liquid of pH less than 2.5 can cause bronchospasm, pneumonitis, bronchopneumonia and acute respiratory distress syndrome.

Strategies to reduce risk of pulmonary aspiration revolve around minimising residual gastric volumes and rapidly securing the anaesthetised airway. Fasting is routinely used to reduce residual gastric volumes. In general, for a normal healthy patient undergoing elective surgery, stomach emptying is complete within 6 hours after food and milky drinks, 4 hours after breast milk and 2 hours after water. Excessive fasting times are unpleasant for patients and are associated with medical morbidity and modern practice seeks to minimise fasting times. The preoperative assessment allows for opportunity to risk-assess the likelihood of altered gastric transit times.

PATHOPHYSIOLOGY

Both the active process of vomiting and the passive process of regurgitation of gastric contents are more hazardous in a patient with a full stomach. A full stomach and any reduction in the functional integrity of the lower oesophageal sphincter (LOS) predispose a patient to regurgitation. In prolonged intestinal obstruction, faeculent material may retrogradely enter the stomach. The rate of emptying of the stomach after oral intake varies according to stomach contents and the context of the patient. No patient can ever be assumed to have a completely empty stomach.

FACTORS DELAYING GASTRIC EMPTYING

- Mechanical obstruction of the gastrointestinal tract
- Ileus
- Following surgical manipulation of the bowel (postoperative)
- Recent trauma
- Electrolyte derangement
- Peritonitis
- Pain
- Fear and anxiety
- Third trimester of pregnancy
- Drugs

THE LOWER OESOPHAGEAL SPHINCTER

The LOS is an area at the gastro-oesophageal: it is an increased pressure zone caused by the pinch-cock action of the diaphragm; it is not an anatomically distinct area. Heartburn and acid-brash are symptoms suggestive of an incompetent lower oesophageal sphincter and place the patient in a higher risk category for passive regurgitation on the induction of anaesthesia. The sphincter is nonfunctional when a hiatus hernia is present.

PREOPERATIVE ASSESSMENT

HISTORY

- Last oral intake of food and drink, especially alcohol
- Possibility of swallowed blood

- Factors known to delay gastric emptying (above)
- History of reflux, heartburn or hiatus hernia
- Drugs known to reduce LOS tone
    - Alcohol, opiates
    - Anticholinergics
    - Tricyclics
    - Dopamine
    - Beta agonists

Emergency patients are more likely to have a full stomach as:

- Presenting pathology causes a mechanical obstruction, e.g. laparotomy for small bowel obstruction
- Surgery is urgent and cannot wait for the full fasting time
- The surgical pathology results in pain and anxiety

PREPARATION

Where possible surgery should be delayed to allow time for the stomach to empty. Various strategies can be used to neutralise and reduce gastric contents:

- Aspirate stomach contents through a nasogastric tube, particularly for patients with an acute abdomen.
- Increasing gastric motility with pharmacological agents such as metoclopramide.
- Neutralise gastric contents using a nonparticulate antacid such as sodium citrate. This offers a short window of protection and should be given immediately prior to induction.
- Administration of H_2 blocking drugs or a proton pump inhibitor; these drugs take hours to work.

PERIOPERATIVE MANAGEMENT

PREMEDICATION

Avoid opiates and anticholinergics – both reduce LOS tone and delay gastric emptying.

ANAESTHETIC MANAGEMENT

Management of the induction of anaesthesia depends on the cause of the full stomach and clinical

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circumstances. Intubation of the trachea with a cuffed endotracheal tube is mandatory. In most cases, a rapid sequence induction with full preoxygenation, cricoid pressure and a head-up position is preferred. In the case of blood in the stomach where bleeding into the airway is responsible (e.g. post-tonsillectomy bleeding) an inhalation induction with the patient in the lateral position and tilted head-down has been recommended.

Emergence from anaesthesia carries the same potential hazards as induction and the patient should be placed in the lateral position before anaesthesia is terminated. The trachea should be extubated only on return of protective airway reflexes.

POSTOPERATIVE MANAGEMENT

- Risk continues until larynx is competent.
- Gastric emptying is delayed by pain and opiates.
- Maintain lateral position.
- Avoid sedative agents.

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CROSS-REFERENCES

THE JAUNDICED PATIENT

Jaundice is a yellowing of the skin and sclera that results from increased amounts of bilirubin in the body. Surgery in this group carries a poor prognosis and should be avoided, being limited to emergency procedures only.

Jaundice is classically divided into prehepatic, hepatocellular and obstructive causes. Prehepatic jaundice results from an increased breakdown of haem-containing molecules; it is not considered

further in this section. Hepatocellular jaundice may be caused by toxins, viruses and drugs. Obstruction can be caused by calculus, stricture and cancer.

It is useful to understand the time course of the underlying liver disease. The accepted cut off between acute and chronic liver disease sits at 26 weeks. Acute liver disease may progress to chronic liver disease, or may exist as a self-limiting episode. Jaundice and the associated liver failure occur both in acute liver failure, acute-on-chronic liver failure and end-stage chronic liver disease.

This section focusses upon acute disease; the following section is devoted to chronic disease. The reader should appreciate individual patients present along the spectrum.

ASSOCIATED PROBLEMS

ACUTE OLIGURIC RENAL FAILURE

This occurs in up to 17% of all jaundiced patients undergoing surgery, with a mortality of up to 50%; 75% will have a fall in glomerular filtration rate postoperatively. Factors implicated include:

- Hypovolaemia and hypotension
- The presence of bile salts
- Bilirubin
- Endotoxins

Glomerular and peritubular fibrin deposition has been demonstrated in affected kidneys. Hepatorenal syndrome may occur associated with deterioration of hepatic function.

COAGULOPATHY

The vitamin K dependant coagulation factors (II, VII, IX and X) are reduced and this is manifest as a prolonged prothrombin time. Hepatocellular coagulopathy is often refractory to vitamin K administration. Disseminated intravascular coagulation is associated with secondary biliary tract infection, and increases mortality.

ALTERED DRUG HANDLING

Drugs excreted via the biliary system have prolonged elimination half-life in cholestasis. Increased volume

of distribution and reduced clearance produces initial pancuronium resistance; repeated dosing is associated with prolongation of action. Atracurium is the drug of choice for muscular relaxation. Narcotics may produce spasm in the sphincter of Oddi (biliary colic, and difficulty with cholangiography).

Plasma cholinesterase has a very long half-life, and suxamethonium apnoea is not a feature, even in fulminant liver failure although the duration of a dose of suxamethonium may be longer than expected.

GASTROINTESTINAL TRACT

Stress ulceration occurs, with gastrointestinal haemorrhage demonstrated in 16% of cases.

WOUND HEALING

This is significantly reduced, and correlates closely with degree of malnutrition and the presence of sepsis and malignancy.

PREOPERATIVE ASSESSMENT

HISTORY AND CLINICAL EXAMINATION

Look for malnutrition, malignancy, anaemia, dehydration, jaundice, pyrexia, signs of drug abuse and concomitant diseases.

INVESTIGATIONS

- Haemoglobin – At presentation and current
- White cell count – Cholangitis, isolates and sensitivities
- Platelet count – Reduced with severe infection and DIC
- Clotting screen – Note effect of vitamin K administration
- Urea, electrolytes, creatinine, glucose – Creatinine clearance if poor or deteriorating renal function
- Serum bilirubin (beware if >200 mmol l^{-1})
- Serum albumin, calcium and magnesium
- Liver function tests – Serum transaminases are raised in hepatocellular dysfunction, whereas alkaline phosphatase and Gamma-glutamyl transpeptidase are raised in obstructive disease
- Blood gases – Respiratory alkalosis and hypoxaemia
- Serology – Consider infective risks to staff
- Biopsy – Pattern and degree of damage
- Ultrasound, CT scan or MR scan to visualize the biliary and pancreatic ducts

PREOPERATIVE PREPARATION

Depends on severity of disease. Commonly required preparation involves:

- Rehydration
- Appropriate cross-match and preoperative transfusion
- Perioperative antibiotic administration
- Administer vitamin K
- If prothrombin time remains abnormal, arrange for a plasma transfusion
- Either H$_2$ antagonists or proton pump inhibitors
- Urinary catheter early
- Optimization of concurrent disease
- Percutaneous drainage (symptoms improve, prognosis unaltered)

PREMEDICATION

Premedication is rarely required. Avoid sedatives in encephalopathy. Continue H$_2$ antagonist and vitamin K.

PERIOPERATIVE MANAGEMENT

A reduction in hepatic blood flow during the intraoperative period is associated with poor prognosis and total hepatic necrosis can occur. Renal insult must be avoided. Stable anaesthesia which avoids hypotension and preserves cardiac output with fluid loading is essential. The actual drugs used are less important than the physiological goals.

Altered drug metabolism depends upon high and low extraction ratio and is therefore difficult to predict. Acid–base disturbance may occur; alterations in electrolytes may contribute to encephalopathy and should be monitored. Hypoglycaemia and lactic

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acidosis may occur (with severe injury); monitor and treat aggressively.

Hypothermia worsens coagulopathy. Therefore, warm fluids, humidify respiratory gases, use warming blanket and/or warm air over-blanket and reduce body surface heat losses.

In all cases a low threshold for invasive monitoring is to be recommended; in the presence of hepatocellular disease it is essential. Blood gas analysis (including electrolytes, lactate and glucose) should be conducted at least hourly. Temperature, urine output and estimated blood loss should all be monitored. An oesophageal Doppler may be of value.

POSTOPERATIVE MANAGEMENT

Transfer to a high dependency area is advisable for the first 24–48 hours. Hypoxaemia is common. Drain losses should be aggressively replaced; hepatocellular disease may require additional clotting factors. Monitoring with thromboelastography is helpful. Replace urine losses appropriately. Continue dopamine until cardiovascularly stable. Catecholamines may reduce hepatic and renal blood flow. Epidural analgesia can be considered in the absence of coagulopathy. Intramuscular opiate administration is inappropriate in all but minor cases.

OUTCOME

Relates to severity of disease. Minor worsening of liver function tests is not uncommon; morphological change is. The following may worsen jaundice postoperatively:

Hepatocellular damage

- Postoperative cholestasis
- Circulatory failure
- Drug induced
- Exacerbated chronic disease

Extrahepatic obstruction

- Duct stone
- Bile duct injury
- Postoperative pancreatitis

Laparotomy in the presence of hepatocellular disease has been associated with perioperative mortality of 9.5% and morbidity of 12%. Approximately 25% of jaundiced patients undergoing surgery for relief of biliary obstruction have subsequently been demonstrated to have hepatocellular disease. A combination of anaemia at presentation, serum bilirubin >200 mmol L^{-1} and the presence of malignancy carries a mortality of 60%.

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CROSS-REFERENCES

Acute renal failure, Chapter 5
Open cholecystectomy, Chapter 10
Chronic liver disease, Chapter 4

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Genitourinary tract

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BRIAN J POLLARD AND GARETH KITCHEN

ACUTE KIDNEY INJURY

Acute kidney injury is an acute decline in renal function sufficient to result in the retention of nitrogenous end-products of metabolism. It was previously termed 'acute renal failure' and is usually marked by an increase in serum creatinine concentration or blood urea nitrogen (BUN) concentration. Patients are not necessarily oliguric.

In 2004, the Acute Dialysis Quality Initiative work group published a consensus definition and classification system for acute kidney injury (AKI) – the RIFLE classification: **R**isk of renal dysfunction, **I**njury to the kidney, **F**ailure or **L**oss of kidney function, and **E**nd-stage kidney disease (Figure 5.1). It defines three grades of severity of AKI (risk, injury, failure) based on changes to serum creatinine concentration, urine output and defines two clinical outcomes (loss, end-stage). The RIFLE criteria have clinical relevance for the diagnosis, classification and monitoring of progression of AKI, with modest predictive value for mortality.

GFR is commonly estimated using the Cockcroft-Gault (C-G) and MDRD (Modification of Diet in Renal Disease) study equations.

CAUSES

- *Pre-renal* – Inadequate perfusion – 40%–70%
- *Renal* – Intrinsic renal disease – 10%–50%
- *Post-renal* – Obstructive uropathy – 10%

The most common situation is the development of acute tubular necrosis due to ischaemia, or occasionally, renal toxins. Necrosis is not the sole form of cell death in AKI – tubular cell apoptosis (principally in the distal nephron) is an important factor in sepsis-related AKI.

ACUTE TUBULAR NECROSIS (ATN)

CAUSES

- Hypoperfusion
  - Decreased intravascular volume
  - Sepsis
  - Cardiogenic shock or tamponade

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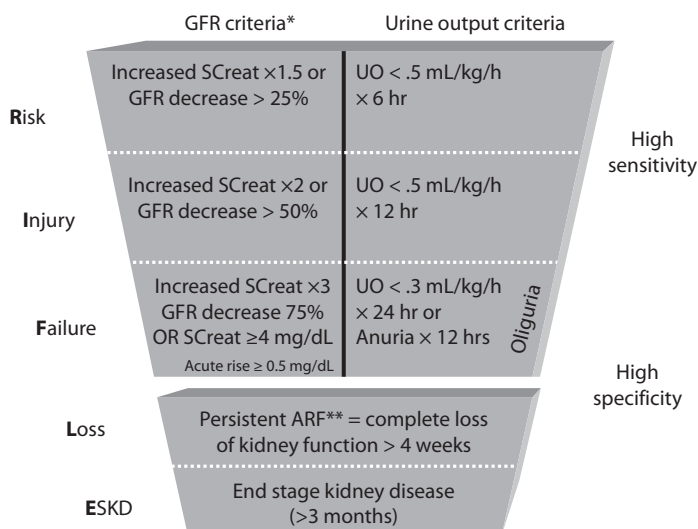
Figure 5.1 RIFLE classification of AKI. The classification system includes separate criteria for creatinine and urine output (UO). A patient can fulfil the criteria through changes in serum creatinine (S_{creat}), changes in UO, or both. (*GFR = glomerular filtration rate, **ARF = acute renal failure). (Reproduced with permission. *Crit Care*. 2004; **8**(4): R204–R212. Published online May 24, 2004. doi: 10.1186/cc2872. Copyright ©2004 Bellomo et al.)

- Embolic occlusion of the renal arteries
- Severe hypoxia
- Drugs
  - Aminoglycosides
  - Amphotericin B
  - Radiocontrast agents
  - Non-steroidal anti-inflammatory drugs
  - Frusemide
- Endogenous toxins
  - Free haemoglobin after a transfusion reaction
  - Myoglobin from rhabdomyolysis
  - Abnormal reaction to succinylcholine
  - Myeloma renal damage
  - Crystals (urate, oxalate)
  - Hypercalcaemia

BOX 5.1: Conditions associated with increased risk of perioperative acute kidney injury

- Preoperative renal disease
- Diabetes mellitus
- Hypertension
- Preoperative 'shock' states
- Cirrhosis
- Biliary obstruction
- Sepsis
- Multiple system trauma
- Multiple organ failure
- Cardiac failure
- Extracellular fluid volume deficit
- Elderly patients
- Aortorenal vascular disease

PREVENTION OF ATN

PREOPERATIVE ASSESSMENT

Elicit detailed information on patient factors known to be associated with increased risk for AKI (Box 5.1) and identify modifiable factors.

PERIOPERATIVE MANAGEMENT

Prevention of new AKI strategies:
- Maintain oxygenation
- Maintain normocarbia

- Maintain renal perfusion pressure
- Optimize intravascular volume and cardiac output
- There is no definitive evidence that any pharmacological agent can prevent or treat AKI
  - *Furosemide* – Little or no evidence to support its use as a renoprotective agent but may assist in managing volume overload
  - *Dopamine* – Increases renal blood flow but offers no beneficial effect on renal outcome
  - *Mannitol* – May have renoprotective effect in renal transplantation but no evidence to support its role otherwise in preventing AKI.
  - *Atrial natiuretic peptide* – May be associated with improved outcomes when used in low doses for preventing AKI and in managing postsurgery AKI

PROGNOSIS

Perioperative AKI occurs in approximately 1% of patients undergoing general surgery procedures and its development is associated with an eightfold increase in 30-day mortality. AKI is an independent risk factor for hospital mortality.

ESTABLISHED AKI

PREOPERATIVE ASSESSMENT

Pay particular attention to previous renal disease, infection, stones or prostatism. Include a thorough assessment of intravascular volume status. Insert a urinary catheter in all but the most minor procedures. Consider central venous pressure or more invasive volume monitoring.

INVESTIGATIONS

- Serum urea, creatinine and electrolytes
- Ratio of urine/blood osmolality – if over 1.5:1 suggests hypovolaemia
- Urine specific gravity and urea – intrinsic renal failure leads to a fixed specific gravity

of 1010 and a urea concentration of <600 mg mL^{-1}. Urinary SG >1015 and urinary urea concentration >2 g/100 mL are consistent with intravascular hypovolaemia

PERIOPERATIVE MANAGEMENT

- Avoid drugs that require renal function for elimination
- Maintain renal perfusion pressure
- Monitor any urine output

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CROSS-REFERENCES

Assessment of renal function, Chapter 5
Urological surgery – general principles, Chapter 13
Preoperative assessment – specific medical problems, Chapter 25

ASSESSMENT OF RENAL FUNCTION

Why is assessment of renal function important?

1. Preoperative renal dysfunction is associated with greater risk of postoperative complications.
2. Certain well-defined risk factors for new AKI or deterioration in renal function are detectable preoperatively (Box 5.2).

BASIC FUNCTIONS OF THE KIDNEY

- *Glomerulus* – Responsible for filtration and subsequent excretion of nitrogenous wastes.
- *Tubules* – Selective reabsorption or secretion regulating the movement of water and ions to maintain fluid balance and the excretion or reabsorption of hydrogen ions to maintain acid–base homeostasis.
- *Endocrine* – The production of renin which is involved in water and electrolyte homeostasis, the release of prostaglandins and the activation of both erythropoietin and vitamin D.

Perioperative AKI accounts for 50% of all patients requiring dialysis and is associated with a mortality incidence of 14%–19% overall and 28%–69% in patients requiring renal replacement therapy. Preoperatively

BOX 5.2: Factors affecting renal function

- ■ Intrinsic renal disease
- ■ Extra-renal factors
    - – Intra- and extra-vascular fluid status
    - – Cardiovascular function
    - – Neuroendocrine factors

Table 5.1 Normal values for 70 kg male

Cardiac output	5000 mL min^{-1}
Renal blood flow	1250 mL min^{-1}
Renal plasma flow	750 mL min^{-1}
Glomerular filtration rate	125 mL min^{-1}
Urine flow	2 mL min^{-1}

it is important to identify patients at risk, and to take measures to protect them from developing postoperative renal complications. There is no single comprehensive test of renal function. Assessment of renal function comprises history, examination, laboratory tests, investigations, e.g. ECG chest X-ray and echocardiogram. The normal values for a 70 kg male are given in Table 5.1.

HISTORY AND EXAMINATION

SYMPTOMS

In the early stages of renal disease there may be no symptoms. The loss of function in chronic kidney disease usually takes months or years to occur and may be asymptomatic until kidney function is less than 10% of normal. When symptoms do occur, they include:

- Fatigue, general malaise, headaches
- Nausea, weight loss, loss of appetite
- Pruritus, dry skin
- Polyuria
- Polydypsia
- Dysuria

SIGNS

- Hypertension of long-standing
- Hypervolaemia (e.g. oedema, dyspnoea)
- Hypovolaemia (e.g. decreased skin turgor, tachycardia, hypotension)

MEDICATION

- Diuretics
- Potassium supplements
- Immunosuppressive agents

- Antihypertensive therapy
- Dialysis schedule

INVESTIGATIONS

PLASMA

Electrolytes

Sodium, potassium, chloride and bicarbonate remain normal until advanced disease, when a hyperkalaemic, hyperchloraemic acidosis develops. These changes will exacerbate dysrhythmias and compromise resuscitation. Frank renal failure results in hypocalcaemia, hyperphosphataemia and hypermagnesaemia.

Urea and nitrogen

Urea is produced by protein catabolism in the liver. It is filtered and reabsorbed by the kidney. The amount reabsorbed varies with the state of hydration. Thirty-three percent is reabsorbed when the urine flow is <2 mL/min. Creatinine is a by-product of muscle metabolism and production is related to muscle mass. It is filtered and excreted by the kidney. Serum creatinine is an insensitive indicator of renal function – the value may be normal even if GFR is reduced by 50%. This is due to a combination of increased extrarenal metabolism and secretion of creatinine by the renal tubules. It is difficult to assess renal function using serum creatinine as it overestimates GFR (Box 5.3).

URINE

As a sole investigation, urinalysis is normally sufficient screening in patients with no history of renal or systemic disease.

APPEARANCE

- *Gross* – bleeding, infection
- *Microscopic* – casts, bacteria, cell forms

pH

Normally urine is acidic. Therefore, acidification is a measure of function.

BOX 5.3: Nonrenal variables affecting urea and creatinine levels

Increased nitrogen absorption
Increased nitrogen waste production, e.g. sepsis, trauma
Diet
Body mass
Activity
Hepatic disease
Diabetic ketoacidosis
Large haematoma
Gastrointestinal bleeding
Drugs (steroids, cimetidine)

SPECIFIC GRAVITY (SG)

SG refers to the concentration of solutes in urine; the ability to concentrate is a measure of tubular function. This is, however, nonspecific (Box 5.4).

OSMOLALITY

Osmolality is the number of osmotically active particles per unit solvent (units: mOsm/L).

Osmolality is more specific than SG, and is helpful at extreme values:

- Oliguria + Osmolality > 500 suggests prerenal azotaemia
- Oliguria + osmolality < 350 likely to be acute tubular necrosis

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BOX 5.4: Substances/ conditions affecting SG

- Protein
- Glucose
- Mannitol
- Diuretics
- Extremes of age
- Antibiotics (carbenicillin)
- Temperature
- Hormonal imbalance (pituitary, adrenal and thyroid disease)

Oliguria itself also affects the osmolality value. Osmolality is only useful in low urine output states coupled with a low SG. An osmolality of <350 mOsm kg^{-1} suggests an inability to concentrate urine and excrete electrolytes.

PROTEIN

- <150 mg/24 h: normal excretion (exercise and standing can increase this)
- >750 mg/24 h: indicator of renal parenchymal disease
- Massive: glomerular damage

GLUCOSE

Freely filtered and reabsorbed. Glycosuria occurs when an abnormally heavy load is presented to the tubules (e.g. diabetes mellitus, intravenous glucose).

CREATININE CLEARANCE

Measures the glomerular filtration of creatinine (Cr) which approximates (although overestimates) GFR.

$$\text{Cr clearance} = \frac{\text{Urine Cr} \times \text{Urine volume}}{\text{Plasma Cr}}$$

The Cockroft-Gault formula for creatinine clearance:

$$\text{Cr clearance} = \frac{(140 - \text{Age}) \times \text{Lean body weight (kg)}}{72 \times \text{Plasma Cr (mg/dL)}}$$

Note: multiply value by 0.85 for females.

This approximation overestimates GFR by 10%–20% due to creatinine secretion by peritubular capillaries.

GFR is estimated from equations using serum creatinine, age, race, gender and body size. One such equation is the Modification of Diet in Renal Disease (MDRD) Study equation.

A newer equation had been described to estimate GFR by the Chronic Kidney Disease Epidemiology Collaboration. The CKD-EPI is more accurate than the MDRD Study equation overall and across most subgroups.

BIOMARKERS

The diagnosis of AKI has to date depended on detection of a decrease in kidney function by an increase in serum creatinine concentration which only occurs after a significant decrease in renal function. Earlier detection of AKI would be beneficial. A number of early biomarkers of AKI are currently being investigated. Neutrophil gelatinase-associated lipocalin (NGAL), a 25 kDa protein that is bound to neutrophils and expressed in injured epithelial cells in organs including the kidney has emerged as an accurate early biomarker of acute kidney injury. Plasma and urine NGAL have proved sensitive, specific and predictive early biomarkers of AKI after cardiac surgery. Other promising biomarkers are cystatin C, interleukin-18 and kidney injury molecule-1 (KIM-1). Further studies are required to validate the sensitivity and specificity of these biomarkers in clinical samples from large cohorts and from multiple clinical situations. Clinically relevant urinary biomarkers are summarized in Box 5.5.

HAEMATOLOGY

- Established renal failure Hb = 3–9 g dL^{-1}
- White cell count may be abnormal if the patient is immunosuppressed
- Uraemia causes platelet dysfunction and impaired platelet-vessel wall interaction, which can lead to impairment of coagulation and increased perioperative blood loss. A prothrombotic state may also exist in these patients. Thromboelastographic indices in

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BOX 5.5: Clinically relevant urinary biomarkers

Cystatin C
N acetyl β D glucosaminidase
Interleukin-18
Kidney Injury Molecule 1
Neutrophil gelatinase-associated lipocalin

patients with CKD show that all aspects of coagulation are increased, including initial fibrin formation, fibrin-platelet interaction, and qualitative platelet function. There is also a reduction in fibrinolysis.

CHEST RADIOGRAPH

Look for signs of hypertensive cardiovascular disease, pericardial/pleural effusions and, rarely, uraemic pneumonitis.

ECG

- Hyperkalaemia
  - Tall peaked T-waves
  - ST depression
  - QRS widening
  - Ventricular dysrhythmias
- Hypocalcaemia
- Signs of hypertension
- Signs of ischaemic heart disease

ECHOCARDIOGRAPHY

In the presence of symptoms and signs of heart failure, left ventricular dysfunction should be evaluated. Preoperative left ventricular dysfunction has been shown to be a major risk factor for postoperative renal dysfunction and mortality.

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CROSS-REFERENCES

CHRONIC KIDNEY DISEASE

Chronic kidney disease (CKD) produces retention of nitrogenous waste products and inability to maintain fluid, electrolyte and acid–base homeostasis. The glomerular filtration rate (GFR) with units of mL/min/1.73 m² is usually used to stratify severity of CKD. CKD is then defined as either (1) a GFR <60 for 3 months or more, irrespective of cause, or (2) kidney damage leading to a decrease in GFR, present for 3 months or more. Decreasing GFR can also be used to define stages of chronic kidney disease. From a normal GFR >90, 60–89 would represent mild impairment, 30–59 moderate impairment, 15–29 severe impairment and <15 established renal failure. These changes are associated in a graded fashion with increased risk of death, cardiovascular events and hospitalization demonstrating its importance as a public health problem. The damage may manifest as abnormalities in the composition of blood or urine, on radiological imaging or in histology. Other evidence of CKD includes persistent microalbuminuria, proteinuria or haematuria (after exclusion of other causes), structural abnormalities of the kidneys (e.g. polycystic kidney disease, reflux nephropathy) or biopsy-proven chronic glomerulonephritis.

AETIOLOGY

- Diabetes mellitus
- Glomerulonephritis
- Pyelonephritis
- Renovascular disease
- Polycystic kidneys
- Hypertension
- Uncertain aetiology (glomerulonephritis unproven)
- Other (e.g. systemic lupus erythematosus, amyloidosis, gout, analgesic nephropathy, nephrocalcinosis)

PATHOPHYSIOLOGY

CKD is associated with multisystem dysfunction resulting from the primary disease process and/or the effects of uraemia.

BIOCHEMICAL

- Uraemia
- Increased serum creatinine (>180 mmol L^{-1})
- Electrolyte disturbance: hyperkalaemia, hyponatraemia, hyperphosphataemia, hypermagnesaemia, hypocalcaemia
- Metabolic acidosis

ASSOCIATED CARDIOVASCULAR ABNORMALITIES

- Hypertension
- Left ventricular hypertrophy
- Accelerated atherosclerosis
- Fluid overload (unless dialysed)
- Cardiac failure (secondary to hypertension and increased cardiac output)
- Pericarditis and/or pericardial effusion
- Peripheral vascular disease
- Stroke
- Hyperhomocystinaemia

RESPIRATORY

- Pulmonary oedema
- Pleural effusion

HAEMATOLOGICAL

- Anaemia (3–9 g dL^{-1})
- Early stages CKD – prothrombotic tendency
- End stage CKD – prothrombotic tendency and bleeding diathesis (partly due to platelet dysfunction)

IMMUNOLOGICAL

- Immunosuppression due to uraemia or drugs

NEUROLOGICAL

- Drowsiness, convulsions and coma (uraemia)
- Peripheral and autonomic neuropathies, especially if diabetic

GASTROINTESTINAL

- Autonomic neuropathy (delayed gastric emptying)
- Malnutrition
- Stress ulceration

SKELETAL

- Renal osteodystrophy

PREOPERATIVE ASSESSMENT

HISTORY

- Drug history
  - Immunosuppressives
  - Antihypertensives
  - Oral hypoglycaemics.
- Review of systems particularly regarding cardiovascular disease
- Presence of other medical conditions, e.g. diabetes mellitus
- Method and time of last dialysis as well as the biochemical and haemodynamic results

EXAMINATION

- Signs of fluid overload
- Body weight and dialysis related fluctuation

- Location of arteriovenous fistula site or potential sites

INVESTIGATIONS

- Full blood count (normochromic, normocytic anaemia)
- Clotting studies (including bleeding time)
- Full biochemical screen
- ECG
- Chest radiograph

PREMEDICATION

The effects of premedication with sedative drugs and opioids are unpredictable. Decreased tolerance is due to derangements in plasma proteins and the effect of altered blood pH on their pharmacokinetics. Consider administration of agents to reduce gastric acidity and volume. Antihypertensives should generally be continued in the perioperative period (with the possible exception of angiotensin-converting enzyme inhibitors). CKD is a risk factor for perioperative cardiovascular complications. The ACC/AHA guidelines recommend that the presence of one or more clinical risk factors, in patients having vascular or intermediate risk surgery, should prompt consideration of beta-blocker therapy.

PERIOPERATIVE MANAGEMENT

Avoid cannulating arteries or veins that might be used in the future to fashion arteriovenous fistulae. Central venous access may be indicated for monitoring fluid balance.

INDUCTION

The pharmocokinetics of propofol are unaltered by CKD although patients may require greater doses to achieve hypnosis than patients with normal renal function (1.42 [0.24] mg/kg versus 0.89 [0.2] mg/kg, respectively). Thiopental has an increased volume of distribution and decreased plasma protein binding resulting in an increase in free drug concentration so administer slowly and with caution.

Avoid suxamethonium if the serum potassium level is greater than 5 mmol L^{-1} or if the patient has a peripheral neuropathy. Atracurium, cisatracurium and mivacurium are the neuromuscular blocking agents of choice because of their rapid elimination and independence of renal metabolism and excretion. The duration of action of vecuronium and rocuronium are increased and they are best administered as a single dose rather than by infusion. Sugammadex is excreted unchanged in the urine but its efficacy as a reversal agent does not appear to rely on renal excretion of the cyclodextrin-relaxant complex. Sugammadex administered at reappearance of T_2 rapidly and effectively reverses NMB induced by rocuronium in renal failure and healthy patients. However, it is not currently recommended for use in CKD patients with creatinine clearance <30 mL/min or in those on dialysis.

MAINTENANCE

- CVP monitoring or trans-oesophageal Doppler may be a useful guide to volume status and systemic blood pressure.
- Urine output, if any, should be monitored.
- Brachial plexus block is suitable to facilitate fistula formation; however, balance the benefits against the risk of a bleeding tendency. Bupivacaine and lidocaine are safe but have a shorter duration of action in patients with CKD.
- Remifentanil does not accumulate in CKD and in patients on haemodialysis has a decreased clearance and prolonged elimination half-life.
- Isoflurane and desflurane are effective inhalational agents, with minimal metabolism. Enflurane use is controversial due to the potential accumulation of fluoride ions, usually <15 µmol L^{-1} (nephrotoxic concentration >50 µmol L^{-1}). Sevoflurane degrades to Compound A, which is nephrotoxic in rats although this does not seem to be a problem clinically when Amsorb is used.

ANALGESIA

- Paracetamol is safe and does not require dose adjustment. Avoid NSAIDs.
- Administer morphine and pethidine with caution, as their metabolites

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(morphine-6-glucuronide, norpethidine) may accumulate and can result in delayed onset of sedation and respiratory depression.

- Oxycodone undergoes hepatic metabolism to noroxycodone and oxymorphone which accumulate in patients with CKD. Therefore, reduce the dose and increase the dose interval.

POSTOPERATIVE MANAGEMENT

- Consider admission to HDU after major surgery or if significant comorbidities exist.
- Reevaluate fluid balance and hydration regularly using clinical assessment, CVP and urine output (if any).
- Give supplemental oxygen.
- Measure serum electrolytes regularly.
- Thromboembolic prophylaxis is important.
- CKD patients usually require further anaesthetics, including for fistula formation or dialysis.

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CROSS-REFERENCES

Preoperative assessment – specific medical conditions, Chapter 25
Upper limb blocks, Chapter 29
Assessment of renal function, Chapter 5

GOODPASTURE SYNDROME

'Goodpasture syndrome' originally described the entity of *pulmonary haemorrhage* and *glomerulonephritis.* Goodpasture disease is glomerulonephritis with pulmonary haemorrhage and the presence of circulating antiglomerular basement membrane (anti-GBM) antibodies. It therefore includes those diseases which are anti-GBM antibody negative as well as positive, such as:

- Polyarteritis nodosa
- Wegener's granulomatosis
- Primary crescentic glomerulonephritis
- Following treatment with penicillamine
- Systemic lupus erythematosus

Anti-GBM antibody disease describes patients with serum antibodies against the basement membrane and encompasses both Goodpasture syndrome and Goodpasture disease.

Anti-GBM and antineutrophil cytoplasmic autoantibodies (ANCA) can be assayed by immunofluorescence, permitting a more rapid and accurate diagnosis than was possible in the past.

PATHOPHYSIOLOGY

The pathogenetic mechanism involves the development of antibodies to pulmonary and glomerular

basement membranes, with an ensuing autoimmune process accounting for the renal lesions (crescentic glomerulonephritis), and pulmonary alveolitis (haemoptysis). Exposure to environmental factors such as viral infections, hydrocarbons and tobacco can precipitate the disease and worsen the pulmonary lesions.

Patients with specific HLA types are more susceptible and may have a worse prognosis. Patients with Goodpasture disease have an increased incidence of *HLA-DR2* compared to control populations. In addition, *HLA-B7* is found more frequently and is associated with more severe anti-GBM nephritis.

EPIDEMIOLOGY

- Annual incidence estimated 0.5–1 per million
- Bimodal distribution–more common in the third and sixth decades of life
- Male preponderance
- Most patients are Caucasian
- Genetic predisposition: HLA/DRA carriers in >80% of cases

PRESENTING FEATURES

- Pulmonary features appear early
  - Dyspnoea
  - Haemoptysis (rusty sputum to massive bleed) occurs in 80% of patients and is the most common presenting symptom
- Renal
  - Haematuria
  - Nephrotic picture
  - Oliguria/anuria
  - Hypertension

NATURAL HISTORY

Once respiratory symptoms develop, oliguria and anuria usually follow. The renal recovery rate at 1 year is 95% for patients with a creatinine concentration at presentation of <500 μmol/L. Risk factors for renal non-recovery are early oligo/anuria and requirement for haemodialysis: only 5% of patients who are dialysis-dependent at the start of treatment recover renal function. Renal transplantation can

be performed after disappearance of the circulating anti-GBM antibodies.

Treatment includes steroids, renal support, cytotoxic drugs (e.g. cyclophosphamide) and plasmapheresis.

Pulmonary signs and symptoms are improved by decreasing anti-GBM titres with plasmapheresis and immunosuppression. Clinical lapses during treatment are characterized by fever and decreased pulmonary and renal function. Patient survival is 77% at 1 year. Mortality is usually due to overwhelming sepsis or pulmonary haemorrhage.

PERIOPERATIVE MANAGEMENT

Elective surgery should be carried out during quiescent periods. Preoperative blood transfusion and dialysis may be necessary to optimize fluid, electrolyte and haemodynamic status.

INVESTIGATIONS

- *Full blood count* – Microcytic hypochromic anaemia; >90% of patients have a haemoglobin concentration of <12 g/dL
- *Coagulation* – Usually normal
- *Urea and electrolytes* – Derangement reflects degree of renal impairment
- *Chest radiograph* – Small discrete shadowing; confluent densities; bilateral alveolar infiltrates
- *Pulmonary function* – Restrictive picture; DLco elevated
- *ECG* – May show signs of electrolyte abnormalities; systemic hypertension; pulmonary hypertension

PREMEDICATION

- Avoid respiratory depressants
- Steroid cover

SPECIFIC MONITORING CONCERNS

- Monitor airway pressures and total pulmonary compliance as changes may reflect intraoperative pulmonary haemorrhage.

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SPECIFIC PROBLEMS

- If employing IPPV, maintain normocapnia and minimize the risk of volutrauma and barotrauma.
- Pulmonary haemorrhage can lead to airways/endotracheal tube obstruction. Use the largest appropriately sized tube with frequent suctioning.
- Avoid renally excreted drugs.
- Aseptic techniques for immunosuppressed patients.

POSTOPERATIVE MANAGEMENT

- Pulmonary physiotherapy
- Monitor renal function
- Increase steroid dosage

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CROSS-REFERENCES

Acute kidney injury, Chapter 5
Assessment of renal function, Chapter 5

HAEMOLYTIC URAEMIC SYNDROME

Haemolytic uraemic syndrome (HUS) is a triad of *renal failure, haemolytic anaemia* and *thrombocytopaenia* and is the most common cause of renal failure in infancy and childhood.

There are two predominant types: D+ HUS, which is associated with a prodromal diarrhoeal illness (90%–95% of cases) and D-HUS which is not. D+HUS is preceded by 4–6 days of diarrhoeal illness most commonly caused by infection with shigatoxin-producing *E coli. E coli* serotype O157:H7 has been associated with more than 60% of infections leading to HUS.

Of D-HUS cases, 30%–50% can be attributed to dysregulation of the alternative complement pathway. This involves mutations in factor H, factor I, CD46/MCP, factor B and C3 components. The aetiology of the remainder of cases is unknown. HUS patients present for anaesthesia most commonly for the creation of arteriovenous fistulae and shunts.

PATHOPHYSIOLOGY

This is a multisystem disease affecting not only the kidneys, erythrocytes and platelets, but also the gastrointestinal tract, liver, heart and CNS. It is classed as a thrombotic microangiopathy.

CARDIOVASCULAR SYSTEM

- Myocarditis, congestive heart failure and severe systemic hypertension.

RESPIRATORY SYSTEM

- Severe respiratory insufficiency may occur, unrelated to volume overload, pulmonary oedema or congestive heart failure.

CNS

- Drowsiness, seizures, hemiparesis and coma.

BIOCHEMICAL

- Evidence of acute renal failure, including acid–base and electrolyte disturbances
- Abnormal liver function tests associated with hepatitis

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HAEMATOLOGICAL

- Haemolysis rapidly appears; haemoglobin falls to as low as 4 g L^{-1}
- Thrombocytopaenia (lasting 7–14 days)
- Hepatosplenomegaly

RENAL SYSTEM

- Proteinuria, haematuria and oliguria, leading to anuria

GASTROINTESTINAL TRACT

- Haemorrhagic gastritis

IMMUNOLOGICAL

- Severe infections are common, e.g. peritonitis, meningitis and osteomyelitis

NATURAL HISTORY

- In D+ HUS, the mortality rate is 3%–5%. Older children and adults have poorer prognoses. Death is nearly always associated with severe extrarenal disease, including severe CNS involvement. Approximately two-thirds of children with D+ HUS require dialysis although 85% regain normal renal function.
- D-HUS has a relatively poor prognosis, with a mortality rate of up to 25% in the acute phase. Fifty percent of patients require renal replacement therapy at some point in the illness.

MANAGEMENT

- Fluid resuscitation may be necessary.
- Hyperkalaemia may occur and should be treated.
- Dialysis may be necessary.
- Hypertension should be controlled with standard antihypertensive agents.
- Plasma exchange (plasmapheresis in combination with fresh-frozen plasma replacement) may be necessary. Plasma exchange is performed daily until remission is obtained. However, because 85% of children with haemolytic uraemic syndrome recover after supportive therapy alone, plasma exchange is generally reserved for the most severe cases.

PREOPERATIVE ASSESSMENT

EXAMINATION

- Full neurological and cardiovascular examination
- Evidence of hepatic dysfunction
- Evidence of clotting disorders

INVESTIGATIONS

- Full blood count
- Urea, electrolytes and creatinine
- Liver function tests
- Glucose
- Coagulation studies
- Arterial blood gases
- Chest X-ray
- ECG

PREOPERATIVE MANAGEMENT

Premedication is unnecessary as patients in the acute phase tend to be lethargic and drowsy. Correct acid–base status, electrolyte and coagulation prior to surgery. Preoperative transfusion may be necessary. Continue any anticonvulsant therapy.

PERIOPERATIVE MANAGEMENT

General anaesthesia is preferred due to the presence of coagulation disorders in an uncooperative and severely ill child. A reduction in the dose of thiopental (less protein binding in hepatic disease) is usual. Rapid sequence induction should be performed. Isoflurane and atracurium are the ideal agents for maintenance, although desflurane or mivacurium are also appropriate. Frequent monitoring of acid–base and electrolyte status, temperature and urine output is required.

POSTOPERATIVE MANAGEMENT

Postoperative ventilation may be required in patients with severe cerebral involvement. Sepsis is a common complication.

Repeated procedures are common. A haemolytic crisis may last more than two weeks and anaemia can persist for months. Renal function may recover completely, or the child may require permanent haemodialysis.

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CROSS-REFERENCES

Anaesthesia for paediatric surgery – general principles, Chapter 24
Acute kidney injury, Chapter 5

NEPHROTIC SYNDROME

Eighty percent of cases are secondary to glomerulonephritis. Nephrotic syndrome can be primary, being a disease specific to the kidneys, or secondary to a systemic condition. Injury to glomeruli is an essential feature.

PRIMARY CAUSES

- Minimal-change nephropathy
- Focal glomerulosclerosis
- Membranous nephropathy
- Hereditary nephropathies

SECONDARY CAUSES

- Diabetes mellitus
- Lupus erythematosus
- Amyloidosis and paraproteinemias
- Viral infections (e.g. hepatitis B, hepatitis C, HIV)
- Pre-eclampsia

PRESENTING FEATURES

- Proteinuria (>3 g/24 h)
- Hypoalbuminaemia
- Hypercholesterolaemia
- Thromboembolic episodes

PATHOPHYSIOLOGY

A defect in the glomerular barrier leads to an increase in glomerular permeability which results in proteinuria/albuminuria. Reduced albumen leads to a decrease in plasma oncotic pressure, retention of sodium and water, peripheral oedema, ascites, pleural effusions and hypovolaemia. This physiologically deranged state puts the patient at risk of thromboembolism, commonly venous (deep venous thrombosis, renal vein thrombosis), but also arterial. Venous thromboembolism occurs in 10% of patients within 6 months of presentation. The definitive diagnosis is made by renal biopsy.

NATURAL HISTORY

Most cases spontaneously remit without treatment; hypertension occurs commonly; renal failure is rare.

The interval between initiating steroid therapy, and disease remission is a prognostic indicator for children with idiopathic nephrotic syndrome. Patients who respond to steroid treatment within 7 days do not relapse or relapse infrequently.

Patients may develop renal failure as well as secondary complications including thrombotic episodes and infection (which may be associated with immunosuppressive treatment).

In patients with secondary nephrotic syndrome, morbidity and mortality are related to the primary disease process, such as diabetes mellitus or lupus. In diabetic nephropathy, the magnitude of proteinuria relates directly to mortality.

TREATMENT

- Diuretics
- ACE inhibitors and angiotensin-receptor blockers are used alone or in combination
- Prednisolone
- Cyclophosphamide
- Mycophenolate
- Cyclosporin

Specific treatment depends on the aetiology. Glucocorticosteroids, e.g. prednisone, are used for minimal-change nephropathy. Prednisone and cyclophosphamide are useful in some forms of lupus nephritis. Secondary amyloidosis with nephrotic syndrome may respond to anti-inflammatory treatment of the primary disease. Patients with membranous nephropathy and a low risk for progression should be managed expectantly for the first 6 months.

PREOPERATIVE MANAGEMENT

- Drug history: diuretics, steroids, antihypertensives.
- Clinical signs of oedema.
- CVP monitoring (to assess volume status, as these patients are likely to have depleted intravascular volume).
- Assess renal function.
- Potassium supplementation may be required.

ANAESTHETIC CONSIDERATIONS

- Precautions as for any patient with renal impairment/failure.
- Low plasma protein concentrations can influence the pharmacodynamics of drugs which are highly protein-bound. Reduce dose of induction agents, especially thiopental and monitor neuromuscular blockade.
- Thromboembolic prophylaxis.

POLYCYSTIC DISEASE

Autosomal dominant inheritance. The disease progresses slowly leading to end-stage kidney disease in middle age.

PATHOPHYSIOLOGY

- Hypertension.
- Proteinuria.
- Decrease in urine concentrating ability early in the disease.

ASSOCIATED CYSTS

- Liver
- CNS (intracranial aneurysms)

TREATMENT

- Renal replacement therapy
- Renal transplantation

ANAESTHETIC CONSIDERATIONS

- As for end-stage kidney disease, if present.

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CROSS-REFERENCE

Assessment of renal function, Chapter 5

PATIENT WITH A TRANSPLANT

Transplant recipients may present for any surgery and it is imperative that no damage occurs to the organ. Most elective surgical procedures performed on renal transplant recipients are well tolerated; in general the renal handling of drugs is adequate, although renal function is rarely normal (Box 5.6).

COMMON UNDERLYING DISEASES

- Diabetes mellitus
- Glomerulonephritis
- Polycystic disease
- Hypertension

PROBLEMS AFTER TRANSPLANT

- Opportunistic infection
- Hepatitis B (<5% due to vaccine)
- Cancer risk (increased 30–100 times)
- Complications arising from associated medical conditions
- Large cell lymphoma (Epstein–Barr virus infection)

BOX 5.6 Causes of death in transplant recipients

- Sepsis
- Cardiovascular disease
- Suicide
- Gastrointestinal perforation

PREOPERATIVE PREPARATION

A formal assessment of renal function is essential. A careful history and examination should be undertaken. Take particular note of medications including immunosuppressives and antihypertensives.

CARDIOVASCULAR FACTORS

- Hypertension:
  - Essential
  - Secondary to end-stage renal failure
- Left ventricular failure:
  - Secondary to hypertension
  - Secondary to a chronic increase in cardiac output (shunts, atrioventricular fistulae, anaemia)
- Ischaemic heart disease, accelerated atherosclerosis
- Peripheral vascular disease
- Autonomic neuropathy: postural hypotension, delayed gastric emptying

ANAESTHETIC CONSIDERATIONS

- Continue immunosuppressive regimen
- Risk of pulmonary aspiration
- Infection risk
- Protect arteriovenous fistulae
- Gastrointestinal bleeding
- Steroid therapy
- Stress of surgery/anaesthesia
- Osteoporosis (care in moving and handling of patients)
- Potential for altered drug handling

PREMEDICATION

- Prophylactic antibiotics
- Supplementary steroid administration
- H_2 antagonist/metoclopramide/proton pump inhibitors
- Benzodiazepines (avoid diazepam due to its long half-life)
- Atropine/glycopyrrolate (20%–50% renal excretion)
- Opioids are not contraindicated

- Continue immunosuppression – if enteral administration is not possible then IV administration with dose adjustment

ANAESTHETIC TECHNIQUE

Local, general and regional techniques are well tolerated. General anaesthesia tends to decrease renal blood flow. Avoid hyperventilation, and excessive concentrations of volatile agents. Ensure adequate graft perfusion by maintenance of intravascular volume and avoidance of systemic hypotension.

Enflurane and sevoflurane are metabolized to fluoride and so usually avoided although the risk is small. Isoflurane and desflurane are recommended.

If the graft is functioning, all muscle relaxants and anticholinesterases are eliminated as normal. Atracurium, cisatracurium and mivacurium are the muscle relaxants of choice.

Opioids are safe although accumulation of morphine-6-glucuronide or norpethidine is possible.

If a regional technique is planned, then coagulation studies, including bleeding time, are indicated.

POSTOPERATIVE MANAGEMENT

Most surgery is performed without any specific complications. Monitor renal function closely, especially if undertaking major procedures. Immunosuppression should be continued, with antibiotics if indicated, and any infection appropriately managed. If there is any doubt concerning graft function, opioid infusions are best avoided.

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CROSS-REFERENCE

Assessment of renal function, Chapter 5

6

Endocrine system

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ACROMEGALY

Excessive secretion of growth hormone in adults causes hypertrophy of soft tissues and bone, particularly in the tips (Greek: *akros*, meaning extremities), leading to the name of the disease. Treatment may be surgical (hypophysectomy) or medical (dopamine antagonists or somatostatin analogues).

PATHOPHYSIOLOGY

GH and IGF-1 stimulate cell growth. Soft tissues, including viscera such as kidneys, heart and muscles, ligaments and cartilage are enlarged: large hands are often the first visible sign of acromegaly. Bones cannot grow in length so changes lead to deformity. Heart and kidney failure contribute to the two- to threefold increase in morbidity and mortality. GH induces hepatic gluconeogenesis and reduces glucose tolerance. IGF-1 is involved in the regulation of tumour growth, and epidemiological studies suggest that patients with acromegaly have a higher risk for malignancies, particularly of the thyroid, breast, prostate and probably colon. Patients complain of fatigue, headaches, arthralgias and hyperhidrosis.

An elevated IGF-1 level in peripheral blood is the most sensitive laboratory test. Single GH determinations are of little value, since GH secretion is pulsatile and blood levels vary widely. The failure of GH

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Table 6.1 Signs and symptoms of acromegaly

- Cardiovascular
  - Hypertension and cardiomegaly
  - Cardiomyopathy
  - Angiopathy (diabetic)
- Respiratory system
  - Obstructive sleep apnoea
  - Lower and rougher voice
  - Possible glottic stenosis or vocal cord paresis
- Soft tissues and skeleton
  - Hypertrophy of hands and feet
  - Macroglossia and prognathism
  - Coarse facial features
  - Carpal tunnel syndrome
- Neuromuscular system
  - Peripheral neuropathy
  - Myopathy with reduced strength
- Endocrine system
  - Impaired glucose tolerance, diabetes mellitus
  - Hypercalcaemia and hypercalcuria
  - Goitre (diffuse or nodular) with tendency to autonomy
  - Deficiency of pituitary hormones due to pressure of adenoma (ACTH – hypocortisolism; TSH – hypothyroidism; vasopressin – diabetes insipidus)

levels to decrease in response to a 75 g oral glucose tolerance test is helpful in confirming the diagnosis.

The pathological changes seen in acromegaly affect virtually every system (Table 6.1).

PREOPERATIVE ASSESSMENT

Primary concerns are potential difficulties in securing the airway and cardiovascular complications. Careful assessment of the airway is essential, including a history of snoring, intermittent nocturnal apnoea and daytime drowsiness. Preparations should be made for a difficult intubation; consider a possible awake fibre-optic intubation. Pay attention to possible concomitant cardiovascular, respiratory, neuromuscular and endocrine disorders. A history of reduced exercise capacity is important. Continuous postoperative respiratory monitoring in the ICU should be arranged beforehand, especially for the patient with obstructive sleep apnoea.

Premedication with an oral benzodiazepine may be used, but opiates should be avoided in patients with upper airway involvement. If glucose intolerance is present, the patient might require management as a diabetic.

INVESTIGATIONS

- Blood tests: creatinine, electrolytes, glucose, calcium, thyroid hormones
- Neck X-rays (for glottic involvement, pharyngeal tissue overgrowth)
- Indirect laryngoscopy may reveal vocal cord involvement
- Chest X-ray
- ECG
- Echocardiography if cardiomyopathy or valvular involvement suspected
- Baseline arterial blood gas analysis
- Cardiopulmonary exercise testing

PERIOPERATIVE MANAGEMENT

AIRWAY

Due to the facial deformity, it may be difficult to obtain a tight seal with the face mask. Intubation is the safest option, particularly for longer procedures or when access to the head is impaired.

Videolaryngoscopes may be useful, but awake fibre-optic intubation should be considered the safest option. Orotracheal intubation is preferred since the nasal mucosa may be hypertrophied. If there are no signs of upper airway involvement, intravenous induction and intubation under direct laryngoscopy is appropriate. Administer a neuromuscular blocking drug only after ventilation by bag and mask is secured. In the rare case that the glottis cannot be visualised, elective tracheostomy under local anaesthesia should be considered.

MONITORING

Routine AAGBI standard. If the fingers are too large for the SpO_2 probe, an ear lobe clip may be needed.

Consider an arterial cannula, central venous catheter and cardiac output measurement or oesophageal

Doppler in patients with cardiovascular impairment, particularly for extensive surgery.

POSTOPERATIVE MANAGEMENT

Extubate only when muscle relaxation has completely recovered. Patients with airway obstruction should be closely monitored with pulse oximetry and serial arterial blood gases during the first 24 hours postoperatively, preferably in the ICU or HDU. Supplemental oxygen should be administered as necessary. Drugs that reduce muscular tone (e.g. benzodiazepines) must be administered with great caution to avoid inducing loss of pharyngeal muscle control and upper airway obstruction. Forced inspiration against the occluded airway can precipitate negative pressure pulmonary oedema.

Hypophysectomy will terminate excessive GH production with associated gluconeogenesis and hyperglycaemia. Insulin therapy must be carefully adapted to the reduced requirements. The operation can also cause a deficiency of other pituitary hormones, such as vasopressin, ACTH or TSH that will require appropriate treatment.

Some clinical features of acromegaly may resolve slowly following treatment (e.g. vocal cord changes, left ventricular hypertrophy) but osseous hypertrophy will be permanent.

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CROSS-REFERENCES

ADRENOCORTICAL INSUFFICIENCY

Adrenocortical insufficiency, characterized by inadequate cortisol secretion and low serum cortisol concentration, is classified as primary, secondary or tertiary depending on whether the disturbance is in the adrenal cortex, pituitary or hypothalamus. Differentiation of the latter two is of academic interest only and both are referred to as secondary.

Primary adrenocortical insufficiency is the inability of the adrenal cortex to secrete cortisol and aldosterone due to destruction of the gland. Classical Addison's disease is caused by an autoimmune process and is important because it is frequently part of the autoimmune polyendocrine syndrome, type 2 (APS-II) that includes Hashimoto thyroiditis hypothyroidism and type I diabetes mellitus. Patients should therefore undergo appropriate endocrine screening. Other causes of primary adrenocortical insufficiency are given in Table 6.2.

Secondary adrenocortical insufficiency is due to a lack of ACTH stimulation. The most common cause is suppression of CRH and ACTH secretion by exogenous glucocorticoids. Under normal circumstances these patients do not suffer from lack of cortisol and may present with signs of hypercortisolism. Insufficiency becomes manifest when glucocorticoid administration is stopped or not adapted to increased perioperative requirements. Further causes of secondary adrenocortical insufficiency are damage to the pituitary or hypothalamus, e.g. by tumours, postpartum or surgery. If untreated, these

Table 6.2 Aetiology of primary adrenocortical insufficiency

- Idiopathic
- Autoimmune (Addison's disease)
- Congenital adrenal hyperplasia (mainly adrenal 21 hydroxylase deficiency)
- Infection (e.g. tuberculosis, meningococcal)
- Surgical removal
- Haemorrhage/infarction (e.g. Waterhouse–Friderichsen syndrome)
- Infiltration, malignant destruction, amyloid
- Adrenal leukodystrophy (rare)

patients are likely to show signs and symptoms of adrenocortical insufficiency.

Determining the cause of abnormally low serum cortisol concentrations requires testing the components of the hypothalamic-pituitary-adrenal (HPA) axis. A normal response to stimulation with synthetic ACTH (Synacthen test) virtually rules out primary insufficiency, but further testing is required if secondary insufficiency with involution of the adrenal cortex is likely, such as in chronic glucocorticoid therapy. Low circulating ACTH concentrations and a lack of response to CRH stimulation can indicate pituitary dysfunction and consequently secondary adrenal insufficiency. Insulin-induced hypoglycaemia ('insulin tolerance test') tests the integrity of the HPA axis as a whole, but is hazardous and restricted to specialist facilities.

For clinical purposes, differentiation between patients with nonfunctioning adrenal cortex glands who are on glucocorticoid substitution therapy and those who are not is important. The former are compensated and can be treated as normal patients, whereas the latter tend to suffer circulatory collapse and develop symptoms of manifest insufficiency in periods of stress and illness.

Signs and symptoms of manifest adrenocortical insufficiency are

- Muscle weakness, myalgia, easy fatigability
- Loss of appetite, weight loss (in part due to fluid losses)
- Disorientation, confusion, dizziness
- Postural hypotension
- Nausea, vomiting and diarrhoea

PATHOPHYSIOLOGY

CARDIOVASCULAR SYSTEM

Aldosterone increases sodium reabsorption in the distal tubule, and its lack leads to sodium wasting and fluid loss with hypovolaemia. Cortisol up-regulates beta-adrenergic receptors and facilitates the effects of circulating catecholamines. Its prolonged absence leads to catecholamine-refractory hypotension.

SKIN

Patients with Addison's disease have a typical dark pigmentation particularly in the mouth and on skin creases. This is caused by melanocyte-stimulating hormone (MSH), which is derived from the same polypeptide precursor and is co-secreted with ACTH. MSH levels increase together with ACTH due to the absence of negative cortisol feedback control.

BIOCHEMICAL/METABOLIC

- Sodium loss, potassium retention (hyponatraemia, hyperkalaemia, high urinary sodium excretion)
- Hyperuricaemia
- Hypoglycaemia on fasting in isolated adrenocortical insufficiency
- Hyperglycaemia if associated with diabetes mellitus as part of AES-II
- Hypercalcaemia

BLOOD COUNT

- Haematocrit normal or elevated due to dehydration
- Normochromic normocytic anaemia, low haematocrit (after rehydration)
- Eosinophilia, leucocytosis

PREOPERATIVE ASSESSMENT

Previously undiagnosed adrenocortical insufficiency is difficult to detect because the symptoms are non-specific and tend to be attributed to more likely causes. The typical hyperpigmentation and blood chemistry are the most helpful clues to the diagnosis. Stress of surgery, infection or trauma can cause the condition to decompensate and precipitate an Addisonian crisis with its high mortality.

Prolonged steroid therapy can suppress the HPA axis without causing symptoms, since these are prevented by the exogenous steroid. In these patients, symptoms will become manifest if the steroids are withheld or the dose not adapted to increased requirements.

In addition to routine laboratory investigations (electrolytes, fasting blood glucose, full blood count, creatinine, urea), these patients should be screened for hypothyroidism and diabetes mellitus. A chest X-ray for signs of pulmonary or cardiac disease and an ECG should be performed.

PREOPERATIVE MEASURES

Patients with adrenocortical dysfunction maintained on adequate corticosteroid substitution therapy are not necessarily at any additional risk. However, they will require perioperative increase of the dose to compensate for the increased requirements posed by the trauma of surgery. How much to give depends on the surgical procedure.

Patients with untreated adrenocortical insufficiency should not be scheduled for elective surgery until corticosteroid therapy has been given for a sufficient length of time as determined by the endocrinologists. This can take days to weeks.

Patients with untreated manifest adrenocortical insufficiency or Addisonian crisis presenting for emergency surgery are high-risk patients who require invasive intraoperative monitoring and must be managed postoperatively on the ICU. Preoperative measures are aimed at stabilizing the circulation, correcting fluid deficits and electrolyte disturbances and initiating glucocorticoid therapy. Infusion pumps with noradrenaline and/or vasopressin should be immediately available.

PERIOPERATIVE MANAGEMENT

PATIENTS ON STABLE LONG-TERM GLUCOCORTICOID SUBSTITUTION THERAPY

These patients will generally not require more invasive measures for any given operation than a comparable patient with functioning adrenal glands. There is no particular requirement for the choice of anaesthetic. Additional glucocorticoid is needed to meet the increased demands after the surgical trauma.

PATIENTS WITH LATENT ADRENOCORTICAL INSUFFICIENCY

Patients with poorly controlled adrenocortical insufficiency are at a higher risk and require a more invasive approach. They are frequently dehydrated, hypoglycaemic and have cardiovascular instability, in part due to the down-regulation and desensitization of the catecholamine receptors associated with cortisol deficiency.

In addition to routine monitoring, the following are recommended:

- Arterial cannula for continuous invasive blood pressure recording
- Urinary catheter (renal function, fluid balance)
- Central venous catheter (estimation of right atrial filling pressure, inotrope infusions)
- Cardiac output measurement for haemodynamically unstable patients

Depending on the extent of surgery and the patient's tolerance of the procedure, the patient can be managed in a postanaesthetic care unit, but might require transfer to ICU.

PATIENTS WITH MANIFEST ADRENOCORTICAL INSUFFICIENCY

A patient with Addisonian crisis who requires emergency surgery should be prepared and stabilized as long as possible in the ICU. Insert large-bore venous cannulae for rapid fluid infusions, an arterial cannula for invasive BP monitoring and measuring blood gases and acid-base status, a CVP line for estimating right atrial filling pressure and infusing vasopressors and inotropic agents. Cardiovascular function monitoring is recommended (cardiac output, TOE). Place a urinary catheter for monitoring renal function and assessing fluid balance.

Extravascular dehydration and hyponatraemia are treated with normal saline (hypernatraemia must be corrected very slowly to avoid pontine myelinosis); plasma expanders can be given to correct hypovolaemia. Use glucose infusions to correct hypoglycaemia; an insulin infusion may also be needed to lower elevated serum potassium concentrations.

Hydrocortisone (200 mg IV bolus) or equivalent dose of other glucocorticoid then a hydrocortisone infusion (100 mg in 24 hours or equivalent). Fludrocortisone (0.1 mg daily) for mineralocorticoid deficiency.

Noradrenaline infusion may be needed to counteract hypotension due to vasodilatation and a vasopressin infusion if the response to noradrenaline is insufficient.

GENERAL ANAESTHESIA

Induction should be performed cautiously with small doses of hypnotic repeated at delayed intervals to

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allow for increased sensitivity, hypovolaemia and prolonged circulatory times. Blood pressure control can be impaired and they can be overly sensitive to negative inotropic effects and suppression of the baroreceptor reflex. There is no particular indication for any one anaesthetic technique or agent. Etomidate is not contraindicated because its transitory inhibitory effect on steroid synthesis is irrelevant and its cardiostability is beneficial.

Infusion of vasoconstrictors (noradrenaline, vasopressin) and inotropic agents may be required. Continuous infusions of crystalloid solutions will be necessary to correct preoperative fluid deficits and replace perioperative losses. Colloids may be necessary to replace blood loss.

LOCAL ANAESTHESIA

Cardiovascular instability during surgery may require additional intravenous fluid replacement and inotropic support.

POSTOPERATIVE MANAGEMENT

Invasive haemodynamic monitoring is recommended. Balance fluid intake and output, correct electrolyte abnormalities, monitor renal function and continue corticosteroid therapy.

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CROSS-REFERENCES

Iatrogenic adrenocortical suppression, Chapter 6
Fluid and electrolyte balance, Chapter 30

CARCINOID SYNDROME

Carcinoid syndrome is caused by peptides, particularly serotonin (5-hydroxytryptamine [5-HT]) and bradykinin, which reach the systemic circulation in abnormally high concentrations after release by carcinoid tumours. The incidence is approximately 8 in 100,000 population.

Carcinoid tumours are derived from argentaffin cells and may occur in several locations, e.g. bronchus and pancreas, although 75% are found in the gastrointestinal tract, most commonly the appendix. Appendicial tumours are usually benign and nonsecreting. The amines and peptides responsible for the symptoms of carcinoid syndrome are produced by malignant tumours. Up to 20 peptides and amines have been isolated including 5HT, bradykinin, histamine, somatostatin, prostaglandins, vasoactive peptide and substance P. Only about 7%–18% of patients with carcinoid tumours exhibit the carcinoid syndrome, as only 25% of malignant tumours produce peptides and these are normally cleared from the portal circulation by the liver if the tumour is in the gastrointestinal tract. Carcinoid syndrome usually results from the presence of liver secondaries which secrete peptides directly into the hepatic veins and thus into the systemic circulation. Bronchial tumours release peptides which bypass the portal circulation, resulting in symptoms at an earlier stage.

PREOPERATIVE EVALUATION

A CT or MR scan will determine the site of the tumour and the possible existence of metastases. Symptoms may be caused by the primary tumour, e.g. intestinal obstruction, haemoptysis or from systemic effects of peptides released by the tumour, e.g. right heart valve lesions.

5HT may produce:

- Watery diarrhoea (75%) associated with cramps possibly resulting in dehydration, hyponatraemia, hypokalaemia and hypochloraemia.
- Malabsorption with steatorrhoea and hypoproteinaemia.
- Pallor.
- Hypertension (5-HT stimulates release and inhibits uptake of norepinephrine and potentiates the response of alpha-1 adrenoreceptors to catecholamines).
- Tachycardia.
- Hyperglycaemia.
- Right heart failure (33%) due to pulmonary stenosis and tricuspid regurgitation resulting from subendocardial fibrosis.
- Raised urinary 5-hydroxyindoleacetic acid (5-HIAA) levels, which are diagnostic of carcinoid syndrome. A 24-hour urinary collection is usually undertaken.

Bradykinin may produce:

- Flushing (90%) of the face and upper body increasing in duration as the disease progresses.
- Hypotension.
- Bronchospasm (20%), especially in previous asthmatics and in the presence of cardiac disease.

Histamine may produce:

- Flushing
- Hypotension
- Bronchospasm

PREOPERATIVE DRUG THERAPY

Preoperative drug therapy is aimed at antagonizing the mediators of the carcinoid syndrome or preventing their release from carcinoid tumours.

SEROTONIN ANTAGONISTS

Cyproheptadine and methysergide are effective against gastrointestinal manifestations. Ketanserin blocks the 5-HT$_2$ receptor (vasoconstriction, bronchoconstriction, platelet aggregation), and has adrenergic antagonist

activity, reduces central sympathetic outflow and is used to treat carcinoid induced hypertension.

BRADYKININ ANTAGONISTS

Aprotinin inhibits the kallikrein cascade. It is used by infusion to control flushing and treat hypotension. Steroids reduce prostaglandin synthesis which mediates the action of bradykinin.

HISTAMINE ANTAGONISTS

H$_2$ antagonists or combination antihistamines are more effective than H$_1$ blockers on their own.

INHIBITORS OF MEDIATOR RELEASE

Somatostatin inhibits the release of mediators from carcinoid tumours. It must be given by infusion. Octreotide, a long-acting synthetic somatostatin analogue, is used as a sole agent to treat diarrhoea, hypertension, hypotension and bronchospasm in patients with carcinoid syndrome. It reduces the plasma levels of mediators by inhibiting their release from carcinoid tumours.

ANAESTHETIC CONSIDERATIONS

- Hypovolaemia and electrolyte abnormalities may be significant in patients with severe diarrhoea.
- Give somatostatin or somatostatin analogues to prevent the release of mediators.
- Avoid factors that can trigger a carcinoid crisis by causing mediator release.
  - Catecholamines, (release peptides from carcinoid tumours)
  - Anxiety, hypercapnia, hypothermia and hypotension (release catecholamines)
  - Morphine, atracurium, suxamethonium (release histamine)
  - Hypertension (causes bradykinin release)
- Prepare for a carcinoid crisis (resistant bronchospasm and sudden variations in arterial pressure, particularly at induction of anaesthesia and when the tumour is handled).

PREOPERATIVE MANAGEMENT

- Correct fluid and electrolyte abnormalities.
- Consider nutritional support if malabsorption is severe.
- Echocardiography may be valuable to investigate right heart and tricuspid valve function.
- Octreotide, 100 mcg subcutaneously two or three times a day for 2 weeks prior to surgery followed by 100 mcg IV at induction of anaesthesia and a slow postoperative wean over a few days.
- Continue antagonists of serotonin, bradykinin and histamine to minimize symptoms and maintain haemodynamic stability.
- All mediator antagonists should be available for immediate administration if required perioperatively.
- Premedication should include an anxiolytic drug and a sedative antihistamine.

PERIOPERATIVE MANAGEMENT

Monitoring starts prior to induction and should include:

- Intra-arterial BP
- ECG
- Central venous pressure
- Blood gases
- Blood sugar
- Airway pressure
- In patients with right-sided heart lesions, pulmonary hypertension must be avoided and pulmonary artery catheterization should be considered.
- Oesophageal Doppler may be useful

Regional anaesthesia is relatively contraindicated, as hypotension may occur. General anaesthesia should be induced with drugs which maintain haemodynamic stability, obtund the hypertensive response to laryngoscopy and tracheal intubation, and do not release histamine. Avoid suxamethonium and ketamine. Volatile agents may delay recovery and cause myocardial depression but are often used. A technique including high-dose narcotics has been used successfully.

An anaesthetic machine with different ventilatory modes may be required to compensate for episodes of bronchospasm.

Treat hypotension with fluid or aprotinin infusion guided by CVP or oesophageal Doppler. Angiotensin and vasopressin have also been used. Catecholamines should not be given as they cause the release of peptides. Control hypertension with intravenous ketanserin and cyproheptadine. Adrenergic receptor antagonists and clonidine have also been used, but can precipitate hypotension. Somatostatin and its analogues prevent pre- and perioperative episodes of hypertension, hypotension and bronchospasm, which may be resistant to other forms of drug therapy. IV octreotide may be useful in the treatment of an intraoperative carcinoid crisis.

POSTOPERATIVE MANAGEMENT

Recovery may be delayed and close monitoring should continue on HDU or ICU. If octreotide has been used preoperatively it should be reduced slowly over the first postoperative week.

The severity of symptoms does not predict the severity of perioperative complications, so that patients with minor preoperative symptoms may have significant intraoperative complications. Perioperative preparation and vigilance is of great importance. The introduction of somatostatin and its analogues has shifted the emphasis of treating perioperative carcinoid crises from antagonizing mediators which have been released to inhibiting their release from carcinoid tumours altogether.

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patients undergoing laparotomy for carcinoid syndrome. *Br J Anaesth* **72**: 335–341.

CROSS-REFERENCES

CONN SYNDROME

Hypersecretion of aldosterone leads to Conn syndrome or hyperaldosteronism. Excess aldosterone production may be primary due to an adrenal adenoma (Conn syndrome – 30%), bilateral adrenal hyperplasia (70%) or adrenal carcinoma (rare), or secondary caused by an overactive renin-angiotensin system resulting from renal artery stenosis, congestive heart failure or hypoalbuminaemia as in liver cirrhosis or nephrotic syndrome.

Primary (hyporeninaemic) hyperaldosteronism was thought to be rare, but more recent epidemiological studies have shown it to be responsible for up to 20% of cases of moderate to severe hypertension. Adrenal hyperplasia, the most common cause, has its highest prevalence in men over the age of 60.

SIGNS AND SYMPTOMS

Primary hyperaldosteronism can be asymptomatic but usually presents as hypertension. The patient might complain of muscle weakness, muscle spasms or intermittent paralysis, fatigue and headaches. Polyuria, polydipsia and nocturia are described. Laboratory investigations show normal to low serum potassium, low renin levels, hypernatraemia and metabolic alkalosis. Aldosterone levels are useful in the diagnosis but aldosterone antagonists, ACE inhibitors, AT-II receptor antagonists and beta-receptor blockers must be discontinued for up to four weeks prior to testing to avoid false positive or false negative results. Renal function may be abnormal, and the chest X-ray might show a widened heart silhouette due to hypokalaemic cardiomyopathy. The hypertension of hyperaldosteronism predisposes to cardiovascular complications.

CARDIOVASCULAR SYSTEM

- Hypertension, moderate to severe
- ECG signs of hypokalaemia and hypomagnesaemia (U waves and flattened T wave)
- Congestive heart failure, cardiomyopathy

LABORATORY

- Low renin
- High aldosterone
- Hypokalaemic alkalosis from renal tubular loss of potassium and magnesium
- Hypernatraemia
- Possible abnormal glucose tolerance test

TREATMENT

Treatment is based on the results of MR scans and possibly selective adrenal vein sampling. Primary hyperaldosteronism due to an adenoma (unilateral, aldosterone in adrenal venous sample) is treated by surgical removal. Hyperaldosteronism due to adrenal hyperplasia (bilateral) is treated with spironolactone or potassium canreonate, both aldosterone antagonists.

PREOPERATIVE EVALUATION

INVESTIGATIONS

- Creatinine (or urea), electrolytes, haemoglobin and blood glucose
- Acid-base status (alkalosis)
- ECG (arrhythmias, left ventricular hypertrophy)
- Chest X-ray (cardiomegaly)
- Blood typing

PREPARATIONS

- Restore potassium losses: infusion of up to 20 mmol K^+ h^{-1}. Replacing the deficit will require at least 24 hours. Normal ECG indicates therapeutic success.
- Blood pressure control with aldosterone antagonist (if not already part of the

treatment regimen), e.g. oral spironolactone or eplerenone, or intravenous canreonate potassium.

- Control of hyperglycaemia with insulin infusions, if necessary.

PERIOPERATIVE MANAGEMENT

- Premedication with benzodiazepine to prevent hypertensive episodes.
- Induction in customary manner (etomidate could be used and its inhibitory effect on steroid synthesis may be exploited to reduce aldosterone production).
- A balanced technique with opioids and controlled ventilation is the method of choice.
- Continuous thoracic epidural anaesthesia continued into the postoperative period helps to prevent hypertensive episodes, but may require a perioperative infusion of noradrenaline to counteract hypotension from vasodilatation.
- The technique used for adrenalectomy (laparoscopic or open) and the patient's position (supine or lateral decubitus) depends on the size of the tumour, its location and the surgeon's preference.
- Intraoperative blood loss is generally minimal but can be considerable if major adjacent blood vessels are injured. Muscle relaxation must be adequate to ensure easy surgical access. The diaphragm may be breached with resulting pneumothorax.

INTRAOPERATIVE MONITORING

- Arterial cannula for close monitoring of blood pressure and to monitor acid–base balance.
- Central venous line for monitoring fluid requirements (inaccurate in patients with cardiomyopathy).
- ECG monitoring during induction and intubation (high risk of arrhythmia, ischaemia).
- Capnography (note that hyperventilation exacerbates alkalosis).
- Urinary catheter to monitor renal function.
- Peripheral nerve stimulation (increased sensitivity to neuromuscular blocking agents).

- Hypertensive episodes can occur during initial inflation of pneumoperitoneum or surgical manipulation of the gland. Treat with vasodilators (e.g. labetolol, urapadil, glyceryl trinitrate, phentolamine). Avoid beta-blockers except to treat reflex tachycardia.

POSTOPERATIVE CARE

- HDU or ICU for possible postoperative cardiovascular instability and electrolyte disturbances. Potassium, sodium and blood pressure slowly return to normal.
- Effective analgesia, e.g. thoracic epidural or patient-controlled analgesia after open surgery.
- Postoperative respiratory support for impaired ventilation (dorsolumbar or abdominal incision; preexisting myopathy).
- Watch for: cardiac arrhythmias, abnormal glucose tolerance, impaired renal function, residual pneumothorax.
- Requirement for glucocorticoid or mineralocorticoid supplementation must be decided in each individual case, e.g. after bilateral adrenalectomy.

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CROSS-REFERENCES

Iatrogenic adrenocortical suppression, Chapter 6
Intraoperative hypertension, Chapter 30

CUSHING SYNDROME (HYPERCORTISOLISM)

This term refers to a typical set of symptoms caused by high circulating levels of cortisol or other glucocorticoids that can be of endogenous or exogenous origin. Endogenous Cushing syndrome can be secondary to excess ACTH production: the eponymous Cushing disease is caused by an ACTH-secreting pituitary tumour. ACTH secretion can be part of the paraneoplastic syndrome seen with bronchial or pancreatic carcinoma.

CLASSIFICATION AND CAUSES OF CUSHING SYNDROME

PRIMARY (NON-ACTH DEPENDENT)

- Adrenal tumours (adenoma, carcinoma)
- Adrenal hyperplasia

SECONDARY (ACTH-DEPENDENT, 60% OF NON-IATROGENIC CASES)

- Pituitary adenoma (Cushing disease)
- Ectopic ACTH-producing tumours (bronchial, pancreatic carcinoma)

IATROGENIC

- Glucocorticoid administration

OTHER

- Pseudo-Cushing syndrome (alcoholism, polycystic ovarian syndrome, depression)

PATHOPHYSIOLOGY AND SURGICAL THERAPY

The features of Cushing syndrome are a direct result of the physiological effects of glucocorticoids. The symptoms with the greatest relevance for the anaesthetist are arterial hypertension, impaired glucose tolerance, susceptibility to infections, fragile skin, fat deposits on the back of the neck (positioning), osteoporosis, facial obesity and hypokalaemia. In hypercortisolism due to exogenous glucocorticoid treatment, ACTH secretion is suppressed and cortisol secretion from the adrenal glands might not be sufficient to meet the demands of stressful situations.

Cushing's disease is treated surgically by removal of the pituitary, preferably using a minimally invasive technique such as an endoscopic trans-sphenoid procedure. The standard surgical therapy of primary hypercortisolism is laparoscopic adrenalectomy, which has a very low perioperative mortality rate.

PREOPERATIVE ASSESSMENT AND PREPARATION

Two scenarios involving patients with Cushing syndrome are possible:

1. Surgery undertaken to cure the condition (e.g. hypophysectomy, bilateral adrenalectomy).
2. Where hypercortisolism is not related to the surgical procedure (most common).

Both groups require similar intraoperative care but postoperative management differs.

In addition to standard preoperative assessment, note must be taken of the severity and treatment of hypertension, presence and treatment of diabetes mellitus, ability of patient to recline the head, presence of skin lesions and serum potassium. These conditions and not cortisol levels themselves will determine the necessity of extended intraoperative monitoring (arterial cannulation, central venous catheter) and intensive postoperative care.

HISTORY

- *Metabolic* – Diabetes
- *Cardiopulmonary* – Hypertension, dyspnoea
- *Skin* – Tendency to bruising, fragile skin, poor wound healing
- *Muscles/bones* – Weakness, osteoporosis, pathological fractures
- *CNS* – Depression, psychiatric symptoms
- *Glucocorticoid therapy* – Drug, dose, duration
- *Medication* – Antihypertensives, antidiabetics, diuretics

EXAMINATION

- *Skin and subcutaneous tissue* – Oedema, veins for venous access, skin infection, moon face, nuchal fat
- *Airway* – Mouth opening, obstructions
- *Bone* – Kyphosis, neck movement
- *Muscle* – Proximal muscle wasting, endocrine myopathy

LABORATORY AND TECHNICAL INVESTIGATIONS

- Full blood count, renal function parameters, electrolytes, blood glucose
- ECG: ischaemia, arrhythmias
- Chest X-ray: cardiomegaly, pulmonary congestion, pulmonary masses or atelectasis (bronchial carcinoma and paraneoplasia)
- Respiratory function test (myopathy affecting respiratory muscles)

PERIOPERATIVE MANAGEMENT

Premedication if required. Continue preoperative medications as usual. Patients taking glucocorticoids should receive their morning dose and may require augmented intraoperative supplementation. Patients with diabetes should be treated appropriately. Careful positioning is crucial to avoid damage to fragile skin and fractures of osteoporotic bones.

No particular anaesthetic technique is indicated, but intubation with positive pressure ventilation is generally recommended. Administer drugs slowly in patients with impaired cardiovascular function. Distribution volumes and protein binding may be altered and sensitivity to neuromuscular blocking agents may be increased.

Induction of anaesthesia may be complicated by difficulties in establishing venous access and by a difficult airway. Appropriate equipment should be immediately available.

Intraoperative monitoring is tailored to the surgical procedure and the preexisting diseases, but is generally more invasive.

MONITORING

BASIC

- ECG (with ST-segment analysis), noninvasive blood pressure, pulse oximetry, capnography, neuromuscular blockade

SUPPLEMENTAL

- CVP, invasive arterial pressure, arterial blood gases, urine output

INVASIVE

- Pulmonary arterial pressures, cardiac function (e.g. TOE or thermodilution)

POSTOPERATIVE MANAGEMENT

Except for minor procedures, transfer to ICU particularly after intra-abdominal or thoracic surgery. Provide adequate analgesia (e.g. PCA). Respiratory and cardiovascular complications are the primary concern. Electrolyte imbalance and hyperglycaemia may persist or reappear and there is an increased risk of cardiovascular events and deep vein thrombosis with pulmonary thromboembolism. Early mobilisation is recommended although muscle weakness and obesity can interfere with mobilisation. Stress ulcer prophylaxis is mandatory. Blood glucose control with insulin will be needed for diabetes mellitus. Glucocorticoid therapy in patients with iatrogenic Cushing syndrome or surgical removal of the source of excess cortisol.

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CROSS-REFERENCE

Iatrogenic adrenocortical suppression, Chapter 6

DIABETES INSIPIDUS

Diabetes insipidus (DI) is characterized by polyuria (up to 1 L h^{-1}) with dilute urine (50–100 mOsm kg^{-1}), excessive thirst and polydipsia. Untreated DI leads to dehydration with hypernatraemia and hypokalaemia; plasma osmolality and sodium concentrations may be in the normal range if the patient is allowed to drink *ad libitum*.

Several forms of DI exist (Table 6.3) but the most common and most relevant is neurogenic, or central DI resulting from reduced or absent vasopressin secretion from the posterior pituitary.

In nephrogenic DI, which can be hereditary or drug-induced and is the second most common form of DI, the kidneys do not respond to the water-retaining action of vasopressin, while in gestational DI, circulating vasopressin is degraded by vasopressinase produced in the gravid uterus. DI in pregnancy may also be a symptom of preeclampsia or gestational liver disease; conditions that are treated by delivery of the baby to avoid maternal or neonatal mortality.

Central DI is not affected by fluid restriction but does respond to desmopressin – this differentiates it from nephrogenic DI.

A form known as dipsogenic DI is caused by exaggerated fluid intake, either habitual or due to psychiatric disturbances or to a defect in hypothalamic thirst control. Desmopressin is ineffective in dipsogenic DI and is even likely to increase the sense of thirst, but fluid restriction should reduce diuresis and increase urine osmolality.

About 25% of the cases of central DI have no obvious cause and are classified as idiopathic. That most likely to be encountered by the anaesthetist is acquired and results from brain damage due to tumour, trauma or during neurosurgery. This may be transient or permanent, but most frequently tends to run a triphasic course. Initial polyuria can begin intraoperatively and last for up to 5 days. This is followed by an antidiuretic phase of approximately 5 days. A third phase of permanent DI can follow depending on the extent of damage to the hypothalamus and pituitary.

Patients with concomitant untreated cortisol and vasopressin deficiencies (e.g. after hypophysectomy) may not present with polyuria because hypocortisolism reduces renal free water clearance. Treatment with cortisol will unmask the vasopressin deficiency and induce polyuria.

PREOPERATIVE ASSESSMENT

Patients with preoperative DI are usually already being treated for the disorder. No patient with suspected DI should undergo scheduled surgery until it is either ruled out, or confirmed and treated.

Ongoing therapy should be recorded and decisions made how it is to be continued. Patients with central DI will usually be taking desmopressin (DDAVP) as nasal spray (5–20 μg twice daily) or orally (0.1–0.4 mg three times daily). This should be given on the day of surgery.

Patients should not be subjected to prolonged periods without fluids, and those managed with *ad libitum* oral fluid intake will require an infusion to be established several hours before surgery in order to avoid a full stomach at induction.

Table 6.3 Forms of diabetes insipidus

Form	Pathophysiology	Cause	Treatment
Central	Lack of vasopressin	Idiopathic (25%)	Desmopressin (DDAVP)
		Genetic (rare)	Carbamazepine
		Acquired: trauma, neurosurgery, tumour (brain, lung), infection	
Nephrogenic	Insensitivity of kidney to vasopressin	Genetic: vasopressin receptor defect (V_2), aquaporin 2 defect	Hydrochlorothiazide
		Acquired: kidney disease, drugs (e.g. lithium, demeclocycline, methoxyflurane, amphotericin B)	Amiloride
		Electrolyte imbalance: e.g. hypercalcaemia, hypokalaemia	
Gestational	Lack of vasopressin	Increased degradation of vasopressin by vasopressinase	DDAVP
Dipsogenic	Polydipsia	Disturbed thirst sensation	Fluid restriction
		Psychiatric disorders	

Preoperative assessment should include signs of dehydration, low cardiac filling pressures, serum electrolytes, osmolality and renal function (urea, creatinine). Information on urine volume is needed. Serum and urine osmolalities should be determined.

Hypernatraemia and hypovolaemia should be treated gradually. Too rapid changes of serum osmolality or acute volume loading can precipitate cardiac decompensation or cerebral oedema.

PERIOPERATIVE MANAGEMENT

One of the challenges in managing a patient with DI is maintaining fluid and electrolyte balance. This task is easier if diuresis is controlled with titrating doses of DDAVP (e.g. 1 µg IV doses). In addition to standard monitoring, measure urine output and serum concentrations of sodium and potassium. If possible, serum and urine osmolalities should be measured frequently and DDAVP given if serum osmolality exceeds 290 mOsm L^{-1}.

Be aware that DI can arise without warning at any time during neurosurgical procedures. The diagnosis should be considered if diuresis continuously exceeds 200–300 mL h^{-1} without volume loading or diuretics (e.g. mannitol).

Fluid and electrolyte balance must continue in the postoperative period and DDAVP administered as necessary. Urine output, vascular filling pressures and serum osmolality should be monitored in the ICU following major surgery or significant blood losses.

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE HYPERSECRETION (SIADH)

SIADH is characterised by the excessive release of vasopressin despite low plasma osmolality. It is frequently seen in patients with CNS lesions (trauma, tumour, infections), but can also occur due to ectopic AVP secretion, e.g. in pulmonary tuberculosis or as a paraneoplastic syndrome in small cell bronchial carcinoma. SIADH can also be caused by drugs, such as neuroleptics, antidepressants (tricyclic, SSRI), amiodarone, chlorpropramide, anticonvulsants (carbamazepine), ecstasy and others. It must be differentiated from the hypo-osmolar hyponatraemia that can occur as a complication of hypothyroidism or adrenal insufficiency.

The water retention and natriuresis caused by AVP leads to dilutional hyponatraemia occasionally, but not always, with fluid overload. The patient complains of headache, nausea and vomiting and can be confused. Serum sodium concentrations below 120 mmol L^{-1} can cause convulsions and coma.

Management includes treating the cause whenever possible. SIADH is usually asymptomatic and restricting fluids to 800–1,000 mL per day usually

suffices to increase serum sodium. Demeclocycline is a potent AVP receptor antagonist that can be used off-label when fluid restriction is difficult to enforce. Severe symptomatic hyponatraemia requires treatment with intravenous hypertonic saline. Correction of the serum sodium concentration should not be more than 12 mmol L^{-1} per day since a more rapid increase can cause central pontine myelinolysis.

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CROSS-REFERENCES

Adrenocortical insufficiency, Chapter 6
Anaesthesia for trans-sphenoidal hypophysectomy, Chapter 14
Conn's syndrome, Chapter 6
Cushing's syndrome, Chapter 6
Diabetes mellitus (IDDM), Chapter 6
Intraoperative hypertension, Chapter 30

DIABETES MELLITUS

TYPE 1 (INSULIN-DEPENDENT DIABETES MELLITUS)

- An autoimmune disorder of the pancreas characterized by β-cell destruction.
- Onset usually in younger age group.
- Absolute insulin deficiency.
- Abrupt onset of symptoms.
- Tendency to ketosis.
- Diagnosed by consistently raised random plasma glucose >11.1 mmol L^{-1} (venous whole blood glucose >10 mmol L^{-1}) or fasting plasma glucose >7.0 mmol L^{-1} (blood glucose >6.1 mmol L^{-1}).

COMPLICATIONS

LONG TERM

- Retinopathy
- Ischaemic heart disease: 2–4 times higher than the general population
- Hypertension: BP increased in 30%–60% of diabetics
- Nephropathy: 30%–40%
- Neuropathy: peripheral and autonomic (autonomic in up to 40%)
- Respiratory disease: poor glycaemic control has been associated with impaired lung function
- Stiff joint syndrome: may cause difficulty with intubation
- Skin: foot ulcers

SHORT TERM

- Hypoglycaemia.
- Hyperglycaemia with metabolic disturbance; may be exacerbated by the 'stress response' during surgery.
- Gastric stasis common, especially with hyperglycaemia and ketoacidosis.

PREOPERATIVE ASSESSMENT

HISTORY

- Diabetes
  - Duration
  - Control
  - Insulin type, quantity and dose timing
- Coexisting disease
  - Nephropathy (mild-severe)
  - Ischaemic heart disease with decreased exercise tolerance

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- Hypertension
- Respiratory compromise
- Peripheral and autonomic neuropathy
- Full drug history

EXAMINATION

Pay attention to cardiac, respiratory and renal disease depending on history. Airway assessment should include the 'prayer sign' indicative of the stiff joint syndrome.

INVESTIGATIONS

- Glycaemic control
  - Blood glucose (preferably fasting)
  - Glycosylated haemoglobin (HbA1C); HbA1C >9% is indicative of poor control, and hyperglycaemia, hypovolaemia and electrolyte abnormalities should be anticipated and corrected preoperatively. HbA1C >7% can be used as a predictor of coronary heart disease. If associated with other risk factors such as a poor exercise tolerance, age over 55, obesity or physical inactivity, perform a 'stress ECG' to look for silent ischaemia. An inability to climb 2 flights of stairs has a positive predictive value of 89% for postoperative cardiopulmonary complications.
- Full blood count.
- Renal function: Urinalysis, plasma urea, creatinine and electrolyte concentration.
- Chest X-ray, if clinically indicated.
- ECG or exercise ECG: silent ischaemia.

AIMS OF PERIOPERATIVE MANAGEMENT

Maintain normoglycaemia. Keep blood glucose within the range 6–11 mmol L^{-1}. Hyperglycaemia causes dehydration, electrolyte disturbance, acidosis, poor tissue perfusion, organ ischaemia, impaired wound healing and increased susceptibility to infection. Cerebral or myocardial ischaemia will be aggravated by hyperglycaemia. Hypoglycaemia may cause cerebral damage.

Coexisting disease is a greater cause of morbidity than the diabetes itself. Cardiovascular drugs including beta-blockers should be continued in the preoperative period, as they may be protective. Fluid and electrolytes should be optimised preoperatively. Autonomic neuropathy may cause cardiovascular instability during anaesthesia.

GENERAL PRINCIPLES OF MANAGEMENT

MAJOR SURGERY

Ideally, admit 24 hours preoperatively. Well-controlled diabetic patients can be safely admitted on the evening before surgery or on the morning of surgery. Long-acting insulins should be omitted the night before surgery. Ideally patients with diabetes should be placed at the beginning of a list. If this is not possible, a sliding scale should be started in the morning.

GLUCOSE

Glucose should be started when calories or fluids are required and cannot be obtained by the enteral route. It is recommended that 5–10 g h^{-1} of glucose is usually sufficient to prevent hypoglycaemia and to provide basal energy requirements. This usually works out at about 100–200 mL per hour of 5% dextrose. For longer-term infusions, 0.9% sodium chloride is also needed to prevent hyponatraemia.

INSULIN

Consult the local hospital guidelines for management of the diabetic patient. The following regime will provide satisfactory management in the absence of any local guidelines. Whatever regime is used, it is important to measure blood glucose regularly (every 1–2 hours) using a point-of-care capillary glucose monitor.

The morning dose of insulin should be omitted if the patient is starved. If the patient is due to have surgery in the afternoon, either (1) set up an infusion of glucose + insulin + potassium in the morning, or (2) if allowed to eat, give half the usual dose of insulin with a light breakfast, then set up an infusion mid-morning when the effects of the insulin are wearing off.

Table 6.4 Typical sliding scale for insulin

Blood glucose (mmol L⁻¹)	Insulin infusion[a] (units h⁻¹)
0–5	0–0.5
6–10	1–2
11–15	2–3
16–20	3–4
>20	Medical intervention needed

[a] Infusion: make up 50 units insulin to 50 mL with saline (1 unit mL⁻¹). No need to add colloid provided the first few millilitres are flushed through the giving set.

Table 6.5 The Alberti regimen

Glucose 10%	500 mL
Insulin (actrapid or equivalent)	15 units
Potassium	10 mmol

- Add together.
- Infuse this at 100 mL h⁻¹.
- Blood glucose is monitored 2 hourly and the insulin content of a bag adjusted up or down by 5 units if the blood glucose falls outside the range 6–11 mmol L⁻¹.

Insulin should be given by an intravenous infusion, following a sliding scale regimen (Table 6.4). IV boluses have too short a half-life and may impair metabolic control. Absorption of subcutaneous insulin is variable, especially in the perioperative period; this route is not recommended for patients undergoing major surgery.

Insulin may be also added to a bag of glucose (the Alberti regimen, see Table 6.5). The usual requirements are 0.25–0.35 units per gram of glucose.

Insulin requirements are increased with steroid therapy, sepsis, liver disease, obesity and during cardiopulmonary bypass.

POTASSIUM

Potassium should be added as required; usually, 20 mmol L⁻¹ of glucose.

MINOR SURGERY/DAY-CASES

These patients commonly present for day-case surgery. Ideally the procedure should be performed in the morning, be minor, be unlikely to cause much pain and have a low incidence of PONV. The patients should have a good understanding of how to alter their insulin dose and good support at home. HbA₁C should be <7%. Facilities must exist to admit patients postoperatively, if necessary.

Glucose and a sliding scale insulin infusion should be started if the fasting glucose is outside the range 6–11 mmol L⁻¹, or if there is any delay to the start of surgery. It may also be needed if the patient suffers with PONV. The infusion should then continue until a normal diet is resumed. Nausea and vomiting may indicate the development of ketoacidaemia.

MONITORING

Both hypoglycaemia and hyperglycaemia are harmful. Under anaesthesia symptoms of hypoglycaemia are masked.

Blood glucose should be checked 0.5–1 hourly preoperatively and in theatre using a capillary blood glucose meter. Periodically confirm with laboratory blood glucose tests for longer procedures. Postoperatively, check hourly until a normal diet is established. Plasma potassium should be monitored 3–4 hourly, or more frequently if clinically indicated.

ANAESTHESIA

No technique has been show to be superior. Regional techniques are preferable to general anaesthesia, as they usually allow a swifter return to normal eating patterns. They may partially decrease the 'stress response' associated with surgery.

TYPE 2 (NON-INSULIN-DEPENDENT DIABETES MELLITUS)

More common than type 1, it is characterized by insulin resistance (hepatic, extrahepatic or both) probably due to decreased stimulation of glycogen synthesis in muscle by insulin, related to impaired glucose transport. Insulin secretion and/or insulin action are thought to be deficient with excessive hepatic glucose production.

The age of onset is variable. It is usually a disease of adults with slow onset. Ketoacidosis is uncommon.

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There is an increased incidence of macrovascular disease, especially peripheral vascular and cardiovascular disease, irrespective of age at diagnosis. Silent myocardial ischaemia is common, particularly if there is poor glycaemic control and in the presence of other risk factors such as obesity, physical inactivity and age >55 years. Nephropathy is common and is associated with cardiovascular disease.

The mainstays of management are diet, exercise and drugs.

- Sulphonylureas (e.g. gliclazide) increase pancreatic β-cell sensitivity to glucose thereby enhancing insulin release. Long-acting drugs (e.g. chlorpropamide) may exacerbate hypoglycaemia during fasting and are now rarely used.
- Biguanides (e.g. metformin) reduce hepatic glucose production and enhance glucose uptake in muscles.
- Thiozolidinediones (e.g. pioglitazone, rosiglitazone) improve peripheral glucose uptake in muscle and fat and inhibit hepatic glucose production. They are not associated with lactic acidosis but may cause hepatotoxicity.
- Prandial glucose regulators (e.g. nateglinide and repaglinide) stimulate release of insulin from the pancreas. They have a fast onset and short duration of action and are therefore taken just before meals. They are less Acarbose inhibits α-glucosidases in the brush border of small intestinal mucosa and thus delays absorption of glucose.
- The 'gliptins' (e.g. sitagliptin and vildagliptin) increase insulin secretion and reduce glucagon secretion.

PREOPERATIVE ASSESSMENT

This should follow the same lines as for type 1 diabetes mellitus.

PERIOPERATIVE MANAGEMENT

These patients are still able to secrete some insulin but are insulin resistant.

Minor surgery/day surgery

Well-controlled, diet-managed patients do not usually need special treatment apart from regular blood glucose monitoring. Those on oral hypoglycaemic agents should continue treatment as normal up to the day before surgery and then omit oral hypoglycaemics on the day of surgery. Ideally, surgery should be undertaken in the morning. If the patient is scheduled for afternoon surgery, regular monitoring of blood glucose should take place. As most patients are allowed fluids up to 2 hours preoperatively, give glucose-containing drinks if the blood glucose decreases. If blood glucose is <11 mmol L^{-1} it is usually sufficient to just monitor the blood glucose. Start a sliding scale infusion of insulin if the patient is an in-patient and the fasting blood glucose is >11 mmol L^{-1}.

The most important feature is careful, frequent monitoring of blood glucose and early corrective measures if the blood glucose goes outside the range 6–11 mmol L^{-1}.

Major surgery

Treat as type 1 diabetic patients. Hyperosmolar, hyperglycaemic, nonketotic coma may occur postoperatively. There is some evidence against the use of Hartmann's solution, but it is unlikely to be deleterious if given slower than 1 L h^{-1}.

ANAESTHESIA

Many present for surgery for complications of their diabetes. Careful management of preexisting medical problems is important. Anaesthesia should cause minimal metabolic disturbance. Regional techniques are usually preferable to general anaesthesia, unless there is severe cardiovascular disturbance. Local hospital guidelines for the management of diabetic patients should be followed if possible.

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HYPERPARATHYROIDISM

There are usually between four and six individual glands located directly behind the thyroid gland. They secrete parathormone (PTH).

PTH increases serum calcium concentrations. It directly increases renal tubular calcium reabsorption and decreases phosphate reabsorption. It indirectly increases calcium concentrations by stimulating osteoclast activity to release calcium from bones, and by increasing calcium absorption from the gut through activation of vitamin D in the kidney. Excess PTH can cause hypercalcaemia, while PTH deficiency causes hypocalcaemia.

Hyperparathyroidism refers to excessive secretion of PTH from the parathyroid glands that may or may not be associated with hypercalcaemia. The aetiological classification is complicated and differentiates primary, secondary and tertiary disease.

PRIMARY HYPERPARATHYROIDISM

Primary hyperparathyroidism is associated with hypercalcaemia due to unstimulated PTH secretion from parathyroid glands caused by:

- Single adenoma (80%) or multiple gland hyperplasia (10%–15%)
- Adenoma (e.g. multiple endocrine neoplasia syndromes MEN 1 or MEN 2a)
- Parathyroid carcinoma (1%–3%)

SECONDARY HYPERPARATHYROIDISM

Secondary hyperparathyroidism is the physiological regulatory response to hypocalcaemia or hyperphosphataemia, e.g. in chronic renal failure. PTH is elevated but serum calcium is low to normal.

TERTIARY HYPERPARATHYROIDISM

Tertiary hyperparathyroidism is a primary dysfunction arising when chronic stimulation leads to the formation of an autonomous adenoma. Serum calcium is elevated.

The hypercalcaemia of malignancy is induced by various mechanisms, among which is the secretion of parathyroid hormone-related protein (PTHrP) with PTH-activity by the tumour. Ectopic PTH secretion is rare.

CLINICAL PRESENTATION

Most of the signs and symptoms of hyperparathyroidism are due to hypercalcaemia, and their severity is related to the level of serum calcium. Patients with mild hypercalcaemia (serum calcium <3.0 mmol/L) are often asymptomatic. Serum calcium concentrations above 4 mmol/L can cause coma and cardiac arrest and require emergency therapy.

SIGNS AND SYMPTOMS

- Fatigue
- Mental symptoms, especially psychosis and depression
- Polyuria, dehydration, polydipsia, renal calculi, nephrocalcinosis, renal failure
- Anorexia, nausea and vomiting, dyspepsia, peptic ulcers, abdominal pain, constipation
- Tachycardia, arrhythmias, hypertension, shortened QT interval, widened T wave

PREOPERATIVE ASSESSMENT

HISTORY

Search for the symptoms and signs of hypercalcaemia. A mnemonic for these is groans (abdominal pain), moans (depression, confusion), bones (bone pain) and stones (kidney stones).

INVESTIGATIONS

- Serum calcium, electrolytes, creatinine, urea
- ECG (shortened QT interval, widened T-wave)

Patients with severe hypercalcaemia may present with hypovolaemia and coma. Emergency treatment

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consists of intravenous mitramycin, hydration, forced diuresis and phosphate repletion; emergency parathyroidectomy.

PERIOPERATIVE MANAGEMENT

Perioperative management for parathyroidectomy will follow the recommendations for thyroid surgery:

- Protect the eyes against accidental opening and mechanical damage
- Reinforced endotracheal tube
- Secure airway connections
- Balanced anaesthesia

A urinary catheter and arterial cannula may be necessary in patients with critically elevated serum calcium concentrations.

A central venous catheter is occasionally requested by the surgeon to draw blood samples for intraoperative PTH measurements to determine whether the overactive glands have been correctly removed.

POSTOPERATIVE COMPLICATIONS

Most complications of parathyroid surgery are similar to those of thyroidectomy.

Oedema of the glottis and pharynx may occasionally follow parathyroid surgery.

The characteristic complication of parathyroidectomy is hypocalcaemia due to excessive resection, or traumatic or ischaemic damage to the remaining glands. Hypocalcaemia increases neuromuscular irritability. Symptoms are parasthesiae in the hands, perioral tingling, carpal spasms, hyperactive tendon reflexes, positive Chovstek and Trousseau's signs, laryngospasm and long QTc with the risk of malignant cardiac arrhythmias. Calcium gluconate or calcium chloride is given intravenously to treat symptomatic hypocalcaemia.

Repeated determinations of serum calcium, phosphate, magnesium and parathyroid hormone will be required for several days postoperatively.

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CROSS-REFERENCES

Thyroidectomy, Chapter 20
Parathyroid surgery, Chapter 20

HYPERTHYROIDISM

Hyperthyroidism is caused by the excessive secretion of thyroid hormones. This occurs most frequently in women between the ages of 20 and 40, often within 6 months postpartum. The F:M gender ratio is 5:1 to 10:1.

The main cause is autoimmune multinodular diffuse enlargement (toxic nodular goitre) caused by thyroid-stimulating immunoglobulins (TSI) that act as 'long-acting thyroid stimulator' (LATS) and thyroid-growth immunoglobulins (TGI) that induce the growth of thyroid follicles.

Other causes of hyperthyroidism are early-stage Hashimoto thyroiditis, choriocarcinoma, TSH-secreting pituitary tumours, and autonomous thyroid adenoma ('hot nodule'). The latter is the most frequent cause in elderly patients. Iatrogenic causes are prolonged treatment with amiodarone or thyroxine overdosing.

Most clinical features are directly related to the effects of T_3 and T_4, but the myxoedema that presents as exophthalmus and pretibial oedema in patients with Graves disease is a subcutaneous deposition of glycosaminoglyans caused by antibody stimulation of the TSH receptor and is unrelated to thyroid hormone action.

Diagnosis is confirmed by elevated serum concentrations of free and total T_4 and T_3 and undetectable serum TSH. However, high levels of thyroxine are seen in clinically euthyroid patients during fasting or during treatment with beta-blockers or glucocorticoids due to reduced conversion of T_4 to T_3 ('high T_4 syndrome'). A high total T_4 level with normal response to TSH stimulation is also seen under

opiate therapy due to the increased concentration of thyroxine binding globulin (TBG).

SIGNS AND SYMPTOMS

GENERAL AND METABOLIC

Nervousness, tremor, mental impairment, heat intolerance, warm moist skin, weight loss despite increased appetite, fatigue, diarrhoea, menstrual disturbances and impaired glucose tolerance. The presentation is different in older patients with constipation, apathy, depression and loss of appetite prevailing.

CARDIOVASCULAR

Tachycardia, systolic hypertension, hyperdynamic circulation, arrhythmias, atrial fibrillation, dyspnoea and congestive heart failure.

NEUROMUSCULAR

Muscular weakness, proximal myopathy, hyperreflexia, nerve entrapment syndromes and increased central neuronal apoptosis.

LABORATORY

Anaemia, thrombocytopaenia and elevated liver enzymes.

Serum catecholamine concentrations are not increased and the signs of sympathetic stimulation are due to thyroid hormone-induced sensitisation and up-regulation of beta-adrenergic receptors. Hyperplasia of the adrenal cortex is frequently observed.

PREOPERATIVE ASSESSMENT

Manifest hyperthyroidism increases the risk of perioperative complications and is a contraindication for elective surgery, with the exception of thyroidectomy as measure of last resort when conservative treatment has failed to control the condition (Table 6.6). Patients should be treated with antithyroid drugs, e.g. carbimazole or propylthiouracil, until euthyroid. Iodide is given to reduce the vascularity of the gland.

Table 6.6 Hyperthyroidism and eligibility for surgery

	Type of surgery	
	Elective	**Emergency**
Subclinical (only suppressed TSH)	+	+
Elevated T_3, T_4, no symptoms	(+)	+
Clinically manifest	–	With supportive therapy and aggressive thyroid suppressive measures

Note: Iodine and substances containing iodine should be avoided in patients with active hyperthyroidism (contrast medium, amiodarone, disinfectants such as iodoform, povidone-iodine).

Symptoms can be alleviated with beta-receptor antagonists.

Patients with manifest hyperthyroidism requiring emergency surgery have a high-risk of perioperative, particularly cardiovascular, complications and require special care.

HISTORY AND EXAMINATION

Thyroid hormone status should be evaluated in patients with goitre. Goitre alone is most often associated with iodine-deficiency hypothyroidism, but may also be present in patients with hyperthyroidism. The patient must be examined for signs and symptoms of increased thyroid function. The neck should be inspected and the presence of stridor on forced inspiration noted. Engorged jugular veins can indicate retrosternal goitre.

INVESTIGATIONS

Patients with suspected hyperthyroidism require determination of T_4, FT_4, T_3 and TSH in addition to routine laboratory data. Elevated hormone levels may exist without clinical signs of hyperthyroidism. Chest and neck X-rays will show the position of the trachea and reveal any compression or deviation caused by goitre. Retrosternal goitre usually does not interfere with intubation even when the

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trachea is displaced. Indirect laryngoscopy is performed preoperatively by many surgeons to document vocal cord function. CT scan and MR scans can reveal the magnitude and extent of tracheal stenosis.

PERIOPERATIVE MANAGEMENT

Premedication may be necessary to reduce anxiety. Antithyroid drugs, including beta-adrenergic receptor blockers, should be continued on the day of surgery. The perioperative management of patients undergoing nonthyroid surgery who are euthyroid under medication does not differ in any relevant manner from patients without a thyroid disorder, except for possible difficulties in airway management.

AIRWAY MANAGEMENT

Equipment for a difficult intubation should always be available. Minor tracheal involvement is usually of little importance, but awake fibre-optic intubation or inhalation induction should be considered in patients with severe displacement of the trachea, tilting of the glottis or when the trachea is compressed by a fibrotic Riedel's stroma.

THYROIDECTOMY

Use a reinforced oral tracheal tube. Special endotracheal tubes are available that allow intraoperative localisation of the superior laryngeal and recurrent nerve and monitoring of their integrity. These must be positioned with the sensor area in the correct location.

The use of a laryngeal mask airway has been advocated for thyroid surgery, but the risk of intraoperative dislodgement cannot be ignored, particularly since correction is difficult due to the obstructed access to the patient's head.

The tip of the tube should be advanced beyond the distal edge of tracheal compression and firmly taped in position with all connections secured, as access to the airway is limited when surgery has commenced.

Thyroidectomy can be performed under deep or superficial cervical plexus block, but general anaesthesia is the usual practice. There is no evidence that the choice of anaesthetic agent is important, and none of the newer volatile anaesthetics have been associated with organ dysfunction in hyperthyroid subjects. There is some concern regarding the use of drugs that can precipitate or worsen tachycardia. Among these are anticholinergics, neuromuscular blocking agents with significant vagolytic activity (e.g. pancuronium) and ketamine.

The eyelids should be taped and the eyes protected against mechanical damage, particularly in patients with exophthalmus associated with Graves disease.

The patient is positioned with the neck fully extended, and this may be the source of postoperative discomfort. The surgical site is elevated to reduce venous bleeding, but this position can predispose to venous air embolism. If adrenaline-containing local anaesthetic solutions are used to infiltrate the wound, the maximum doses should be observed.

Bradycardia and hypotension are complications of carotid sinus manipulation and can be treated with atropine IV if necessary. Infiltration of the area with lidocaine can suppress the glossopharyngeal nerve afferents and prevent recurrence. Persisting tachycardia and hypertension can result from manipulation of the gland.

Vocal cord motility can usually be visualised by direct laryngoscopy directly after extubation. Otherwise have the patient speak and listen for hoarseness or aphonia.

> **BOX 6.1: Management of thyroid surgery in hyperthyroid patients**
>
> - General anaesthesia with balanced technique (avoid anticholinergics)
> - Prepare for difficult intubation
> - Non-kinking reinforced endotracheal tube advanced beyond any stenosis
> - Secure connections
> - Protect eyes
> - Prepare for cardiac responses (brady/tachycardia, hypo/hypertension)
> - Prepare for exacerbated hyperthyroid symptoms in non-euthyroid patients
> - Assess voice after extubation

The management of surgery in hyperthyroid patients is summarised in Box 6.1, and the eligibility of patients for surgery is summarised in Table 6.6.

NONTHYROID SURGERY

HYPERTHYROID PATIENTS

In general, only emergency surgery is performed on patients with manifest hyperthyroidism. Anaesthetic management is designed to control the symptoms and prevent deterioration.

Insert arterial and central venous cannulae. Large-bore venous cannulae should be inserted to allow adequate and rapid fluid replacement. Temperature monitoring is mandatory.

Treatment of the tachycardia with a beta-blocker should begin before induction and continue throughout the entire perioperative period. Cardiac arrhythmias may require treatment with lidocaine.

Regional anaesthesia should be considered whenever feasible. Care is required in treating associated hypotension, since the hyperthyroid patient has an exaggerated sensitivity to adrenergic stimulation. Direct-acting vasoconstrictors such as phenylephrine or metaraminol are preferred to indirectly acting drugs.

A balanced technique is recommended for general anaesthesia with an opioid and either propofol or a volatile anaesthetic. Muscle relaxation when required should be adapted to the muscular weakness of the hyperthyroid patient and neuromuscular monitoring is mandatory. A possible exaggerated response to atropine must be anticipated when reversing neuromuscular blockade. Glycopyrrolate may be a better choice due to its weaker chronotropic effects. Relaxation with rocuronium and reversal with sugammadex might be a suitable alternative. Vasopressors must be used cautiously due to the increased sensitivity to catecholamines.

Fluid infusions must be adequate to replace the loss due to elevated temperature.

POSTOPERATIVE COMPLICATIONS

NERVE INJURY

Nerve injury is a recognised complication of thyroid surgery and can affect the recurrent and the superior laryngeal nerves. The lesion is frequently caused by nerve distension and is reversible within a few days, but permanent damage will result if the nerve is severed.

Unilateral injury to the recurrent laryngeal nerve is well tolerated because abduction of the contralateral vocal cord is not affected, but after bilateral injury both cords remain in a paramedian position with an extremely narrow glottic opening that requires reintubation.

Injury to the superior laryngeal nerve interrupts the function of the cricothyroid muscle and causes a hoarse voice. It also disrupts the sensory input from the larynx above the vocal folds that triggers reflex closure of the glottis and prevents aspiration.

THYROTOXICOSIS (THYROID STORM)

Thyroid storm is the result of the sudden release of thyroid hormones causing the exacerbation of the symptoms of hyperthyroidism, particularly in inadequately treated or untreated hyperthyroid patients. The result is tachycardia, cardiac arrhythmias, hyperthermia, altered consciousness, congestive heart failure, dehydration and shock. The tachycardia and elevated temperature can initially be mistaken for malignant hyperthermia. Thyroid storm can appear intraoperatively, but usually becomes manifest between 6 and 18 hours after surgery. The condition is not common but has a high mortality rate.

The mainstays of therapy are symptomatic support with a beta-blocker to reduce heart rate, high-dose corticosteroids, aggressive intravenous fluid therapy and cooling, and suppression of thyroid hormone synthesis and secretion with sodium iodide and thyrostatic drugs. The long-standing beta-blocker of choice was propranolol, but the shorter-acting esmolol has been recommended to avoid the occasional cardiovascular collapse that has been described with propranolol.

OTHER COMPLICATIONS

Haematoma with compression of the trachea is a postoperative emergency requiring immediate reintubation to prevent asphyxia. The cause is usually a slipped arterial ligature, but some see a connection with

postoperative coughing and thus advocate extubation while the patient is deeply anaesthetised. Opening the wound might relieve the pressure and stitch cutters or clip removers should be immediately available.

Long-standing goitre can cause tracheomalacia that allows the trachea to collapse during inspiration once the supporting surrounding structure is removed. Reintubation or tracheotomy is often required.

Less common surgical complications are tracheal laceration with or without subcutaneous emphysema, tracheo-oesophageal fistula leading to aspiration and recurring pneumonia, pneumothorax and pneumomediastinum. Injury to the phrenic nerve can present as postoperative hypoxaemia or respiratory distress. Accidental removal of the parathyroid glands will lead to postoperative hypocalcaemia with its associated symptoms.

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CROSS-REFERENCES

Difficult airway management, Chapter 26
Thyroidectomy, Chapter 20

HYPOTHYROIDISM

Found in about 5% of the population, and it is endemic in some areas. In patients over 65, the prevalence of manifest hypothyroidism (elevated TSH, low T_4) is 7% and a further 5%–10% have subclinical hypothyroidism with elevated TSH and normal T_3, T_4. About one-third of patients with subclinical hypothyroidism develop manifest disease within four years. Hypothyroidism is usually a primary disease of the thyroid gland and the main causes are iodine-deficiency, chronic autoimmune thyroiditis (Hashimoto), previous radioiodine therapy and thyroidectomy. Overabundant intake of iodine (amiodarone, dietary iodide) or lithium therapy can induce hypothyroidism. Patients with Hashimoto hypothyroidism are at increased risk for concomitant Addison's disease as well as type 1 diabetes mellitus. Secondary hypothyroidism is caused by a lack of TSH due to pituitary or hypothalamic dysfunction, and its diagnosis must be based on the circulating levels of thyroid hormones alone.

SIGNS AND SYMPTOMS

Typical symptoms result from decreased metabolism and include lethargy, hypothermia, intolerance to cold, cool dry skin, coarse features, hoarse voice and brittle hair.

Cardiac output is reduced with bradycardia, reduced stroke volume and hypotension. The baroreceptor reflex is obtunded. Cardiomegaly with pericardial effusion may be present. The myocardium is overly sensitive to the negative inotropic effects of anaesthetics.

The ventilatory response to hypoxia and hypercapnia is decreased. Oedema can reduce diffusion capacity measured by carbon monoxide transfer and can progress to pleural effusions that further reduce ventilatory capacity.

Lethargy, depression, confusion, ataxia, myalgia, delayed relaxation of deep tendon reflexes (Woltman sign) and increased sensitivity to the respiratory depressive effects of opioids.

Anaemia (association of Hashimoto thyroiditis with pernicious anaemia) and hyponatraemia.

Loss of appetite, constipation, paralytic ileus and ascites.

The most severe manifestation of hypothyroidism is myxoedema coma (not identical with myxoedema as a symptom) with impaired consciousness, myopathy, hypothermia, hypoglycaemia, hypotension, hyponatraemia and hypoventilation.

Mild hypothyroidism can prolong the recovery period, and in the course of serious illness or major surgery, even subclinical hypothyroidism can decompensate to myxoedema coma with a mortality rate of up to 80%. On the other hand, subclinical hypothyroidism appears to have a protective effect. Elderly males with high TSH and low normal FT_4 have a lower overall mortality rate and subclinical hypothyroidism presenting only with elevated TSH but normal serum concentrations of T_3 and T_4 does not appear to be a risk factor for minor to moderately invasive surgery.

Table 6.7 Hypothyroidism and eligibility for surgery

	Type of surgery	
	Elective	**Emergency**
Subclinical (only elevated TSH)	+	+
Low T_3, T_4	(+)	+
Clinically manifest	–	With supportive therapy, intravenous T_3, glucocorticoids

PREOPERATIVE ASSESSMENT

Due to its high prevalence, an active search for signs and symptoms of hypothyroidism is indicated. Elective surgery is not advisable in patients with symptomatic hypothyroidism (Table 6.7). These patients should be treated with oral thyroxine, ideally until serum T_3 and T_4 levels are normal and TSH is no longer elevated. Cortisol substitution will be required for concomitant adrenal cortex dysfunction.

Patients with severe symptomatic hypothyroidism and emergency surgery can be treated with intravenous T_3 and glucocorticoids in an ICU under full monitoring. Myocardial ischaemia and congestive heart failure are the most serious complications of this therapy.

INVESTIGATIONS

- Thyroid parameters
- Full blood count, urea and electrolytes.
- ECG (low voltage [pericardial effusion] and pathological T-waves)
- Chest X-ray (cardiomegaly, pulmonary vascular congestion, pleural effusion)
- X-rays of neck and thoracic inlet in patients with goitre

PERIOPERATIVE MANAGEMENT

Patients who are euthyroid under thyroxine substitution therapy are no different than patients without thyroid dysfunction.

The untreated or insufficiently treated patient presents a high perioperative risk, in part due to the frequently coexisting adrenocortical insufficiency. A dose of glucocorticoid should be administered as a pragmatic prophylactic measure.

The dose of the oral premedication should take into account the increased CNS sensitivity with greater risk of respiratory depression.

Blood pressure and ECG must be monitored closely during induction and continued into the postoperative period. Insertion of an arterial cannula under local anaesthesia before induction should be considered. A central venous catheter can be useful in all but very minor procedures, and cardiac output monitoring should be considered for major surgery. Temperature monitoring is mandatory, since hypothyroid patients are at risk of hypothermia.

Intravenous induction agents must be given in small doses at larger intervals, since intravascular volume is reduced and cardiac output decreased. Rapidly injecting a standard dose will critically suppress the impaired myocardial function and the baroreceptor reflex with severe consequences. Thiopental is probably preferable to propofol, but etomidate should be seriously considered for intravenous induction. The problems inherent in intravenous induction can be avoided by inhalational induction with sevoflurane.

The airway should be secured and ventilation controlled since the respiratory responses to hypoxia and hypercapnia are reduced. Forced air heating and infusion warmers should be used to prevent intraoperative heat losses.

Recovery can be delayed and a prolonged period of postoperative ventilation may be necessary due to the slow elimination of the anaesthetic drugs. The patients should be cared for in ICU and monitored for a worsening of their condition and the occurrence of myxoedema coma.

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IATROGENIC ADRENOCORTICAL SUPPRESSION

Iatrogenic adrenocortical suppression is commonly the result of glucocorticoid hormone therapy. Glucocorticoids are prescribed for a wide variety of diseases. Perioperative complications can arise depending on steroid dose and duration of therapy.

PATHOPHYSIOLOGY

Glucocorticoids administered in higher doses and over a prolonged period suppress cortisol secretion from the adrenal cortex and cause involution of the gland. The mechanism is activation of the negative cortisol feedback loop with suppression of CRH secretion from the hypothalmus and ACTH from the pituitary.

Aside from the complications associated with hypercortisolism, patients with glucocorticoid therapy are asymptomatic. Infection, stress or trauma place increased physiological demands on cortisol secretion. The patient with steroid-suppressed adrenocortical function is unable to respond adequately and can develop cardiovascular symptoms if the glucocorticoid dose is not suitably adapted. In analogy to the patient with endogenous Cushing syndrome undergoing adrenalectomy, the patient with glucocorticoid therapy is at risk of developing an Addisonian crisis if the steroid therapy is terminated or not increased adequately to meet the physiological demands in the perioperative period.

PREOPERATIVE ASSESSMENT

Determine the indication for glucocorticoid therapy, dose and duration of administration. The indication may be highly relevant, e.g. a severe autoimmune disease such as systemic lupus erythematosus or rheumatoid arthritis with atlanto-occipital subluxation.

The degree of adrenal suppression correlates with dose and duration of the glucocorticoid therapy. This is difficult to assess and is usually not attempted prior to surgery. Confusion exists with regard to the duration of administration and dose of glucocorticoid that suppresses adrenal cortical function to an extent relevant to surgery and anaesthesia. In general, short-term use (less than 1 month) and doses of less than the equivalent of 7.5 mg prednisolone per day carry little risk of suppressing the hypothalamic–pituitary–adrenal axis (Table 6.8). Suppression may occur after topical, oral, parenteral, nebulised or inhaled preparations, but less consistently.

The basic perioperative management of patients with possible iatrogenic adrenocortical suppression but without symptoms of hypercortisolism is identical

Table 6.8 Potency of adrenocortical hormones compared to cortisol

Steroid	Glucocorticoid	Mineralocorticoid
Cortisol (hydrocortisone)	1	1
Prednisolone	4	0.7
Dexamethasone	40	2
Aldosterone	0.1	400
Fludrocortisone	10	400

Table 6.9 Steroid treatment regimens

Patients on long-term steroid therapy (prednisolone equivalent dose)		
<10 mg day^{-1}	Assume normal HPA response	Usual daily steroid dose; additional cover not required
>10 mg day^{-1}	Minor surgery	Usual daily steroid dose plus 25 mg hydrocortisone at induction or
		Twice the usual daily steroid dose
	Moderate surgery	Usual daily steroid dose plus 25 mg hydrocortisone at induction plus 100 mg per day for 24 h
	Major surgery	Usual daily dose plus 25 mg hydrocortisone at induction plus 100 mg per day for 48–72 h
High dose immunosupression	Usual immunosuppressive dose in perioperative period	
Patients who have stopped taking steroids		
<3 months		Treat as if on steroids
>3 months		No perioperative steroids necessary
Abbreviation: HPA = hypothalamo-pituitary-adrenal.		

to that for patients with adrenocortical insufficiency on long-term corticoid substitution therapy.

Patients with high-dose glucocorticoid therapy exhibiting signs and symptoms of iatrogenic Cushing syndrome must be treated accordingly. Glucocorticoid therapy must also be continued and adapted to the stress situation in these patients as described in Table 6.9.

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CROSS-REFERENCES

Asthma, Chapter 1
Rheumatoid disease, Chapter 8
Sarcoidosis, Chapter 1
Adrenocorticoid insufficiency, Chapter 6

MUSCULAR DYSTROPHIES

The muscular dystrophies are a group of genetically determined primary degenerative myopathies. They are best classified by their mode of inheritance.

- X-linked:
  - Duchenne (most common and most severe)
  - Becker's
- Autosomal recessive:
  - Limb-girdle
  - Childhood
  - Congenital (? associated with arthrogryposis)
- Autosomal dominant:
  - Facioscapulohumeral
  - Oculopharyngeal

All demonstrate atrophy and weakness of muscle to differing degrees. The onset and groups of muscles involved varies according to the specific dystrophy. Their names often define the muscles involved. Involvement of organs other than muscles is uncommon except in Duchenne muscular dystrophy, which is by far the most common and severe.

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DUCHENNE MUSCULAR DYSTROPHY

Striated, smooth and cardiac muscle fibres may be affected.

Respiratory failure is common due to:

- Muscle weakness.
- Oropharyngeal muscle weakness allowing repeated aspiration.
- Spinal deformities, causing restrictive lung disease.

Obstructive cardiomyopathy occurs, but cardiac failure is often masked due to immobility. Arrhythmias are common; tachycardia and ventricular fibrillation have been reported on induction. Severe bradycardia may occur in the facioscapulohumeral variant. There is a particular ECG pattern with Duchenne dystrophy, namely sinus tachycardia, tall R wave in V1, deep Q wave in the lateral leads and a short P–R interval.

Hypomotility of the gastrointestinal tract and weak pharyngeal muscles predispose to aspiration. Acute gastric dilatation has been reported.

In the musculoskeletal system, pseudohypertrophy of affected muscles occurs and contractures can be problematic. Kyphoscoliosis occurs early on in the disease and further diminishes respiratory reserve. There may be an association with malignant hyperthermia; a malignant-hyperthermia (MH)-like syndrome has been reported following suxamethonium and halothane.

PREOPERATIVE ASSESSMENT

HISTORY

- Review of respiratory function
- Previous anaesthetic history (MH)
- Swallowing difficulties

INVESTIGATIONS

- Respiratory function tests
- Arterial blood gases
- ECG
- Echocardiography if significant cardiovascular (CVS) disease
- Chest X-ray (aspiration, cardiac failure)

PREMEDICATION

- Avoid respiratory depressants
- Acid aspiration prophylaxis and at least 6 h starvation
- If positive history of MH-type reaction, use nontriggering anaesthetic agents

PERIOPERATIVE MANAGEMENT

MONITORING

- AAGBI minimal montoring
- Temperature
- Nerve stimulator

INDUCTION AND MAINTENANCE

Positioning may be difficult due to contractures and kyphoscoliosis.

The association with MH is unproven. Suxamethonium has been associated with hyperkalaemia, cardiac arrest, muscle rigidity and rhabdomyolysis and should be avoided. If there is significant CVS disease, then minimal inhalational agent should be used with opioids. TIVA provides a safe alternative. Sensitivity to nondepolarizing muscle relaxants has been reported; small doses are advised with continued neuromuscular monitoring. Watch $ETCO_2$, ECG and temperature for early signs of MH, and have dantrolene available in theatre.

Local or regional techniques will avoid the risks of general anaesthesia, but may be difficult due to contractures and kyphoscoliosis.

A nasogastric tube should be passed as a precaution against gastric dilatation.

POSTOPERATIVE MANAGEMENT

- Observation on ICU/HDU for at least 24 h.
- Ventilate prophylactically if any doubt about respiratory function.
- Physiotherapy will reduce postoperative respiratory complications.
- Acute gastric dilatation occurs up to 48 h postoperatively, so leave nasogastric tube in situ.

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CROSS-REFERENCE

Cardiomyopathy, Chapter 2

MYOTONIA

A myotonic response in a muscle is where there is a sustained contraction of the muscle which persists after the cessation of voluntary effort or stimulation. It is an abnormality of the muscle itself and not of the neuromuscular junction. It appears in three hereditary syndromes, all of which are of autosomal dominant inheritance:

- Dystrophia myotonica
- Myotonia congenita
- Paramyotonia

The latter two are essentially benign myotonic disorders of skeletal muscle only, which do not shorten life. Dystrophia myotonica (myotonic muscular dystrophy or myotonia atrophica) is a form of muscular dystrophy with myotonic symptoms which precede atrophy and weakness. However, atrophy and weakness, particularly of facial, sternomastoid and distal muscles, are the major complaints. Incidence is 1 in 20,000, with onset between the second and fourth decades. The diagnosis is often made late in the clinical course.

Respiratory failure is common due to:

- Muscle weakness and myotonia
- Central nervous system-mediated respiratory failure
- Oropharyngeal muscle weakness allowing repeated aspiration

There is a reduced response to carbon dioxide.

Smooth muscle involvement in the gut leads to difficulty in swallowing and decreased gastric motility. Both of these predispose to aspiration. There is a high incidence of gallstones.

Presenile cataracts can be the earliest presenting feature.

In the cardiovascular system, rhythm and conduction abnormalities both occur; first-degree heart block is the most common, leading to Stokes–Adams attacks. Cardiomyopathy has been noted and arterial pressure is usually low but rises with worsening congestive heart failure. Cor pulmonale may occur due to respiratory failure.

Abnormal glucose tolerance tests are common.

PREOPERATIVE ASSESSMENT

HISTORY

- Review of respiratory disease
- Swallowing difficulties
- Cardiovascular history (pacemaker for heart block?)
- Drugs for myotonia: quinine, procainamide, phenytoin, steroids

INVESTIGATIONS

- Respiratory function tests and arterial blood gases
- Chest X-ray (bronchiectasis/infection from aspiration)
- Fluoroscopy will detect diaphragmatic myotonia
- ECG and 24 h tape if rhythm disorder suspected
- Echocardiography if significant cardiovascular system (CVS) involvement

PREMEDICATION

- Avoid respiratory depressants.
- Acid aspiration prophylaxis is advisable.
- Intravenous potassium supplementation may make myotonia worse.

PERIOPERATIVE MANAGEMENT

Routine AAGBI monitoring

- Arterial line for pressure and blood gas monitoring
- Invasive CVS monitoring is advisable if there is significant CVS impairment
- Peripheral nerve stimulator (note that this may give false sense of security regarding muscle power)
- Temperature

INDUCTION AND MAINTENANCE

Cardiovascular and respiratory depression may be profound at induction. Minimal dose of induction agent should be used. Inhalational induction may be preferable.

IPPV is usually required and tracheal intubation will protect the airway. Due to muscle atrophy, intubation can usually be performed without muscle relaxation. Avoid suxamethonium as widespread myotonia may occur, making intubation very difficult. Short-acting nondepolarizing muscle relaxants may provide relaxation but often do not; use minimal doses with close monitoring. Reversal of nondepolarizing block with neostigmine may increase myotonia; therefore, it is safest to allow the block to wear off spontaneously. Opioids should be restricted due to respiratory depression.

Normothermia should be maintained to decrease postoperative shivering, which will increase myotonia.

Myotonia may occur with diathermy and surgical handling. This will be refractory to neuromuscular blockade and both regional and peripheral nerve blockade. Myotonia may be treated with intravenous procainamide (note: heart block) or phenytoin. Intravenous regional anaesthesia or direct infiltration of the muscle with local anaesthetic may reduce the myotonia.

REGIONAL TECHNIQUES

These avoid general anaesthesia and its complications; unfortunately, myotonia is not abolished and paralysis of the abdominal muscles may precipitate respiratory failure. Epidural block may be helpful for pain relief, particularly after upper abdominal surgery, and avoid opioids postoperatively.

Local anaesthetic injected directly into the muscle will relieve myotonia and may be used at the surgical site.

POSTOPERATIVE MANAGEMENT

- Patients should be closely monitored in the ICU/HDU.
- Postoperative ventilation is advisable.
- Controlled oxygen therapy should be used in those with chronic hypoxic drive.
- Early physiotherapy.
- Tracheostomy may be required if bronchial secretions are troublesome.
- ECG monitoring should be continued, as arrhythmias and sudden death have been reported.

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CROSS-REFERENCES

PITUITARY DISORDERS AND HYPOPITUITARISM

The pituitary gland or hypophysis is two separate organs; the glandular anterior pituitary derived from Rathke's pouch, an invagination of the oral ectoderm, and the posterior pituitary, an extension of the hypothalamus. Hormones of the anterior pituitary are synthesized and stored in specific pituitary cells,

while the posterior pituitary stores and releases hormones that are synthesized in the hypothalamus and transported through the pituitary stalk.

PATHOPHYSIOLOGY

Pituitary dysfunction refers to either hypersecretion or deficiency of one or several pituitary hormones due to a congenital defect or to lesions in the hypothalamus, the pituitary stalk or the pituitary itself. Clinical features depend on the hormones involved and on the degree of disruption. Patients presenting with symptoms attributable to inappropriate secretion of one pituitary hormone should be evaluated carefully with regard to potential dysfunction of other endocrine systems under pituitary control.

The term panhypopituitarism is used when both the anterior and posterior pituitary are affected.

Hypopituitarism is a common sequel of pituitary surgery, but can also be due to pressure from a primary pituitary tumour. Other causes include brain surgery, postpartum pituitary necrosis (Sheehan syndrome), extrasellar tumours, infection, hypoperfusion or radiotherapy. Hypopituitarism is a common and clinically relevant complication of traumatic brain injury and should be actively searched for in these patients. Diagnosis may be confirmed by circulating hormone levels or by functional tests such as lack of response of GH to hypoglycaemia, of ACTH to corticotropin releasing hormone (CRH), of TSH to thyrotrophin releasing hormone (TRH), or alleviation of polyuria by desmopressin. Hyperpituitarism is nearly always due to tumours or hyperplasia of the secretory cells.

PREOPERATIVE ASSESSMENT

Patients with pituitary tumours or reasons to suspect pituitary dysfunction must be evaluated with regard to endocrine function. Measure serum concentrations of ACTH, cortisol, TSH and thyroid hormones. Patients with hypopituitarism due to intracranial tumours or head injuries must be evaluated with regard to accompanying conditions such as elevated intracranial pressure. Chart visual fields since the optic nerves pass close to the pituitary and can suffer preoperative damage.

PERIOPERATIVE MANAGEMENT

Patients with pituitary tumours, but with confirmed normal endocrine function and without accompanying problems such as raised intracranial pressure require no special treatment, unless they are scheduled for hypophysectomy.

ANTERIOR PITUITARY

Anterior pituitary secretion is regulated by hypothalamic releasing hormones or inhibitory factors. The main hormones of the anterior pituitary are ACTH, TSH, GH, prolactin, FSH, LH, beta-endorphin and MSH (co-secreted with ACTH). Any or all of the anterior pituitary hormones can be affected by pituitary dysfunction but those with the greatest direct relevance for the anaesthetist are ACTH, TSH and GH.

Thyroid-stimulating hormone

TSH is the principal regulator of thyroid function. Its release is controlled by hypothalamic hormones; stimulated by thyrotropin-releasing hormone (TRH), inhibited by somatostatin and modulated by negative feedback from thyroid hormones. TSH levels will therefore be high in patients with primary hypothyroidism and low in patients with primary hyperthyroidism.

Isolated elevated TSH is rare and can be due to a thyrotropic adenoma (thyroid hormones normal), congenital thyroid hormone resistance (thyroid hormones elevated) or combined endocrine deficiency.

Low TSH levels due to pituitary dysfunction produce symptoms and signs of hypothyroidism with low serum thyroxine (T_4) and a blunted TSH response to TRH stimulation. Low TSH levels may also be caused by corticosteroid medication, TSH receptor antibodies, disturbed day-night rhythm (e.g. shift workers) or endogenous depression.

Adrenocorticotropin (ACTH)

ACTH stimulates cortisol and aldosterone secretion from the adrenal cortex. Its release is stimulated by hypothalamic corticotropin-releasing hormone (CRH) and suppressed by the negative feedback

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action of glucocorticoids. Benzodiazepines and opioids reduce CRH secretion.

Pituitary hypersecretion of ACTH from a corticotropic adenoma induces Cushing disease. The nonresponsiveness of the adenoma to negative glucocorticoid feedback control is the rationale behind the dexamethasone suppression test used to differentiate pituitary hypersecretion from other causes of Cushing syndrome. Nonsuppressible ACTH secretion is also seen in paraneoplastic syndromes particularly associated with pancreatic carcinoma and small-cell lung tumours.

ACTH deficiency of either hypothalamic or hypophyseal origin produces cortisol hyposecretion. Aldosterone secretion will usually be normal, since it is also controlled by other pathways. Serum cortisol concentrations will be low but will usually respond to synacthen stimulation in primary hypopituitarism, and usually to CRH stimulation in hypothalamic dysfunction. The clinical picture is that of cortisol deficiency.

Growth hormone (GH)

GH (somatotropin) is secreted by the somatotrophic cells of the anterior pituitary. It stimulates the production of insulin-like growth factor-1 (IGF-1) in the liver. Regulation of GH secretion is complex and under both stimulatory and inhibitory control. Secretion is stimulated by GHRH (growth hormone releasing hormone) and ghrelin as well as androgens during puberty. Dopaminergic mechanisms contribute to the regulation of GHRH secretion; L-DOPA increases and bromocriptine suppresses secretion. GH secretion is inhibited by the hypothalamic hormone somatostatin and is also under negative feedback control of circulating GH and IGF-1. Hyperglycaemia and glucocorticoids also inhibit GH secretion. To illustrate the complex interactions, hypoglycaemia, the standard clinical test of somatotroph function, does not directly stimulate GH release but acts indirectly by inhibiting somatostatin and thus removing its inhibitory effect on GH secretion.

Patients with untreated GH deficiency during childhood are of short stature but otherwise normally proportioned (pituitary dwarfism). Adult-onset GH deficiency is rare and associated with a wide variety of effects including increased cardiovascular morbidity and mortality. The effects of most immediate interest to the anaesthetist are insulin resistance, diastolic cardiac dysfunction and increased levels of fibrinogen and plasminogen activator inhibitor.

Excessive GH secretion is usually due to an adenoma of the somatotroph cells or, in rare cases, from an ectopic tumour. The adenoma can cause headaches, impaired vision and deficiencies of other pituitary hormones in late stages, but its primary clinical relevance is due to the high levels of GH. In the rare instance of a somatotroph adenoma occurring in childhood, pituitary gigantism results. Somatotrophic adenomas in adults cause acromegaly. Self-administration of excessive doses of GH over a long period can also cause similar symptoms.

POSTERIOR PITUITARY

The hormones of the posterior pituitary or neurohypophysis, vasopressin (AVP or antidiuretic hormone [ADH]) and oxytocin, are synthesized in the hypothalamus, transported along the pituitary stalk and stored in the posterior pituitary until they are secreted.

Vasopressin – antidiuretic hormone

AVP is the more relevant to anaesthetic practice due to its effects on blood pressure and fluid homeostasis. It acts through V2 receptors on the collecting duct cells in the kidney tubules to facilitate water reabsorption in the thick ascending limb of the loop of Henle.

Vasopressin deficiency causes diabetes insipidus and compromises intraoperative blood pressure control, particularly during epidural or spinal anaesthesia and in patients with hypovolaemia. The hypersecretion seen in the syndrome of inappropriate antidiuretic hormone secretion (SIADH) manifests itself in hyponatraemia.

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CROSS-REFERENCES

Adrenocortical insufficiency, Chapter 6
Anaesthesia for trans-sphenoidal hypophysectomy, Chapter 14
Conn syndrome, Chapter 6
Cushing syndrome, Chapter 6
Diabetes insipidus, Chapter 6
Diabetes mellitus (IDDM), Chapter 6
Hypertension, Chapter 1
Hyperthyroidism, Chapter 6
Hypothyroidism, Chapter 6

UNCOMMON ENDOCRINE TUMOURS

The acronym 'APUD' (amine precursor uptake and decarboxylation) describes groups of cells that are capable of the synthesis of biologically active amines and peptides. The clinical manifestation of each tumour is characterised by overproduction of particular hormones and/or peptides. APUD cells may be found in the pituitary gland, adrenal medulla, peripheral autonomic ganglia, gastrointestinal tract, pancreas, lung, gonads and thymus. Tumours may occur as part of the multiple endocrine neoplasia (MEN) syndromes.

GASTRINOMA

Gastrinoma is a very rare gastrin-producing tumour with an incidence of 1 per million per year. The second most common functional islet cell tumour, it presents with peptic ulcer disease; the Zollinger–Ellison syndrome is gastric acid hypersecretion with recurrent peptic ulceration and diarrhoea. Between 20 and 60% of patients with gastrinoma have coexisting MEN1; 60% of gastrinomas are malignant, and 50% of patients have metastases at diagnosis.

PERIOPERATIVE MANAGEMENT

- Proton pump inhibitors, e.g. omeprazole
- H2 antagonists, e.g. ranitidine, cimetidine
- Octreotide (octapeptide analogue of somatostatin)

Surgery (resection of tumour) is considered if medical therapy fails. Pre- and intraoperative tumour localisation is important. Patients with MEN1 may have multiple tumours with a tendency towards duodenal wall location.

Acute presentation may occur with gastrointestinal bleeding and perforation. Diarrhoea leads to fluid volume depletion, electrolyte disturbance and dysrythmias. Invasive cardiovascular monitoring is required and surgery is prolonged.

Continue postoperative cardiovascular and biochemical monitoring on HDU or ICU. Mortality is high in emergency procedures.

VIPomas

These extremely rare tumours release *v*asoactive *i*ntestinal *p*eptide. Patients present with severe, large volume diarrhoea, hypokalaemia and metabolic acidosis. The WDHA syndrome refers to the association of *w*atery *d*iarrhoea, *h*ypokalaemia and *a*chlorhydria.

PERIOPERATIVE MANAGEMENT

- Symptom control with octreotide and correction of electrolyte abnormalities.
- Aggressive management of fluid volume status and metabolic acidosis.
- Invasive cardiovascular monitoring.
- Frequent blood sampling for pH and electrolytes.

INSULINOMA

This is the most common type of islet cell tumour; 90% are benign, intrapancreatic, small and solitary. Up to 10% are multiple, associated with MEN. Incidence is 4 per million per year.

Tumours secrete insulin or proinsulin causing hypoglycaemia. Treatment includes:

- *Diazoxide* – Inhibits release of insulin from tumour
- *Diuretics* – Treat oedema associated with use of diazoxide
- *Glucagon infusion* – Maintainance of blood sugar
- *Beta blockers* – Blood pressure control
- *Calcium channel blockers* – Blood pressure control
- *Cytotoxic drugs* – Antitumour effect

PREOPERATIVE ASSESSMENT

Assess for other endocrine tumours. Patients may be obese and hypertensive. Preoperative tumour localisation with CT, MR, ultrasound, angiography and selective venous hormone sampling is important to permit selective removal of the adenoma and avoid blind subtotal pancreatectomy. Palpation of the pancreas at surgery may produce sudden hormone release.

PERIOPERATIVE MANAGEMENT

- Generous premedication
- Prepare for major laparotomy.
- Direct cardiovascular monitoring.
- Consider cardiac output monitoring, e.g. TOE.

- Continuous or intermittent glucose, insulin and potassium sampling.
- Expect wide fluctuations in blood glucose.
- 50% glucose available to treat hypoglycaemic response to tumour manipulation.
- Continue glucose and insulin monitoring postoperatively.

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CROSS-REFERENCES

Phaeochromocytoma, Chapter 10
Intraoperative hypertension, Chapter 30

7

The blood

ALASTAIR DUNCAN AND SANTOSH PATEL

ANAEMIA

ALASTAIR DUNCAN

Anaemia is a common condition with a prevalence of >10% in those aged over 65 years. Preoperative anaemia and allogeneic blood transfusion (ABT) are independent risk factors for adverse outcomes following surgery.

The World Health Organisation (WHO) defines anaemia based on haemoglobin concentration (Hb) (Table 7.1).

CAUSES

- Impaired red blood cell production
- Increased red blood cell breakdown
- Red blood cell loss

Morphological classification of anaemia is summarised in Table 7.2.

Functional iron deficiency, which results from the disruption of iron absorption pathways by hepcidin, is well recognised. Iron transport to the bone marrow is impaired as iron stores are sequestered in ferritin. Inflammation activates hepcidin and patients previously diagnosed with 'anaemia of chronic disease' are now recognised as having iron deficiency anaemia.

PATHOPHYSIOLOGY

Tissue oxygen delivery (DO_2) is determined by the arterial oxygen content (CaO_2) and cardiac output (CO).

$$DO_2 = \left[(1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)\right] \times CO$$

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Table 7.1 WHO definition of anaemia

Population group	Haemoglobin concentration (gL⁻¹)
Adult men	<130
Adult women (non-pregnant)	<120
Adult women (pregnant)	<110
Children aged 12–14	<120
Children aged 5–11	<115
Children aged 6 months – 6 years	<110

Oxygen carrying capacity is determined by the haemoglobin concentration. In anaemia, this is reduced and the consequence is a reduction in DO_2.

A number of compensatory mechanisms are initiated to address this.

In the acute phase:

- Increase in cardiac output secondary to reduced blood viscosity
- Redistribution of cardiac output to organs with high oxygen demands

In the chronic phase:

- An increase in 2,3-diphosphoglycerate (2,3-DPG) shifts the oxyhaemoglobin dissociation curve to the right, promoting release of oxygen to the tissues.

CLINICAL FEATURES

- Fatigue
- Dyspnoea (usually associated with exertion)
- Tachycardia
- Lightheadedness

- Angina (in those with coronary artery disease)
- Symptoms and signs of heart failure

These clinical features may be masked by compensatory mechanisms and not be present until the haemoglobin concentration has reduced by around a third (<90–100 gL⁻¹).

PREOPERATIVE ASSESSMENT

NICE guidelines concerning preoperative testing advise that if anaemia is present on full blood count, further tests should be performed to include a blood film, serum iron studies, measurement of vitamin B12/folate, renal and thyroid function.

It is important to determine the impact of anaemia on the patient's functional capacity and physical performance.

MANAGEMENT

PREOPERATIVE

The severity and cause of anaemia, urgency of surgery and physiological reserve of the patient must be taken into account. If time permits, treat the cause and optimise haemoglobin concentration prior to surgery.

Replacing haematinics

- Iron deficiency is the most common cause of anaemia.
- Oral iron can be effective at least four weeks before surgery and continued into the postoperative period. This relies on iron absorption pathways functioning normally.

Table 7.2 Morphological classification of anaemia

Microcytic (MCV <77fL) Hypochromic MCH <27pg)	Normocytic (MCV 77–95fL) Normochromic (MCH 27–32pg)	Macrocytic (MCV >95fL)
Iron deficiency (IDA)	Haemolysis	Vitamin B_{12} deficiency
Thalassaemia	Haemorrhage	Folate deficiency
	Bone marrow failure	Alcohol excess
	Renal failure	
	Hypothyroidism	
	Hypopituitarism	

Abbreviations: MCH, mean corpuscular haemoglobin; MCV, mean corpuscular volume.

- Intravenous (IV) iron may be considered if a diagnosis of functional anaemia has been made or time between identifying anaemia and surgery is too short for oral iron to be effective.
- Vitamin B_{12} or folate replacement can take up to eight weeks before haemoglobin concentration normalises.

Stimulating erythropoiesis

- Erythropoietin should be reserved for patients with anaemia due to renal failure (often require supplementary iron replacement), those who decline blood products or for those with rare blood types due to antibody expression.
- This should be commenced at least three weeks prior to surgery.

Red cell transfusion

- Preoperative transfusion is generally restricted to patients with sickle cell anaemia (target Hb >100 gL^{-1}), those with cardiovascular compromise secondary to anaemia and those undergoing urgent/emergency surgery with significant anaemia.

PERIOPERATIVE AND POSTOPERATIVE

It is important to optimise oxygen delivery. This can be achieved by manipulating cardiac output (pharmacological agents) or oxygen concentration (oxygenation or Hb). Restrictive transfusion strategies have been adopted but when to transfuse remains a contentious issue.

Current recommendations suggest:

- Transfusion should be considered if Hb <80 gL^{-1}.
- Transfusion is usually indicated if Hb <70 gL^{-1}.
- Decision to transfuse should be based on the patient's clinical condition.

The same transfusion triggers can be applied to those with asymptomatic coronary artery disease. Traditional teaching was to aim for higher Hb in those with acute coronary syndromes, but emerging evidence does not appear to support this. The decision to transfuse in these patients requires an individualised approach.

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BLOOD LOSS AND REPLACEMENT

ALASTAIR DUNCAN AND SANTOSH PATEL

Preoperative anaemia and allogeneic blood transfusion are independent risk factors for adverse patient outcome following surgery. In 2010, it was recommended that all World Health Organisation (WHO) States implement a patient blood management (PBM) strategy to address this issue.

PBM is defined as an evidence-based, patient-centred, multidisciplinary, multimodal initiative aimed to improve clinical outcomes. This approach is individualised and designed around the 'three pillars' of PBM, which incorporates medical and surgical tools.

- Optimisation of erythropoiesis.
- Minimise blood loss.
- Optimise tolerance of anaemia.

PREOPERATIVE

ANAEMIA

The identification and correction of preoperative anaemia is the cornerstone of management.

ANTICOAGULANT AND ANTIPLATELET THERAPY

Protocols specifying the appropriate cessation of anticoagulant and antiplatelet therapies should be constructed in collaboration with surgeons, anaesthetists, haematologists, cardiologists and other appropriate members of the multidisciplinary team. The effects of these treatments may require reversal prior to surgery to reduce the risk of blood loss.

AUTOLOGOUS TRANSFUSION TECHNIQUES

Predeposit autologous donation

The patient may donate up to three red cell units preoperatively. The donated blood is subjected to the same processing, testing and traceability requirements as allogeneic blood donations. Iron supplementation and erythropoietin can be considered to reduce the likelihood of anaemia and facilitate maximal donation.

Acute normovolaemic haemodilution

Several units of blood are collected from the patient immediately preoperatively (usually in the operating theatre). Crystalloid fluids are infused to replace this lost circulating volume. The collected blood is then reinfused at the end of surgery once haemostasis is achieved.

The above two techniques are not commonly used in the UK because of:

- Logistical and financial challenges relating to collection and storage
- Wastage of collected blood that cannot be returned to the donor pool
- Limited evidence to support a reduction in exposure to allogeneic blood transfusion

INTRAOPERATIVE

ANAESTHETIC MEASURES

Anaesthetic technique

- Neuraxial anaesthesia reduces blood loss during total hip arthroplasty, fractured hip surgery, peripheral vascular surgery, prostatic surgery and caesarean section when compared to general anaesthesia. An associated reduction in transfusion requirements has not been demonstrated by meta-analyses.
- Propofol TIVA is associated with reduced blood loss in some endoscopic and spinal surgeries.
- IPPV can affect intraoperative blood loss through its effects on haemodynamics and acid-base balance. It is associated with increased blood loss in primary arthroplasty surgery when compared to spontaneous ventilation during general anaesthesia.
- Controlled hypotensive anaesthesia has been associated with reduced rates of transfusion, but the potential detrimental effects of organ hypoperfusion exclude its use in many circumstances.
- Starch-based solutions are implicated in the development of impaired coagulation. Their use in the perioperative setting has diminished significantly over the past 5 years and their availability in the UK is limited.

General measures

- Patient positioning is important to reduce blood loss for certain types of surgery. In the prone position, inferior vena cava compression must be avoided to prevent diversion of venous blood through alternative plexuses, which can result in increased blood loss. Patient positioning is also important during the postoperative period. Elevation of limb or surgical area can reduce bleeding.
- Normothermia and normal acid-base balance are paramount to maintaining haemostasis.
- Measurement of coagulation status with point-of-care testing can prevent inappropriate blood product transfusion.
- Antifibrinolytics have a growing evidence-base supporting their use in several surgical

specialties and trauma management. Tranexamic acid is the agent used most commonly in the UK. It acts by preventing plasminogen from binding to and degrading fibrin. It has been shown to decrease postoperative bleeding in joint replacement, spinal, cardiac, prostatectomy and liver surgery. It has also been proven effective in reducing bleeding and improving survival in patients who are at risk of haemorrhage due to trauma.

- Recombinant actor VIIa has been used to control bleeding in a wide variety of conditions. Although it may reduce or control bleeding, its effects on long-term outcome are not proven. It is ineffective if the patient is hypothermic, acidotic and has severe thrombocytopaenia.

SURGICAL MEASURES

Surgical technique

- Minimally invasive techniques including laparoscopic surgery and those performed in conjunction with interventional radiologists are associated with reduced blood loss.
- If an open technique is performed, meticulous attention to detail is required to minimise surgical blood loss.
- Robotic-assisted radical prostatectomy shows promise in reducing intraoperative blood loss.
- Newer technologies such as the argon beam coagulator and radiofrequency dissecting sealer should be considered.

General measures

- Topical haemostatic agents and fibrin sealants may be considered. Local infiltration with adrenaline is commonly used to reduce blood loss.
- The use of tourniquets is a well-established method for providing the surgeon with a bloodless field. Despite this, the evidence suggests no significant reduction in blood loss in primary knee arthroplasty.

INTRAOPERATIVE CELL SALVAGE (ICS)

A Cochrane Collaboration review demonstrated a 20% reduction in allogeneic blood transfusion when cell salvage was used intraoperatively (primarily cardiac and orthopaedic surgery).

The process involves three distinct stages.

- Collection of blood from the surgical field, which is anticoagulated before being stored in a reservoir.
- Washing and filtering of red blood cells (RBCs) takes place after separation by centrifugation.
- Reinfusion can occur after the salvaged RBCs have been resuspended in normal saline. The haematocrit is between 0.50 and 0.80.

Indications for the use of ICS

- Anticipated blood loss >20% blood volume
- Preoperative anaemia or increased risk for bleeding
- Patients with rare blood groups or antibodies
- Patient refusal of allogeneic blood transfusion

Cautions in the use of ICS

- Bowel contents in the surgical field
- Heparin-induced thrombocytopaenia
- Presence of overwhelming sepsis
- Sickle cell disease
- Malignancy

The risks and benefits for the use of ICS should be assessed on an individual basis. ICS can be adapted to overcome many of these relative contraindications.

- Use of standard suction until bowel contents cleared from surgical field
- Alternative citrate anticoagulant can be used
- Ensuring antibiotic therapy is initiated in the presence of infection
- Leucodepletion filters can be used in cases of malignancy

POSTOPERATIVE

POSTOPERATIVE CELL SALVAGE (PSC)

PCS is primarily used following primary arthroplasty and corrective scoliosis surgery. Blood from wound drains is collected, filtered, and washed prior to reinfusion.

RESTRICTIVE TRANSFUSION STRATEGY

Guidance regarding postoperative transfusion thresholds and the decision when to transfuse exist. The general principle is to avoid unnecessary transfusion by setting individualised thresholds and employing the other components of PBM.

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CROSS-REFERENCES

Anaemia, Chapter 7
Blood transfusion, Chapter 30

DISSEMINATED INTRAVASCULAR COAGULATION

SANTOSH PATEL

Disseminated intravascular coagulation (DIC) involves widespread activation of those haemostatic mechanisms that normally operate locally to halt bleeding from injured vessels. The diagnosis of DIC requires the identification of a bleeding disorder with evidence of fibrinolysis and consumption of platelets and clotting factors. Microvascular thrombosis occurs and may be associated with multiple organ failure.

PATHOPHYSIOLOGY

The normal physiological response to vascular endothelial damage involves generation of a fibrin clot at the site of injury. Thrombus formation is controlled by physiological anticoagulants including protein C and antithrombin, and by the fibrinolytic system. DIC involves widespread activation of intravascular coagulation with microvascular thrombosis resulting in end organ dysfunction from ischaemia, hypoxia and necrosis. Depletion of coagulation factors results from consumption in the thrombosis process. The fibrinolytic system is activated, resulting in generation of fibrin degradation products (FDPs). FDPs interfere with platelet aggregation, fibrin polymerization and thrombin activity. This causes bleeding.

Coagulation and inflammatory pathways are closely linked. Cytokines produce widespread activation of coagulation and suppression of fibrinolysis, partly by stimulating expression of tissue factor on monocytes and endothelial cells. Antithrombin, protein C, and protein S, which act as inhibitors of both coagulation and inflammation, are depleted in several aetiological conditions particularly sepsis.

The trigger for DIC may be vascular damage, resulting in exposure of the blood to subendothelial collagen, and activation of factor XII. The process of coagulation may also be initiated by tissue damage, releasing thromboplastins into the circulation. Table 7.3 lists the common causes and related pathogenic factors of DIC.

CLINICAL FEATURES

DIC is a syndrome resulting from a wide range of clinical conditions. Therefore, the clinical features of its initiating condition should also be sought. Clinical manifestations are the result of organ damage due to intravascular thrombosis. Microvascular thrombosis may cause tissue ischaemia and necrosis, resulting in organ dysfunction that typically affects

Table 7.3 Aetiopathogenesis of DIC

- Infections/inflammatory conditions: sepsis, pancreatitis
  Release of endotoxin in Gram-negative sepsis, synthesis or release of tissue factor, decreased levels of anticoagulants: thrombomodulin, protein C and S, suppression of fibrinolysis
- Obstetric: pre-eclampsia, placental abruption, amniotic fluid embolism, retained products of conception, placenta praevia, intrauterine foetal death
  Depending on aetiology several mechanisms play a role: release of placental tissue factor, endothelial damage, platelet activation, haemolysis, release of pro-inflammatory cytokines
- Malignancy: Myeloproliferative/lymphoproliferative malignancies, metastatic carcinoma
  Pro-inflammatory cytokines, procoagulant synthesis by cancer cells
- Traumatic: polytrauma, cardiopulmonary bypass, burns, fat embolism
  Activation of extrinsic coagulation pathway due to release of thromboplastic factors, release of pro-inflammatory cytokines, inhibition of fibrinolysis
- Intravascular haemolysis: ABO transfusion reaction, snake venom
- Shock of any cause
  Hypoxia, ischaemia
- Severe liver disease
  Impaired clearance of procoagulants, reduced synthesis of clotting factors

the skin, kidneys, lungs and liver. However, bleeding manifestations are more obvious. Acute DIC is a medical emergency and may present with sudden bleeding from several sites, e.g. skin, gastro-intestinal tract, genito-urinary tract, sites of vascular access and surgical incisions. The clinical presentation varies depending upon the aetiology, so that retained products of conception typically cause uterine haemorrhage, while carcinoma is associated with chronic vascular thrombosis. Scoring systems have been used for diagnosis and to grade severity. In many cases, features of the condition are limited to laboratory abnormalities.

INVESTIGATIONS

DIC is a dynamic process and the laboratory results may change rapidly and significantly.

- Thrombocytopaenia: platelet count <100,000 or rapidly declining
- Prolonged clotting times (PT, APTT)
- Presence of fibrin degradation products or positive D-dimer: high sensitivity, low specificity
- Low levels of coagulation inhibitors: AT III, protein C

- Low levels of coagulation factors: Factors V, VIII, X, XIII
- Low fibrinogen levels

A scoring system based on a decrease in platelet count, raised FDP or D-Dimer level, prolonged prothrombin time and lower fibrinogen count has been proposed for DIC diagnosis.

Thromboelastography has been suggested as a means to evaluate and monitor DIC. It can also be useful to predict prognosis by differentiating hyper- and hypocoagulation states. The latter is associated with a poor prognosis.

PREOPERATIVE PREPARATION

Preoperative management should focus on identification and treatment of precipitating cause, identifying the haemostatic abnormalities. Replace coagulation factors to correct abnormal coagulation and stop bleeding and support organ function.

Administration of blood products should be guided by the clinical condition of the patient and the results of laboratory investigations. It is important that a haematologist is involved. Correction of coagulopathy may require platelet concentrate and fresh frozen plasma. Severe depletion of fibrinogen

(blood level <1 g L^{-1}) is an indication for giving cryo-precipitate or fibrinogen concentrate. Anaemia, due to haemorrhage or microangiopathic haemolysis, will require blood transfusion. An adequate supply of blood and blood components must be available before surgery starts.

Antibiotics should be given for infection and hypovolaemia must be corrected. Fluids and blood products should be warmed, as hypothermia will further impair clotting. Surgery intended to remove the cause of the DIC, such as evacuation of retained products of conception, should be delayed no longer than is necessary to correct the coagulopathy to an acceptable level and to ensure cardiovascular stability.

Pharmacological management remains controversial. Low-dose heparin has been used to interrupt the vicious cycle of intravascular coagulation and consumptive coagulopathy, particularly in conditions such as acute promyelocytic leukaemia and metastatic malignancy. It may be useful for the treatment of thrombosis in the presence of sustained DIC. However, control is difficult, bleeding may be exacerbated and heparin is best avoided before surgery. Antithrombin therapy and activated Protein C have been used in selective septic patients with DIC. Antifibrinolytic agents such as Tranexamic acid may be useful in cases with overwhelming fibrinolysis causing severe bleeding. However, their use may potentiate thrombosis formation.

INTRAOPERATIVE MANAGEMENT

Coagulopathy is a contraindication to regional anaesthesia, so general anaesthesia is used. The aim is to prevent hypoxia, maintain adequate perfusion and prevent hypothermia. The choice of anaesthetic agents and monitoring is dictated by the patient's clinical condition and the nature of the surgery. DIC is usually a disease of the critically ill, so controlled ventilation and invasive cardiovascular monitoring are appropriate. Large-bore intravenous lines are necessary to allow the rapid transfusion of blood and blood products. Nasotracheal intubation should be avoided if coagulopathy is significant and care should be exercised during the insertion of nasogastric tubes.

Blood loss should be measured and promptly replaced. Platelet concentrate, fresh frozen plasma and cryoprecipitate or fibrinogen are given according to clinical need and laboratory results. Therapeutic interventions should be evaluated by frequent clinical and laboratory investigations.

POSTOPERATIVE MANAGEMENT

Patients with clinically significant DIC are best managed in ICU and often require continued mechanical ventilation and invasive cardiovascular monitoring and support. Frequent measurement of haematological parameters should continue, with judicious administration of blood and blood products after consultation with a haematologist. Surgery, particularly in obstetric cases, may remove the cause of DIC and result in rapid resolution.

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CROSS-REFERENCES

Blood transfusion, Chapter 30
Thrombosis and embolism, Chapter 30
Serious complications of pregnancy, Chapter 12

ERYTHROCYTOSIS (POLYCYTHAEMIA)

ALASTAIR DUNCAN

Erythrocytosis is defined as an increase in red blood cell mass. It is associated with an increase in haemoglobin concentration (Hb) and haematocrit (Hct). Diagnostic criteria that quantify these measurements have not been standardised, but the values in Table 7.4 are generally accepted.

CAUSES

- Absolute erythrocytosis (increased red cell mass)
  - Primary (polycythemia vera)
  - Secondary
  - Idiopathic
- Apparent erythrocytosis (normal red cell mass)
  - Reduced plasma volume

PRIMARY ERYTHROCYTOSIS

The term polycythaemia vera (PV) will be used when describing primary erythrocytosis. PV has an incidence of 2 per 100,000 population and a median age of presentation of 60 years.

PATHOPHYSIOLOGY

PV is characterised by clonal proliferation of myeloid cells. Over 95% of affected patients exhibit a genetic mutation for the cell signaling enzyme Janus kinase

2 (JAK2). This stimulates overproduction of erythrocytes, platelets and granulocytes. This mutation is not specific for PV, as it is also found in patients with primary thrombocytosis and primary myelofibrosis.

CLINICAL MANIFESTATIONS

The majority of symptoms and signs result from thrombotic episodes secondary to increased blood viscosity. Production of immature platelets with variable function and acquired von Willebrand's disease (vWD) are thought to be behind the bleeding tendency witnessed in these patients.

Associated laboratory findings:

- Thrombocytosis
- Leukocytosis
- Elevated lactate dehydrogenase

Clinical features:

- Hypertension (46%)
- Splenomegaly (36%)
- Pruritus (36%)
- Erythromelalgia (29%)
- Arterial thrombosis (16%)
- Venous thrombosis (7%)
- Haemorrhage (4%)
- Facial plethora
- Hepatomegaly
- Gout

TREATMENT

The aim of treatment is to reduce the risk of thrombosis, haemorrhage and the transformation to acute leukaemia or myelofibrosis. The mainstay of treatment is venesection (aiming for an Hct <0.45) and aspirin. If these treatment measures fail, hydroxyurea or interferon alpha may be considered.

SECONDARY ERYTHROCYTOSIS

The increased red cell mass in secondary erythrocytosis is due to increased erythropoietin production. This may be a compensatory mechanism in response to chronic tissue hypoxia or to inappropriate production by the kidneys.

Table 7.4 Diagnostic criteria

Measurement	Male	Female
Haemoglobin (gL^{-1})	>185	>165
Haematocrit	>0.52	>0.48

Causes include:

- Chronic obstructive pulmonary disease (COPD)
- Obstructive sleep apnoea (OSA)
- Cyanotic heart disease
- Altitude
- Renal artery stenosis
- Renal tumours
- Transplanted kidneys

Treatment consists of venesection and, where possible, management of the underlying condition. Angiotensin converting enzyme inhibitors (ACEi) and angiotensin II receptor blockers (ARB) have shown promise in the treatment of secondary erythrocytosis due to renal transplantation.

IDIOPATHIC ERYTHROCYTOSIS

Idiopathic erythrocytosis is increased red cell mass with no identifiable cause. Patients are more commonly male and over half present with vascular occlusive complications. Treatment relies upon venesection as cytoreductive therapy is contraindicated.

APPARENT ERYTHROCYTOSIS

A reduced plasma volume results in an increased Hct and Hb on laboratory testing. This gives rise to an apparent erythrocytosis. In these cases, the red cell mass is normal.

The precise pathophysiological mechanism is unclear, but there is a recognised association between the disorder and several clinical conditions.

- Obesity
- Hypertension
- Smoking
- Excessive alcohol intake
- Diuretic use

Treatment focuses on lifestyle modification and venesection if required.

PREOPERATIVE MANAGEMENT

- Preoperative correction of Hct and Hb requires liaison with a haematologist.

- Preoperative assessment may identify modifiable lifestyle changes or suboptimal disease management requiring referral to appropriate specialists.
- If the surgery is urgent, isovolaemic haemodilution can be performed. It may be possible to store the venesected blood for future autologous blood transfusion.

PERIOPERATIVE MANAGEMENT

- Ensure low dose aspirin is continued in PV.
- Consider regional anaesthetic techniques, which may reduce the risk of thromboembolic episodes.
- Close monitoring of expected and unexpected blood loss is essential. Regular haematological testing is justified, which may include point-of-care coagulation testing.
- Careful consideration must be given to postoperative thromboembolism prophylaxis.

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GLUCOSE-6-PHOSPHATE DEHYDROGENASE DEFICIENCY

SANTOSH PATEL

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common inherited metabolic disorder of red blood cells (RBCs), affecting over 400 million people worldwide with varying degrees of severity. G6PD governs the rate at which RBCs

consume, utilise and detoxify oxygen. Its deficiency makes RBCs vulnerable to haemolysis. G6PD deficiency will not usually result in complications during or after anaesthesia, provided oxidant agents known to trigger haemolysis are avoided.

PATHOPHYSIOLOGY

Low levels of G6PD result in failure to generate NADPH in the pentose monophosphate shunt for G-6-phosphate oxidation. NADPH maintains reduced glutathione levels to prevent oxidative damage of red blood cells from free radicals. If G6PD activity is low or absent, a red cell membrane is susceptible to damage and haemoglobin can be denatured causing intravascular haemolysis due to oxidative stress. Table 7.5 lists oxidant drugs that may cause haemolysis in patients with G6PD deficiency, though the response is somewhat idiosyncratic. The haemolytic episode begins 2 to 5 days after drug administration.

Table 7.5 Some oxidant drugs capable of triggering haemolysis in patients with G6PD deficiency

Analgesics	Aspirin in high dose
	Phenacetin
	Acetanilide
Sulphonamides	
Antibiotics	Penicillin
	Isoniazid
	Ciprofloxacin
	Chloramphenicol
Antimalarials	Primaquine
	Chloroquine
	Quinine
Miscellaneous drugs	Methylene blue
	Vitamin K
	Nalidixic acid
	Naphthalene
	Quinidine
	Probenecid
	Phenylhydrazine
	Nitrates
	Nitrofurantoin
	Ascorbic acid in high dose

CLINICAL MANIFESTATIONS

The structural gene for G6PD resides on the X chromosome and is therefore inherited as a sex-linked characteristic. G6PD deficiency is most common in hemizygous males but is also seen in homozygous females. Heterozygous females may occasionally show clinical manifestations. There are more than 200 variants of G6PD and the clinical manifestations range from negligible to severe, depending upon the activity of the abnormal G6PD and whether the patient is a heterozygote or a homozygote/hemizygote. Two distinct clinical syndromes result:

- African variant (the gene in this variant is termed A-). The patient is asymptomatic until exposed to oxidant drugs or a severe infection; 11% of African Americans have the A- gene.
- Mediterranean and Oriental groups of variants. The G6PD deficiency is generally more severe and may be a cause of neonatal jaundice. Nevertheless, the patient is usually asymptomatic until a drug or infection precipitates haemolysis. Some individuals can also develop a fulminant haemolytic anaemia after exposure to the fava bean (favism).

ANAESTHETIC MANAGEMENT

Elective surgery should not be performed during a haemolytic crisis. Patients should be informed of risks along with signs and symptoms of an acute haemolytic crisis. Goals of anaesthetic management are avoid drugs known to cause haemolysis (Table 7.5); keep vigilance for haemolysis; treat haemolytic anaemia if it occurs. Minimise perioperative stress and pain. Oxidative stress can also be caused by hypothermia, acidosis, hyperglycaemia, infection, ischaemia and reperfusion.

The classical features of such a crisis include abdominal pain and jaundice. Laboratory investigations may reveal decrease in haemoglobin concentration, an increasing reticulocyte count, presence of Heinz bodies in red blood cells in the peripheral blood film, increased lactic dehydrogenase, reduced haptoglobin levels, negative Coombs test and increased indirect bilirubin. During the intraoperative period,

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clinical signs may be masked. If a haeomolytic crisis occurs, discontinue any possible triggering agent and correct any precipitating condition. Urine output should be maintained by IV fluids and if Hb is low, blood transfusion may be needed.

In addition to these, it has been recommended that nitroprusside, and prilocaine in large amounts, be avoided. EMLA cream should be used with caution in patients with G6PD deficiency.

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INHERITED COAGULOPATHIES

ALASTAIR DUNCAN

Congenital disorders of coagulation are caused by genetic mutations that result in deficiencies in specific clotting factors (VII, VIII, IX, XI) or their carrier molecules (von Willebrand's factor). The resultant effect of these deficiencies is a propensity to bleed, which has implications for the patient, their preoperative preparation, anaesthesia and surgery.

HAEMOPHILIA

There are a number of different subtypes of haemophilia (Table 7.6). Haemophilias A and B are primarily found in males due to their pattern of inheritance. Haemophilia A is the most prevalent subtype (80%–90%) and the most clinically significant.

Haemophilia C is not linked to the X chromosome and can therefore affect both sexes. It is generally mild in its presentation and despite its increased incidence in the Ashkenazi Jewish population it is extremely rare.

The severity of haemophilia corresponds to the percentage of normal factor activity in the blood (Table 7.7).

CLINICAL MANIFESTATIONS

The clinical implications depend on the severity of the disease. Those with severe haemophilia commonly present as infants with spontaneous bleeding or excessive bleeding following minimally invasive medical intervention (e.g. heel prick). Spontaneous haemarthrosis is the classically described pathognomonic sign of severe haemophilia, with spontaneous intracerebral haemorrhage (ICH) a rare, but potentially life-threatening complication.

Patients with a mild form of haemophilia tend to present later in life and are generally

Table 7.6 Inherited bleeding disorders

Disorder	Inheritance	Gene mutation (gene; chromosome)	Deficiency	Incidence
Haemophilia A	XLR	F8; Ch X	Factor VIII	1:5000 male births
Haemophilia B	XLR	F9; Ch X	Factor IX	1:30,000 male births
Haemophilia C	AD/AR	F11; Ch 4	Factor XI	1:100,000
von Willebrand's disease (vWD)	AD/AR	vWF; Ch 12	vWF	1:100 1:10,000 (clinically significant)
Factor VII deficiency	AR	F7; Ch 13	Factor VII	1:500,000

Abbreviations: AD, autosomal dominant; AR, autosomal recessive; vWF, von Willebrand's factor; XLR, X-linked recessive.

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Table 7.7 Severity of haemophilia

Severity	Factor activity (%)	Factor levels (IU mL⁻¹)	Presentation	Bleeding tendency
Mild	5–40	0.05–0.40	Teenager/adulthood	Excessive bleeding after major surgery
Moderate	1–5	0.01–0.05	<2 years	Excessive bleeding after minor surgery
Severe	<1	<0.01	1–2 months	Spontaneous bleeding

asymptomatic. The disease reveals itself only following surgery or trauma. Any family history of bleeding disorders should be investigated thoroughly preoperatively.

Screening tests may demonstrate a prolonged activated partial thromboplastin time (APTT) in those with moderate or severe forms. The addition of normal plasma corrects this. Quantitative factor assays identify the subtype and severity of the disease.

The development of autoantibodies that inhibit the activity of the relevant exogenous factor complicates the situation in around 25% of those with severe haemophilia. This reduces the effectiveness of factor transfusion and can precipitate hypersensitivity reactions. Specific inhibitor assays can now be performed to guide management.

MANAGEMENT

A multidisciplinary management plan should be developed preoperatively with input from a haematologist, a surgeon and an anaesthetist. Factor levels should be increased to 0.8–1.0 IU mL⁻¹ using specific factor concentrates prior to surgery and maintained >0.5 IU mL⁻¹ for 5–14 days postoperatively depending on type and site of surgery.

Other treatment options to consider:

- Desmopressin (DDAVP) can be used in mild forms of haemophilia A. Given IV (0.3 µg/kg) or intranasally (150 µg in children or 300 µg in adults).
- Tranexamic acid can supplement treatment for mucocutaneous bleeding in haemophilia A and B (25 mg/kg/day).

- Recombinant factor VII (rFVIIa) and activated prothrombin complex concentrate (factor VIII inhibitor bypassing activity) can be used in those patients with autoantibodies to factor concentrates.

VON WILLEBRAND'S DISEASE

vWD results from a deficiency in von Willebrand's factor (vWF), a plasma glycoprotein that acts as a carrier for factor VIII and assists in platelet adhesion and aggregation.

vWD is classified depending on the degree of vWF deficiency:

Type 1	Partial quantitative defect
Type 2	Qualitative defect (subtypes A, B, M, N exist)
Type 3	Complete deficiency of vWF

CLINICAL MANIFESTATIONS

As with haemophilia, the clinical implications depend on the type of vWD and degree of vWF deficiency. Type 1 is generally associated with a mild to moderate bleeding tendency, whilst type 3 is the most severe form of the disease.

Patients may describe epistaxis, menorrhagia, oral bleeding or excessive bleeding following minor wounds. Alternatively, the disease may become apparent following surgery.

Initial screening tests measure factor VIII, vWF antigen, and vWF activity. Diagnosing specific subtypes of vWD requires measurement of ristocetin cofactor and collagen binding activity, and ristocetin-induced platelet agglutination.

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MANAGEMENT

Treatment consists of supplementary nonconcentrate therapies and specific factor concentrates.

- Desmopressin (DDAVP) given IV (0.3 μg/kg) or intranasally (150 μg in children or 300 μg in adults).
- Tranexamic acid can supplement treatment for mild bleeding and surgery (25 mg/kg/day).
- vWF-containing concentrate can be used for surgical prophylaxis.
- vWF-factor VIII concentrate can be used for emergency surgery or acute haemorrhage.
- Platelet transfusions may be required if pharmacological treatment fails.

Recommendations for the maintenance of factor VIII levels are similar to those described in haemophilia.

SPECIFIC ANAESTHETIC CONSIDERATIONS

- Associated history of blood-borne viruses (e.g. hepatitis B, C and HIV).
- Liaison with haematologist and laboratory services to coordinate treatment provision.
- Preoperative coagulation studies and factor assays should be performed.
- Risks and benefits of regional anaesthetic techniques should be explored and discussed with the patient.

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MASSIVE TRANSFUSION AND BLOOD LOSS

ALASTAIR DUNCAN

The management of massive blood loss can be required in a variety of different clinical situations.

- Trauma
- Obstetrics
- Surgery (e.g. cardiac, vascular, hepatobiliary)

A number of descriptions have been used to define massive haemorrhage:

- Loss of ≥1 blood volume in 24 hours
- Loss of ≥50% blood volume in <3 hours
- Haemorrhage ≥150 mL/min

Various definitions of massive transfusion also exist:

- Adults
  - Transfusion of ≥10 units of red blood cells in 24 hours (1 blood volume)
  - Transfusion of >4 units of red blood cells in 1 hour, with the expectation that further blood products will be required
  - Replacement of >50% blood volume within 3 hours
- Children
  - Transfusion of ≥1 blood volume in 24 hours
  - Replacement of >50% blood volume within 3 hours
  - Replacement of ongoing haemorrhage (>10% of the blood volume/min)

PHYSIOLOGICAL RESPONSE TO MASSIVE HAEMORRHAGE

CARDIOVASCULAR COMPENSATION

The initial response to a loss of circulating volume is triggered by arterial baroreceptors. An increase in sympathetic tone is accompanied by a decrease in vagal tone. This results in an increase in heart rate, systemic

vascular resistance (SVR) and a redistribution of blood from the splanchnic circulation to the vital organs.

This mechanism of compensation allows for the loss of up to 20% of blood volume without significant implications to an otherwise healthy patient. When 20%–30% of blood volume is lost, a vagally mediated reflex (Barcroft–Edholm) results in bradycardia, reduced SVR, and hypotension. If haemorrhage continues and >40% blood volume is lost, a preterminal sympathetic surge occurs to override the vagal reflex. This results in tachycardia and an increase in SVR. Despite this, these measures cannot overcome the loss of circulating volume and hypotension persists.

FLUID SHIFTS

Movement of fluid from the interstitial compartment to the intravascular compartment occurs due to changes in Starling's forces. The capillary hydrostatic pressure decreases, which allows the capillary oncotic pressure to facilitate movement of fluid from the interstitial compartment into the capillary. The compensatory mechanism is termed transcapillary refill. This results in a deficiency within the interstitial compartment. The activation of the renin-angiotensin-aldosterone system aims to address this by promoting the retention of sodium and water.

CLINICAL FEATURES

The clinical features associated with haemorrhagic shock have traditionally been described in relation to the volume of blood loss. Classes of shock are typically described as I–IV and correspond to a percentage blood loss of <15%, 15%–30%, 30%–40%, and >40% respectively.

- Class I (<15% or 750 mL)
  - Physiological parameters relatively unchanged
- Class II (15%–30% or 750–1500 mL)
  - Normal blood pressure; reduced pulse pressure
  - Tachycardia (100–120 beats per minute)
  - Delayed capillary refill
  - Pallor and anxiety

- Class III (30%–40% or 1500–2000 mL)
  - Worsening of signs seen in Class II
  - Tachypnoea
  - Blood pressure falling; heart rate rising
  - Urine output reduced
  - Pale, cold, drowsy and confused
- Class IV (>40% or >2000 mL)
  - Further worsening of all clinical signs
  - Blood pressure very low or unrecordable
  - Rapid thready pulse with negligible capillary refill
  - Pale, cold extremities, anuric
  - Very drowsy or unconscious

MANAGEMENT

Early recognition and intervention in massive haemorrhage is essential, as prevention may not be possible. The treatment goals include cessation of bleeding, restoration of circulating volume and managing the effects of massive transfusion.

IMMEDIATE MANAGEMENT

- Surgical, medical or radiological intervention to stop the bleeding.
- Crystalloid fluid resuscitation until blood products are available.
- Group O Rhesus D negative blood after sample for crossmatch taken.
- Initiate the major haemorrhage protocol and administer blood products.
- Serial measurement of haematological and coagulation studies .
- Ensure normothermia.
- Consider administration of tranexamic acid.

MAJOR HAEMORRHAGE PROTOCOL (MHP)

Administration of blood products should be guided by haematological results. Unfortunately, this is not always possible as the situation may develop rapidly. In this case, a pre-agreed combination of blood products should be administered.

The Association of Anaesthetists of Great Britain and Ireland (AAGBI) recommends that every hospital have an MHP. The principle is to replace blood

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Table 7.8 Comparison of blood products

	Red cells	Platelets	FFP	Cryoprecipitate
Volume	220–340 mL	300 mL	274 mL	189 mL
Content	Red blood cells (Hct 0.6)	Platelets (308 × 10⁹/L)	Clotting factors	Fibrinogen FVIII, vWF
Additive	SAG-M	CPD	CPD	None
Storage temp.	2–6°	20–24°	<–25°	<–25°
Administration	<4 hours	<2 hours	<2 hours	<2 hours

Abbreviations: CPD, citrate phosphate dextrose; FFP, fresh frozen plasma; FVIII, factor VIII; Hct, haematocrit; SAG-M, saline, adenine, glucose, mannitol; vWF, von Willebrand's factor.

to restore the circulating volume, whilst achieving haemostasis. This requires a combination of blood products to be transfused (Table 7.8).

Recommended transfusion triggers:

- Prothrombin time ratio (PTr)   >1.5
- Platelet count   <75 × 10⁹/L
- Fibrinogen   <1.5 g/L

The use of adjuncts such as recombinant factor VIIa, prothrombin complex concentrate or fibrinogen concentrate should be discussed with a haematologist.

TARGETED BLOOD PRODUCT ADMINISTRATION

Point-of-care coagulation testing provides the anaesthetist with the tools to administer the appropriate blood product depending on the patient-specific deficiency.

Thromboelastography (TEG®) and rotational thromboelastometry (ROTEM®) provide a continuous measurement of the viscoelastic properties of blood from the start of clot formation to fibrinolysis.

Despite the different terminology and technique used by each machine, the principles of what is measured on each machine and their implications are similar.

- Reaction time (TEG) and clotting time (ROTEM)
  - Initiation of clotting process
  - Dependent on concentration of clotting factors
  - If prolonged, treatment includes FFP or reversal of heparin
- Kinetics time (TEG) and clot formation time (ROTEM)
  - Amplification of clotting process
  - Measures of clot kinetics
  - If prolonged, treatment options include cryoprecipitate
- α angle (TEG and ROTEM)
  - Propagation of clot (thrombin burst)
  - Represents speed of fibrin build up and cross-linkage
  - If decreased, treatment includes cryoprecipitate
- Maximum amplitude (TEG) and maximum clot firmness (ROTEM)
  - Propagation of clot (platelet-fibrin interaction)
  - Measures of platelet number and function
  - If decreased, treatment includes platelets
- Lysis time (TEG) and maximal lysis (ROTEM)
  - Fibrinolysis
  - Measurement of clot stability
  - Measured at both 30 and 60 minutes
  - Treatment of early fibrinolysis includes antifibrinolytics

COMPLICATIONS

Complications due to massive transfusion (Table 7.9) must be recognised and treated promptly.

Table 7.9 Complications of massive transfusion

Transfusion reactions	Immunological reactions	Metabolic complications	Others
Allergy	TRALI	Hypocalcaemia	Hypothermia
Haemolytic	TRIM	Hyper- or hypokalaemia	Coagulopathy
Febrile non-haemolytic	Ta-GVHD	Hypomagnesaemia	Infection
	PTP	Acidosis	TACO
		Alkalosis	

Abbreviations: PTP, post-transfusion purpura; TACO, transfusion-associated circulatory overload; Ta-GVHD, transfusion-associated graft versus host disease; TRALI, transfusion related acute lung injury; TRIM, transfusion related immunomodulation.

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CROSS-REFERENCE

Blood transfusion, Chapter 30

MASTOCYTOSIS

SANTOSH PATEL

Mastocytosis is a heterogeneous group of disorders. It is rare with an incidence of 1–4:10,000 and classified into seven categories. These differ by age of onset (paediatric vs. adult), phenotype (cutaneous vs. systemic) and clinical characteristics (indolent vs. aggressive) of the disease. Two-thirds of patients develop the disease in childhood, with an equal distribution between the sexes. The condition has been reported in all races. Familial cases have been documented, though most patients have no familial association. Symptoms and signs result from immune and nonimmune stimulation of mast cells resulting in the local and systemic release of a variety of biologically active mediators.

PATHOPHYSIOLOGY

Currently, the only established pathological mechanism appears to be the presence of a somatic mutation in codon 816 of the c-kit proto-oncogene, leading to constitutive activation of the KIT receptor (a type III tyrosine kinase receptor), which drives mast cell proliferation and survival.

Stimulation of the mast cells results in the release of preformed mediators (including histamine, heparin, chemotactic factors and cytokines) and newly formed mediators (including prostaglandin D2, leukotrienes and platelet activating factor).

Clinically, the most important of these are histamine and prostaglandin D2 (PGD2), their physiological effects being widespread:

- Cardiovascular
  - Venous dilatation resulting in increased vascular capacity
  - Arteriolar dilatation resulting in decreased arterial pressure
  - Increased capillary permeability with rapid loss of fluid into tissue spaces
- Respiratory
  - Bronchospasm and increased production of airway secretions
- Cutaneous
  - Increased skin blood flow with erythema and flushing
  - Pruritus

- Gastrointestinal
  - Increased gastric secretions
  - Increased gut motility
- Other
  - Increased uterine contraction

CLINICAL FEATURES

The diverse nature of the disease, with both acute attacks and chronic organ involvement, results in symptoms that are variable in terms of severity and duration. Symptoms range from pruritus and flushing to dyspnoea, abdominal pain, and even syncope and are attributable to the wide-ranging physiological effects of the secreted mast cell mediators (see Table 7.10). Episodes of syncope and hypotension are rare, as are deaths associated with mast cell mediator release.

PRECIPITATING FACTORS

Mast cell stimulation results from both immune and nonimmune mechanisms. Perioperatively, the latter are of greater significance and may be classified into nonpharmacological or pharmacological triggers.

NONPHARMACOLOGICAL TRIGGERS

Mechanical irritation of the lesions, temperature changes, exercise, vomiting, pain and psychological stress.

PHARMACOLOGICAL TRIGGERS

A multitude of drugs have been implicated including alcohol, opiates (morphine, tramadol, pethidine, codeine), thiopental, atracurium, rocuronium, mivacurium, suxamethonium, non-steroidal anti-inflammatory drugs, nefopam, vancomycin, aspirin, radiocontrast media, beta-receptor antagonists, alpha-receptor agonists and drug preservatives, including sodium metabisulphate and parabens.

PREOPERATIVE ASSESSMENT

A detailed history may suggest organ involvement and may highlight any known trigger agents. All patients with suspected systemic involvement should have a full blood count and a peripheral smear to exclude an associated haematological disorder. Abnormal findings should prompt a bone marrow biopsy. Although liver function tests are usually normal, an abdominal ultrasound scan should be performed to rule out hepatic or splenic involvement.

Serum alpha-tryptase is elevated in patients with systemic mastocytosis regardless of whether they are experiencing acute symptoms, and it can be used to assess the total-body mast cell burden. A plasma level >20 µg/L should be considered as a cut-off. Urinary methylimidazole acetic acid (a histamine metabolite) levels have been shown to correlate closely to the extent of mast cell disease, being highest in patients with widespread systemic involvement. Beyond these tests, any additional work-up should be tailored to individual specific symptoms and may include gastrointestinal tract endoscopy, bone scans and lymph node biopsy.

PREOPERATIVE PREPARATION

Epinephrine and other resuscitation drugs and equipment must be immediately available. Premedication with H_1 and H_2 histamine receptor antagonists and NSAIDs is recommended, although the latter can act as a trigger agent. Sodium cromoglycate (cromolyn) may help stabilise mast cells, although prophylactic steroids have not been shown to be of any benefit. There are several reports of preoperative intradermal skin tests being used to identify drugs that may be

Table 7.10 Symptoms and signs of mastocytosis

Cardiopulmonary – Chest pain, dizziness, dyspnoea, palpitations, syncope

Gastrointestinal – Abdominal cramps, diarrhoea, nausea and vomiting, hepatosplenomegaly

Skin – Bullae, urticaria and oedema, flushing and pruritus

Neurological – Cognitive disorganisation, headaches

Skeletal – Bone pain, osteoporosis, osteopenia, pathological fractures

Haematological – Anaemia and thrombocytopaenia, bleeding diathesis

Other – Fever, weight loss

safely administered during anaesthesia. However, the specificity of these tests is uncertain.

PERIOPERATIVE MANAGEMENT

PREMEDICATION

Anxiolysis and sedation may be achieved using benzodiazepines. Anticholinergic drugs such as hyoscine should be avoided. In some cases, prophylactic administration of H1 receptor antagonists, glucocorticoids, chromolyn and leukotriene antagonists has been reported. However, there are no evidence-based guidelines available.

INDUCTION AND MAINTENANCE

Mast cell degranulation in patients with mastocytosis is unpredictable, and does not occur consistently in any given patient. However, haemodynamic instability should be anticipated, and resuscitation drugs and equipment must be immediately available at all times. Large-bore intravenous cannulae should be inserted and monitoring instituted before induction of anaesthesia. Drugs known to cause histamine release should be avoided. Drugs that have been safely administered include hydroxyzine, propofol, ketamine, etomidate, fentanyl, sufentanil, remifentanil, vecuronium, volatile anaesthetic agents (ether-linked anaesthetic agents inhibit mast cell degranulation), nitrous oxide, benzodiazepines, paracetamol and preservative-free amide local anaesthetics. If an anticholinergic drug is required, glycopyrrolate is preferred. The number of pharmacological agents used should be kept to a minimum. Regional anaesthetic techniques may be used, although these have still been associated with acute reactions.

ENVIRONMENT

A warm, calm environment should be maintained throughout the perioperative period. Hypothermia should be prevented by both active and passive means. Local tissue trauma should be minimised by careful patient handling, positioning and padding of all pressure points. Tourniquets should be used with caution if they are deemed necessary.

POSTOPERATIVE MANAGEMENT

A calm environment, adequate analgesia and normothermia should be maintained after surgery. Skin irritation and trauma should continue to be minimised, and resuscitation drugs and equipment should remain immediately available.

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CROSS-REFERENCE

Urticaria and angio-oedema, Chapter 9

MULTIPLE MYELOMA

SANTOSH PATEL

Multiple myeloma is a haematological malignancy causing diffuse proliferation of B lymphocytes and plasma cells largely confined to bone marrow. It is characterised by the production of a monoclonal immunoglobulin and the occurrence of osteolytic bone lesions. The diagnosis is made by the coexistence of at least two of the following:

- Excess plasma cells in the marrow
- Monoclonal immunoglobulin concentration of >1 g dL^{-1} (IgG or IgA) or excess light chains in the urine
- X-ray evidence of lytic lesions

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It is a disease of the elderly, with a mean age at diagnosis of 60 years.

PATHOPHYSIOLOGY

BONE LESIONS

Lytic lesions, through which pathological fractures occur, result from the secretion of an osteoclast-stimulating cytokine by the abnormal plasma cells. Bones fracture either spontaneously or after trivial injuries; vertebral collapse is particularly common. Patients often suffer from severe bone pain.

HYPERCALCAEMIA

In general, this occurs only in those patients with extensive osteolysis. It is exacerbated by dehydration secondary to vomiting and the inability to retain salt and water due to renal involvement. It may also be precipitated by bed rest and infection. Hypercalcaemia can cause vomiting, constipation, anorexia, depression, confusion, drowsiness and even coma.

RENAL IMPAIRMENT

It may be present in 50% of cases. Renal impairment is due to a combination of dehydration, hypercalcaemia, pyelonephritis, deposition of myeloma protein in the kidney causing damage, and in some cases renal amyloidosis. In acute renal failure secondary to myeloma, the most important measure is correction of dehydration and treatment of any precipitating renal infection. This has to be considered as a major issue in these patients.

HAEMATOLOGICAL ABNORMALITIES

Normochromic, normocytic anaemia is common and may be severe due to marrow failure or renal failure. Haemostatic abnormalities may be due to interference with clotting and platelet function by monoclonal immunoglobulin, hyperviscosity, renal failure or, rarely, thrombocytopaenia as a result of marrow infiltration or chemotherapy.

HYPERVISCOSITY

This is a rare complication associated with high plasma levels of monoclonal immunoglobulin. It is more likely to occur in IgA myelomatosis where there is polymerisation of monoclonal immunoglobulin molecules. It can cause a variety of neurological, ocular, haematological and cardiac problems, including cardiac failure. It may also result in spurious hyponatraemia and predispose to venous thrombosis.

IMMUNE SYSTEM

Synthesis of all immunoglobulins apart from the monoclonal immunoglobulin is depressed, leading to increased susceptibility to infection, especially by staphylococci and Gram-negative organisms.

NERVOUS SYSTEM

The most important neurological manifestations are peripheral neuropathy, paraplegia secondary to an epidural plasmacytoma and spinal root compression due to paravertebral masses or collapsed vertebrae.

ANAESTHETIC MANAGEMENT

There has been little research into the anaesthetic management of patients with multiple myeloma and therefore all recommendations have to be based purely on an understanding of the abnormalities associated with the disease.

PREOPERATIVE

Management should be directed towards the detection and correction of abnormalities associated with myeloma, with particular attention to fluid balance, hypercalcaemia, haemostatic abnormalities and renal impairment. Patients with symptomatic hyperviscosity should be treated with plasmapheresis preoperatively. Patients may be taking steroids or a variety of cytotoxic agents. Anaemia and coagulation abnormalities may be present. FBC, coagulation and renal function should be evaluated.

INTRAOPERATIVE

Regional anaesthesia

This may be absolutely contraindicated for medical reasons (haemostatic abnormalities, vertebral collapse) or relatively contraindicated for medicolegal reasons (active neurological disease).

General anaesthesia

Attention to the positioning of the patient is essential in view of the increased susceptibility to fractures. Strict asepsis is important in view of the impairment of immune function that may be further impaired by general anaesthesia. In theory, adjustment of doses of intravenous agents may be necessary in view of changes in plasma proteins.

POSTOPERATIVE

Duration of immobility should be minimised to decrease the risk of precipitating hypercalcaemia and of developing venous thromboses.

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PRIMARY IMMUNE THROMBOCYTOPAENIA

ALASTAIR DUNCAN

An isolated thrombocytopaenia in the absence of a demonstrable cause is termed primary immune thrombocytopaenia (previously labelled idiopathic thrombocytopaenic purpura [ITP]).

The incidence of primary immune thrombocytopaenia is thought to be around 5 per 100,000 population. The disease is more prevalent in women between the third and fifth decade, but demonstrates similar incidences outside this age range.

Primary immune thrombocytopaenia can be classified as:

- Newly diagnosed
- Persistent (3–12 months' duration)
- Chronic (>12 months' duration)

In children, primary immune thrombocytopaenia generally follows a viral illness, is self-limiting and resolves within 6 months. In adults, there is rarely a precipitating illness and the disease often progresses to the chronic stage.

PATHOPHYSIOLOGY

Primary immune thrombocytopaenia is an autoimmune mediated disorder. Autoantibodies (IgG) bind to specific platelet membrane glycoproteins (e.g. IIb/IIIa) producing an antigen-antibody complex. This induces phagocytosis by mononuclear macrophages of the reticulo-endothelial system in the spleen. These autoantibodies can bind to and damage megakaryocytes. The result is platelet destruction and suppression of platelet production.

DIAGNOSIS

A platelet count of $<100 \times 10^9$/L is a prerequisite for a diagnosis of primary immune thrombocytopaenia. There is currently no investigation that definitively confirms primary immune thrombocytopaenia. It is a diagnosis of exclusion based on history, examination and associated investigations.

Investigations to exclude identifiable causes of thrombocytopaenia include:

- Blood film
- Bone marrow examination
- HIV and hepatitis C serology
- Quantitative immunoglobulin levels

CLINICAL FEATURES

Patients with primary immune thrombocytopaenia may be asymptomatic or might describe significant clinical manifestations of their condition.

- Petechiae or purpura
- Unusual or easy bruising
- Mucosal bleeding
- Epistaxis
- Menorrhagia

MANAGEMENT

The goal is to achieve a sufficient platelet count to prevent or stop bleeding whilst ensuring an acceptable quality of life with minimal treatment-related side effects.

The decision to commence treatment depends on the platelet count, the patient's symptoms and their risk of bleeding.

FIRST-LINE TREATMENT

- Corticosteroids
  - The side effects associated with long-term steroid use should be taken into account.
- Intravenous immunoglobulin
  - The rapid increase in platelet count (within 24 hours) must be balanced against the risks of toxic side effects.
- Intravenous anti-D
  - Can be used in Rhesus D positive patients. Thought to act by saturating macrophage receptors with anti-D coated red blood cells. Has been removed from some European markets due to safety concerns.

SECOND-LINE TREATMENT

- Pharmacological immunosuppression
  - Azathioprine
  - Cyclosporin A
  - Cyclophosphamide
  - Danazol
  - Dapsone
  - Mycophenolate mofetil
  - Rituximab

- Stimulation of platelet production
  - Romiplastim and eltrombopag are thrombopoietin receptor agonists
- Surgical immunosuppression
  - Splenectomy

Nearly 80% of patients achieve the treatment goal with first- or second-line therapies. Those who do not may be offered combination chemotherapy or stem cell transplantation.

SPECIAL CONSIDERATIONS

TREATMENT FOR PATIENTS UNDERGOING SURGERY

When to treat:

Platelet count (× 10^9/L)	Treatment option
50–100	No treatment unless undergoing neurosurgery or posterior eye surgery
	Corticosteroids are required for these patients to ensure platelet count >100 × 10^9/L
30–50	No treatment if asymptomatic and low risk of bleeding
	Corticosteroids for all patients scheduled for surgery (excepting neuro- and posterior eye surgery)
<30	Corticosteroids
Acute haemorrhage	Platelet transfusion
	Intravenous immunoglobulin
	Corticosteroids
	Tranexamic acid

PATIENTS UNDERGOING SPLENECTOMY FOR PRIMARY IMMUNE THROMBOCYTOPAENIA

- Prophylactic pneumococcal, meningococcal C conjugate, and haemophilus influenza B vaccines ideally administered 4 weeks prior to surgery.
- Prophylactic antibiotics for surgery should be discussed with the surgeon and microbiologist.

- Postoperative thromboprophylaxis is particularly important, as primary immune thrombocytopaenia and splenectomy are independent risk factors for thromboembolism.

THROMBOCYTOPAENIA IN PREGNANCY

Thrombocytopaenia affects up to 10% of pregnancies. Gestational thrombocytopaenia (GT) accounts for 75% of these cases. This is thought to result from haemodilution and increased platelet activation and clearance. This can lead to a 10% reduction in platelet count during the latter stages of the third trimester. The platelet count rarely falls below 70×10^9/L in GT and usually resolves within 8 weeks.

The first presentation of primary immune thrombocytopaenia may occur during pregnancy. This can be extremely difficult to differentiate from GT. Other pregnancy-related causes of thrombocytopaenia should be excluded (e.g. pre-eclampsia or HELLP syndrome).

If a diagnosis of primary immune thrombocytopaenia is confirmed prior to or during pregnancy, some additional precautions should be taken.

- Multidisciplinary treatment plan agreed by obstetrician, haematologist, anaesthetist and neonatologist.
- Standard first-line treatments are safe in pregnancy.
- Second-line therapies are guided after careful consideration by the haematologist and patient.
- Minimum platelet count for delivery is 50×10^9/L.
- Minimum platelet count for regional anaesthetic techniques is 75×10^9/L.
- Placental transfer of autoantibodies can occur, which can result in neonatal thrombocytopaenia requiring treatment.
- Thromboembolic risk is high and thromboprophylaxis should be considered.

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CROSS-REFERENCE

Serious complications of pregnancy, Chapter 12

SICKLE CELL SYNDROME

SANTOSH PATEL

Sickle cell syndromes are inherited haemoglobinopathies in which the dominant haemoglobin (Hb) is the unstable haemoglobin S. They include sickle cell anaemia (HbSS) and the double heterozygote conditions sickle C (HbSC) and sickle thalassaemia (HbSThal).

PATHOPHYSIOLOGY

Sickle gene causes abnormal production of β globin chain because of substitution of glutamic acid with valine. In homozygous sickle gene population (sickle cell disease), haemoglobin S (Hb SS) replaces normal Hb A. In heterozygous (sickle cell trait) sickle gene individuals (HbAS), both Hb S and Hb A exist.

When haemoglobin S is deoxygenated, the molecules polymerise into long chains called 'tactoids' and become insoluble. This results in deformation of the red cell membrane into the characteristic sickle shape. Although the process is often reversible with oxygenation, haemolysis will occur if the cell membrane is damaged. When a sickle cell ruptures, it releases haemoglobin and iron which damages the vascular endothelium and causes an inflammatory response. Haemolysis of a sickle cell also reduces nitric oxide availability.

During prolonged periods of deoxygenation, irreversible sickling may occur. In this situation, the cells aggregate and occlude small blood vessels and this leads to tissue infarction and further hypoxia. Hypoxia is aggravated by lung infarction, which is a common cause of death. The major features of sickle cell disease are therefore chronic anaemia and the occurrence of sickle cell 'crises' in which multiple episodes of tissue infarction occur.

Sickling occurs in individuals who are homozygous for the sickle gene (HbSS) and also in those in whom the sickle gene is inherited along with another variant such as HbC or β thalassaemia. Tactoid formation is enhanced in the presence of HbC compared with the normal HbA. Patients with HbSC may have Hb concentration towards the lower end of the normal range but are liable to sickle and have a high incidence of venous thrombosis. Inheritance of the β thalassaemia gene results in greatly reduced β chain synthesis.

Individuals with sickle cell trait (HbAS) are usually asymptomatic, although sickling may occur under conditions of severe hypoxaemia. Patients with sickle cell trait are not at increased risk during a properly conducted anaesthetic, although the use of a tourniquet may be hazardous.

PREOPERATIVE ASSESSMENT

Patients from susceptible populations should be screened for haemoglobin S with a quick solubility test, e.g. Sickledex. This will not distinguish between HbAS and the more dangerous phenotypes, but other abnormalities in the full blood count, and examination of the blood film, should alert the laboratory to the presence of an important haemoglobinopathy. Haemoglobin electrophoresis is necessary to differentiate between types of HbS.

Preoperative assessment should focus on identifying and evaluating for wider systemic manifestations and complications (Table 7.11). Vaso-occlusive, sequestration, haemolytic and aplastic crises have been described. Serious complications including acute chest syndrome and cerebrovascular accident, and risk factors for acute sickle crisis should be identified and optimised before elective surgery.

History and examination should identify the frequency, pattern, and severity of recent sickle crises and the presence of organ damage.

Patients with HbSS are usually well adapted to their low haemoglobin concentration, and increases in the haematocrit will increase the risk of a vaso-occlusive crisis. Patients who have received multiple transfusions in the past may have many red cell antibodies. Consequently, compatible blood may be difficult to find and transfusion reactions are relatively common. The optimum haemoglobin concentration and the acceptable proportion of HbS are both debatable and are determined in part by the proposed surgical procedure and severity of anaemia. In general,

Table 7.11 Clinical features and complications in patients with sickle cell disease

Haematological	Anaemia in SD. In SCT Hb level usually normal.
	Aplastic anaemia
	Splenic sequestration, infraction, functional asplenia
	Oxyhaemoglobin dissociation curve shifted to left
	Low arterial oxygen saturation
	Increased blood viscosity
Pulmonary	Lung infarctions
	Recurrent chest infections
	V-Q mismatch
	Acute chest syndrome
	Pulmonary hypertension
Skeletal	Bone infarction
	Osteomyelitis, arthritis, synovitis
	Osteonecrosis
Neurological	Stroke
	Peripheral neuropathy
Pain	Acute pain crises, Chronic pain syndromes
Cardiovascular system	Cardiomegaly
	Congestive heart failure
	Myocardial infarction
Gastrointestinal	Cholelithiasis
	Hepatic infarcts
Abdomen	Abdominal crises may mimic a surgical emergency
Renal	Impaired concentration ability causes polyuria
	Chronic renal failure common
Occular	Proliferative retinopathy
	Retinal infract, detachment
Skin	Chronic ulcers

blood transfusion is usually unnecessary for patients undergoing minor surgery. For major surgery, simple red cell transfusion to increase the haemoglobin concentration to 10 g dL^{-1} seems to be as effective in preventing complications as more complex transfusion regimens to reduce the HbS concentration. An HbA level >40% is desirable. Multidisciplinary discussion with both haematologist and surgeon should be carried out to formulate an appropriate management plan. Erythropoetin and hydroxyurea may be useful to increase Hb level. The later also has antisickling effects.

PERIOPERATIVE MANAGEMENT

Common surgical procedures in patients with SCD include cholecystectomy, splenectomy, caesarean section, dilatation and curettage, hysterectomy, adenotonsillectomy and orthopaedic surgery. There is no evidence in support of general or regional anaesthetic techniques. High-risk surgery such as intracranial and cardiac surgery with cardiopulmonary bypass may pose specific challenges. Pregnant women are at risk for complications particularly for anaemia and vaso-occlusive crises during ante-, intra- and postpartum periods.

The principal objectives are to avoid any factors that may promote sickling. In particular, care should be taken to maintain good oxygenation, hydration, normothermia, normal acid-base balance and aseptic precautions. Perioperative stress and infections may trigger sickle cell crises. Prevent low flow states and hypotension. Prophylaxis against thromboembolism is indicated depending on the type of surgery. Limb tourniquets are best avoided although there are reports of their uncomplicated use. If a tourniquet is essential, it should be applied as distally as possible, the limb should be thoroughly exsanguinated and the ischaemic time should be minimised.

The high standard of care delivered in the operating theatre must continue into the postoperative period. This may require advance planning to ensure that a bed on a high dependency unit is available. During the postoperative period, factors precipitating a sickle cell crisis should be avoided. Supplemental oxygen, optimal fluid balance, chest physiotherapy, pain control and high vigilance for complications of sickle cell disease are important.

Pain management presents a number of problems. Opioids may cause respiratory depression with consequent acidosis and hypoxia. Some patients may have developed a tolerance to opioids if they have required frequent treatment for sickle cell crises. In theory, regional pain techniques seem preferable although there is little evidence to support this. Careful patient monitoring is essential.

Painful sickle crises may present in the postoperative period; these need to be distinguished from surgical pain and treated appropriately. The acute chest syndrome is an acute pain crisis presenting as pleuritic chest pain, fever, hypoxaemia and lung infiltrates on the chest X-ray. It has a high mortality rate and requires urgent treatment with oxygen therapy and exchange transfusion to decrease the HbS concentration.

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THALASSAEMIA

SANTOSH PATEL

The thalassaemias are an inherited group of haematological disorders in which there is deficient

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synthesis of α or β globin chains. They are the most common monogenic blood diseases and are more frequently found in people originating in the Mediterranean, Central African, China and South East Asian regions.

In normal adults, the majority of haemoglobin is HbA, which has two α and two β chains, i.e. α2β2. There are four genes controlling α-chain production (two on each chromosome 16) and two controlling β-chain production (one on each chromosome 11).

α THALASSAEMIA

The classification of α thalassaemia into α0 and α+ has replaced the older nomenclature of Type 1 (severe) and Type 2 (mild).

α0 Thalassaemia results from elimination of α1 and α2 genes and/or regulatory sequences. α+ thalassaemia results from a single gene deletion or point mutation that prevents normal α chain production. Deletion of only one gene controlling α chain production produces haematological indices which overlap with those of the normal population. α Thalassaemia trait is caused by the interaction of the normal haplotype with α0 or α+ thalassaemia determinant or homozygosity for two α+ haplotypes. The resulting anaemia is mild and of little anaesthetic significance.

Haemoglobin H disease is caused by the interaction of α0 and α+ determinants (loss of three genes). The clinical picture is of chronic haemolytic anaemia with Hb levels of 8–10 g dL^{-1}. This stimulates erythropoietin production and results in excess production of large amounts of β-chains that form insoluble tetramers (HbH). The anaemia is characterised by jaundice, hepatosplenomegaly, leg ulcers, gallstones and folic acid deficiency. Haemolysis may be increased by oxidant drugs. Inability to produce any α chains results in hydrops fetalis in which death occurs *in utero* or soon after birth.

β THALASSAEMIA

Beta thalassaemia is a defect of the beta globin chains of the haemoglobin A molecule. The clinical presentation typically manifests at approximately 4 to 6 months of age, as during this time period haemoglobin F falls significantly to be replaced by haemoglobin A. This genetic disorder is transmitted by autosomal recessive inheritance. A defect in one beta globin allele will result in beta thalassaemia minor. This is effectively a carrier state, and individuals are usually asymptomatic or present with anaemia. Defects in both alleles result in beta thalassaemia major, which results in a severe clinical picture requiring lifelong blood transfusions. A more moderate phenotype presenting with less blood transfusion dependence is called beta thalassaemia intermedia. The cause behind this differing severity of disease from the same genotype is unknown. However, the type of mutation and gene interactions are thought to play key roles.

Usually inherited as a recessive disorder (some cases in which unstable β chains are produced are dominantly inherited), they present as a quantitative reduction in β chain production. Under-production of β chains results in an excess of α chains, which are unstable and precipitate in red cell precursors, which are destroyed in the bone marrow.

β Thalassaemia trait results from the inheritance of a single abnormal gene (β0 or β+) and is associated with mild iron-resistant hypochromic anaemia but with little or no other disability.

β Thalassaemia major results from the presence of two abnormal genes, and sufferers are unable to produce any β chains. Production of foetal haemoglobin (α2γ2) leads to a haemoglobin concentration of 30%–50% of the normal adult levels. This condition is associated with a severe anaemia that requires blood transfusion from the first year of life. Patients almost invariably develop antibodies making crossmatching of blood difficult. Patients must be treated with desferrioxamine (an iron chelating agent) to decrease iron overload and haemosiderosis. Patients may be treated with bone marrow transplantation early in life before iron overload occurs.

CLINICAL FEATURES AND MANAGEMENT OF THALASSAEMIA

Clinical features (Table 7.12) of thalassaemia are due to anaemia, chronic haemolysis, ineffective extramedullary erythropoiesis and iron overload. Regular blood transfusions may be required depending on the type of thalassaemia. Iron chelating drugs such as desferroximanie have side effects. Splenectomy has been shown to reduce requirement for blood transfusions.

Table 7.12 Clinical features of thalassaemia

- Intravascular haemolysis:
  - Anaemia
  - Jaundice, gall stones
  - Splenic sequestration causing anaemia and other haematological effects
  - Free iron causing vascular endothelial inflammatory response and its consequences, e.g. leading to systemic and pulmonary hypertension, cerebral ischemia, platelet activation, hypercoagulability
- Extramedullary erythropoiesis:
  - Frontoparietal bossing, prominent maxilla and zygomatic arch, compression of neural structures in bony canal including spinal cord, bony deformities e.g. thoracic cage, spinal column
- Systemic effects of anaemia:
  - High output cardiac failure, decreased oxygen delivery leading to poor growth
- Multiple transfusions:
  - Hypervolemia, coagulation defects, immunosuppression, iron overload
- Iron overload:
  - Cardiomyopathy, cardia hypertrophy, myocarditis, pericarditis
  - Liver failure and cirrhosis
  - Renal tubular dysfunction, loss of concentrating renal function and increased excretion of calcium, magnesium and phosphate
  - Endocrionpathy (diabetes, hypothyroidism, hypoparathyroidism, adrenal insufficiency)
  - Immunosuppression

PREOPERATIVE ASSESSMENT

Symptoms and signs of cardiac, respiratory, hepatic, renal or endocrine disorders should be sought. Ease of venous access should be assessed as repeated transfusions may have damaged peripheral veins.

Careful assessment of the patient's airway is essential. Skeletal abnormalities should be assessed as the patient may be kyphoscoliotic, which may have implications for lung function and positioning on the operating table.

Investigations should include full blood count, urea and electrolytes, blood glucose estimation, liver function tests and ECG (and echocardiogram if cardiac dysfunction is suspected). Allow extra time for blood cross-matching.

PERIOPERATIVE MANAGEMENT

There is no specific anaesthetic technique or agent recommended. Choice of anaesthetic technique, agent and monitoring should be tailored to each individual patient's condition and type of surgery. For regional anaesthesia, preexisting skeletal deformities, coagulation problem and any neurological deficits should be evaluated.

Transfusion to normal haemoglobin levels is not indicated unless it is necessary to improve cardiorespiratory function. If the patient has an associated cardiomyopathy, intensive preoperative chelation therapy may improve cardiac function. Individuals who have had a splenectomy (for hypersplenism) should have had pneumococcal vaccine and should receive antibiotic prophylaxis.

Aseptic precautions are necessary as these patients are at increased risk of infection.

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CROSS-REFERENCE

8

Bones and joints

BRIAN J POLLARD AND GARETH KITCHEN

ANKYLOSING SPONDYLITIS

Ankylosing spondylitis (AS) affects young people principally, presenting at around 26 years of age with a male:female ratio of 2:1. The overall prevalence is between 0.1% and 1.4% (in mid-Europe 0.3%–0.5%) and the incidence between 0.5 and 14 per 100,000 population per year. About 80% develop the first symptoms before 30 years and less than 5% present at older than 45 years. Approximately 90%–95% patients are positive for HLA B27 and the risk of this disease developing is about 5% in HLA B27-positive individuals and higher in HLA B27-positive relatives. However, most HLA B27-positive individuals remain healthy.

The diagnostic criteria are given in Box 8.1. Functional restrictions are greater in those with a history of physically demanding jobs, more comorbid conditions and in smokers, than in those with higher levels of education and a family history of this disease. Young age at onset is associated with worse functional outcome. In juvenile patients with spondyloarthritides, clinical symptoms can be different and include severe tarsitis. Male patients have more structural changes, including bamboo spine, than do female patients.

PATHOPHYSIOLOGY

MUSCULOSKELETAL SYSTEM

Characteristic symptoms are spinal stiffness and loss of spinal mobility due to inflammation, structural damage or both. Spinal inflammation can arise as spondylitis, spondylodiscitis or spondylarthritis. Structural changes are mainly caused by osteoproliferation rather than osteodestruction. Syndesmophytes and ankylosis are the most characteristic features and are visible on conventional radiographs. Low bone density, osteoporosis and increased rate of fractures occur. Decreased movement of the lumbar spine results with a proportion progressing to ankylosis and complete rigidity with a classical X-ray picture of 'bamboo spine'.

Complications include fractures with little or no history of trauma, collapse of vertebral end-plates (spondylodiscitis) and spinal nerve root compression. Cervical fractures (commonly C5-6) may occur with minimal trauma or hyperextension. Clinically significant atlanto-axial subluxation occurs in 21%. About 47% with vertebral compression fractures have a neurological complication ranging from paraesthesia to loss of muscle strength.

Diagnosis of AS requires one radiological criterion with at least one clinical criterion. Probable ankylosing spondylitis if >3 clinical criteria or radiological criteria are present.

Clinical criteria
- Low back pain >3 months' duration, improves with exercise and is not relieved by rest
- Limitation of motion of the lumbar spine in sagittal and coronal planes
- Limitation of chest expansion relative to normal values corrected for age and sex

Radiological criteria
- Bilateral sacroilitis – grade 2 (sclerosis with some erosions) or higher
- Unilateral sacroilitis – grade 3 (severe erosions, pseudodilatation of joint space and partial ankylosis) or grade 4 (complete ankylosis)

The most common joints affected are hips and shoulders. Peripheral joint involvement occurs in 50% and is more common in patients with concomitant psoriasis. Temporomandibular joint involvement causes limited mouth opening in 10% of patients rising to 30%–40% with long-standing disease. The disease rarely causes arthritis of the cricoarytenoid joint, but can lead to dyspnoea, hoarseness and vocal cord fixation. AS often affects costovertebral and costo-transverse joints causing local tenderness and pain on coughing or sneezing. Other features include plantar fascitis and Achilles tendonitis.

RESPIRATORY SYSTEM

Upper lobe pulmonary fibrosis is a recognized complication of long-standing AS. This may significantly impair respiratory reserve. Chest X-rays may show apical fibrosis. Pulmonary function tests reveal a restrictive lung defect.

CARDIOVASCULAR SYSTEM

Cardiovascular involvement occurs in up to 10% of patients with severe spondylitis. There is aortitis and aortic insufficiency, occasionally affecting the mitral valve. Involvement of the Purkinje fibres may cause conduction defects increasing the risk of myocardial infarction. Long-term disease is associated with a greatly increased cardiovascular mortality.

NEUROLOGICAL SYSTEM

Neurological effects include spinal cord compression, cauda equina syndrome, cervical spine fracture, focal epilepsy, vertebrobasilar insufficiency and peripheral nerve lesions. Spinal fractures can lead to acute epidural haematoma and neurological deficits.

OTHER

Anterior uveitis (iridocyclitis) occurs in 20%–40%, psoriasis in 9% and inflammatory bowel disease in up to 6%.

PREOPERATIVE ASSESSMENT

Consider possible airway involvement and extra-articular manifestations of the disease. Document any preoperative neurological deficits. Assess the range of movement of all joints to plan optimal positioning. Investigations are given in Table 8.1. In addition, consider echocardiography and arterial blood gas analysis.

There is a significant association between infectious complications following orthopaedic surgery and treatment with anti-TNF-α agents although the perioperative continuation of anti-TNF-α agents is not a significant risk factor for surgical wound infections and infection risk appears not to be a reason to withhold therapy.

If fibre-optic intubation is to be performed or difficulty with intubation is anticipated, then an antisialogogue should be prescribed.

INTRAOPERATIVE MANAGEMENT

Careful airway management is paramount. Difficult intubation should be expected and can be compounded

Table 8.1 Preoperative investigations for patients with AS and rheumatoid arthritis

Investigations	Reason
Full blood count	Anemia normochromic, normocytic (severe, hypochromic; gastrointestinal bleed)
Urea and electrolytes	Abnormal (iatrogenic – gold, cyclosporin)
ECG	Heart block Ischaemic (arteritis) Left ventricular hypertrophy (valvular heart disease)
Chest X-ray	Rheumatoid nodules
Cervical spine X-ray	Atlantoaxial subluxation (Lateral subatlanto axial subluxation and odontoid views)
Spirometry	Restrictive flow pattern
Indirect laryngoscopy	Degree of cricoarytenoid involvement

further when the temporomandibular joint is involved. There is significant risk of neurological injury and vertebrobasilar insufficiency with any excessive neck extension. Injuries to the cervical spine and spinal cord such as dislocation of C6 vertebra and quadriparesis after an emergency intubation have been reported. Fixed cervical flexion deformities limit access to the trachea and tracheostomy may be impossible. Neck supports should be used during anaesthesia and forcible movements of the neck in the presence of neuromuscular blockade avoided. Awake fibre-optic intubation is recommended. It also allows for constant neurological monitoring during placement of the tracheal tube. Retrograde intubation may also be considered.

A laryngeal mask may be considered in patients who do not require intubation. It may not be possible to place a laryngeal mask when the mouth opening is <1.2 cm and if a fixed extension deformity and large cervical osteophytes are present.

The 'intubate at all costs' approach is not appropriate for elective surgery, and if maintaining the airway is reasonably easy a laryngeal mask is useful. If airway maintenance is difficult or impossible, then allowing the patient to wake up and postponing surgery is prudent. If an emergency tracheostomy/cricothyrotomy should become necessary, remember that in the presence of a severe flexion deformity this may be impossible.

There are no specific contraindications to the use of any anaesthetic agent. Care should be taken with regard to patient transfer and positioning in order to avoid vertebral or neurological damage and minimize backache postoperatively. Ensure that the patient has full control of the airway prior to extubation as reintubation may prove very difficult. Postoperative ventilation in the ICU may be required.

Central neuraxial blocks may be difficult due to ankylosis of the intervertebral joints. Caution is advised due to existing neurological complications of the disease. The paramedian approach may be used and the number of attempts limited. AS has been reported as an independent risk factor for spinal haematoma after epidural anaesthesia. Continuous peripheral nerve blocks have much to recommend them and should be considered. The peripheral catheter is very useful for postoperative analgesia and will reduce the need for opioids.

Aortic or mitral incompetence should be treated as in primary cardiac disease, with caution in the use of vasodilating drugs. Antibiotic cover may be appropriate and preoperative pacing may be necessary.

POSTOPERATIVE MANAGEMENT

Continuous peripheral nerve block analgesia is a recommended option. The use of opioids must be balanced against the risk of oversedation and compromising the airway. PCA may be helpful. Reestablishment of regular NSAID therapy will help to relieve pain, which may be greater than the surgical pain.

Physiotherapy, breathing exercises and early mobilisation should be instituted because patients are at increased risk of respiratory complications. The effects of fluid shifts and the effects of medications on peri-operative fluid balance should be monitored.

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CROSS-REFERENCES

Restrictive lung disease, Chapter 1
Aortic valve disease, Chapter 2
Conduction defects, Chapter 2
Mitral valve disease, Chapter 2
Difficult airway, Chapter 26
Regional blocks, Chapter 29

DWARFISM

Individuals with short stature conventionally are divided into two categories: those with proportionate growth and those with disproportionate growth. It is the latter group that are classified as 'dwarfs'. Patients with dwarfism are often considered as having a single disease entity, but this is an oversimplification. Over 100 different types of dwarfism exist, many of which pose specific anaesthetic problems.

Although each particular type of dwarfism is relatively rare, the large number of types means that any practising anaesthetist is likely to meet these patients. Achondroplasia is the most common with an incidence of 1:10,000 to 1:40,000. There is a defect of fibroblast growth factor receptor which affects endochondral bone formation (long bone growth) while membranous and periosteal bones are unaffected. Achondroplasia is congenital and hereditary (autosomal dominant). Approximately 80%–90% of cases are probably new mutations.

ANAESTHESIA

Monitoring and anaesthetic techniques need to be considered in relation to both the underlying diagnosis as well as the planned surgical procedure.

Traditionally, general anaesthesia has been the technique of choice despite the risks of airway obstruction with some syndromes. This is in part due to the difficulties encountered in performance of spinal and epidural blockade: poor landmarks, spinal deformities, lumbar lordosis, spinal stenosis and narrow epidural space. Nevertheless, there are numerous reports of successful spinal and epidural anaesthesia for Caesarean section. Decreased volumes of local anaesthetic are needed and epidural may be preferable to spinal as the dose can be titrated to block height. Continuous peripheral nerve blocks may be challenging, but can be successfully performed.

PREOPERATIVE ASSESSMENT

RESPIRATORY SYSTEM

- Clinical assessment of airway: narrow nose-passage, intraoral anatomical changes
- Obstructive airway lesions, especially in patients with mucopolysaccharidoses
- Previous problems with airway maintenance or intubation
- Positioning problems due to reduced mobility of the atlanto-occipital joint – risk of atlanto-occipital luxation
- Restrictive defects secondary to rib hypoplasia and kyphoscoliosis
- Sleep apnoea

CARDIOVASCULAR SYSTEM

- Pulmonary hypertension
- Congenital heart disease
- Coronary artery disease
- Valvular heart disease
- Cardiomyopathy

NEUROLOGICAL SYSTEM

- Macrocephaly and hydrocephaly
- Cervical spine instability
- Spinal cord compression
- Nerve root compression
- Temperature regulation problems

OTHERS

- Endocrinopathy (hyperglycaemia)
- Prone to infections
- Bleeding problems
- Renal function
- Positioning damages due to deformities

Premedication is not usually necessary. Anticholinergic agents can be considered in patients with marked secretions, and those in whom problems with the airway or intubation are anticipated. Avoid sedatives in patients with potential upper airway obstruction.

INTRAOPERATIVE MANAGEMENT

VENOUS ACCESS

Peripheral and central access may be challenging. Patients are frequently obese and may possess subcutaneous infiltrates with lax skin. Cervical abnormalities, including very short necks and, frequently, stabilization devices, may make access to the jugular vein difficult. There may be no option but to use either a femoral or subclavian approach.

MONITORING

Due to the different proportions, noninvasive blood pressure cuffs may lead to wrong measurements. In the case of hydrocephalus, a possible rise in intracranial pressure must be anticipated.

INDUCTION

Awake intubation or inhalational induction with maintenance by spontaneous respiration are thought to be relatively safe techniques. Nasal intubation should be avoided. Restrictive lung disease, if present, will prolong an inhalational induction. Muscle relaxants should be avoided until it is certain that the patient can be ventilated.

INTUBATION

Exposure of the larynx and intubation may be impossible with a conventional laryngoscope. A short-handled laryngoscope may assist but if this fails then fibre-optic guided intubation either awake or under general anaesthesia will be required. In patients with foramen magnum stenosis or atlantoaxial instability avoid neck movements during attempts at intubation. Under these circumstances fibreoptic intubation may be preferable.

There is some controversy as to the selection of correct size of tracheal tube. For achondroplastics the formula

$$(Age/4) + 4$$

usually correctly predicts the internal diameter. In extreme circumstances, a tracheostomy may be necessary, but in patients with mucopolysaccharidoses this may not completely relieve the tracheal obstruction due to distal tracheal distortion.

RESPIRATORY SUPPORT

The low FRC and high closing volume frequently found in patients with respiratory involvement predisposes these patients to atelectasis and V/Q mismatching. This may cause problems with oxygenation. For all but the shortest and simplest surgical procedures, arterial cannulation is strongly recommended for any patient with respiratory dysfunction.

CARDIOVASCULAR PROBLEMS

Cardiology opinion may be required to determine the extent of cardiac compromise. Pulmonary hypertension is common. Right ventricular enlargement can best be confirmed by echocardiography. In patients with pulmonary hypertension, the anaesthetic has to be planned to avoid pulmonary arterial vasoconstriction whilst still maintaining adequate cardiac output. Care must be taken to avoid hypoxia, hypercapnia and respiratory or metabolic acidosis which can cause profound rises in pulmonary artery pressures. In mildly affected individuals, oxygen and inhalational anaesthetics are often used. In patients with right ventricular failure, high-dose narcotic techniques are preferred. In children, ketamine

has been safely used, even in cases with right ventricular failure, although this is not recommended in adults. In patients with congenital heart lesions, or corrected lesions, endocarditis prophylaxis may be needed.

NEUROLOGICAL PROBLEMS

Most problems revolve around the stability of the cervical spine, especially at the atlantoaxial and craniocervical junctions, and problems with raised intracranial pressure (ICP). In patients with spinal cord compression an autonomic hyper-reflexic state may develop. Document any preexisting neurological deficit if central blockade is considered.

The problems of anaesthetising a patient with raised ICP are formidable: an inhalational induction can be associated with hypercapnia and a rise in ICP, whilst an intravenous induction can be associated with apnoea in a patient who cannot be intubated or ventilated. These patients thus require consideration on a case-by-case basis.

Rarely, hyperthermia, usually without the clinical features of malignant hyperthermia (MH), develops. Therapy consists of simple cooling measures alone. A few cases that are clinically indistinguishable from MH have been observed in patients with osteogenesis imperfecta. It was only by muscle biopsy that the diagnosis was able to be refuted. Thus, potential trigger agents do not need to be avoided unless MH is clinically suspected.

BLEEDING PROBLEMS

Osteogenesis imperfecta is the only chondrodystrophy associated with a coagulopathy. These patients require formal evaluation with a bleeding time preoperatively. Platelets and fresh frozen plasma should be available.

POSTOPERATIVE MANAGEMENT

A period of postoperative management on the HDU or ICU may be required. Postoperative ventilation, which can be prolonged, may be required, especially in patients with thoracic dystrophy.

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CROSS-REFERENCES

MARFAN SYNDROME

First described in 1896, the incidence is 1 out of 10,000 live births in the United States with no gender preference. The inheritance is autosomal dominant, with 25% incidence of spontaneous mutation and clinical variability with complete penetrance. The syndrome is due to mutations in gene *FBN1* encoding fibrillin-1 on chromosome 15q21.1. Clinical diagnosis requires a family history of Marfan syndrome with involvement of an organ system, but increased

organ involvement is needed for the diagnosis if there is no family history.

PATHOPHYSIOLOGY

CARDIOVASCULAR

Cardiac abnormalities are the most serious medical complication. Historically, the mean age of survival was 43 years for males and 46 years for females, but the average life span has now been extended to 72 years with early deaths occurring at an average age of 41 years. Mitral valve insufficiency, aortic valve insufficiency and both ascending and descending aortic dilatation may lead to a dissecting aneurysm which is the most frequent cause of death. Congenital Marfan syndrome is a particularly serious variant with an 80% incidence of cardiac abnormalities and a 14% mortality rate during the first year of life.

SKELETAL

The long bones are slender, with long limbs. Arm span usually exceeds the patient's total height. There is joint laxity and arachnodactyly with abnormal lengthening of the digits. The 'thumb sign' is positive when the nail of the thumb in a clenched fist extends beyond the ulnar border of the small finger. Another sign of arachnodactyly is the 'wrist sign', in which the patient encircles the wrist with their contralateral hand and the thumb overlaps the small finger. Protrusio acetabuli is another feature.

Significant scoliosis is common and spine deformity occurs early. Dural ectasia is common. Approximately 63% of patients were reported to have scoliosis and >50% kyphosis; 12% developed progressive scoliosis requiring surgery. Further complications may include instrumentation fixation failure, pseudarthrosis and curve decompensation.

RESPIRATORY

Pectus carinatum or excavatum, and scoliosis can further contribute to thoracic insufficiency volume as well as depletion deformity of the thorax from spine rotation. There are also intrinsic lung problems. Spontaneous pneumothorax and bronchospasm may be a problem and pulmonary function is often adversely affected.

OCULAR

These include lens dislocation, myopia, retinal detachment, glaucoma and cataracts.

PREOPERATIVE ASSESSMENT

Pay particular attention to cardiopulmonary investigations including chest X-ray, ECG, echocardiography, lung function tests and arterial blood gases. When aortic dilatation exceeds 5–5.5 cm in adults, prophylactic graft replacement of the aortic valve and ascending aorta may be necessary. Vital capacity and FEV_1 may appear to be lower than expected when compared with predicted values, due to greater height or arm span. Before surgery, patients should be screened for platelet count, PT and PTT.

PERIOPERATIVE MANAGEMENT

Prophylactic antibiotics are recommended due to the high risk of bacterial endocarditis. In patients undergoing spine operations a deep infection rate of 10%, dural tears in 8% and a mean blood loss of 2400 mL for scoliosis and 3000 mL for kyphosis has been reported. Careful handling and positioning are essential to avoid joint trauma and dislocation.

Continuous nerve blocks are recommended in order to avoid any intervention on the airways and a good control of the surgical-induced stress reaction. Intubation may be difficult due to the long, high arched palate. Gentle laryngoscopy should be performed to avoid cervical spine and temporomandibular joint damage.

Surges of blood pressure should be avoided, e.g. on laryngoscopy or in response to surgical stimulation. Beta blockade will reduce aortic wall tension. Blood pressure should be maintained with the diastolic pressure high enough to ensure good coronary flow, but not too high so as to risk dissection. There may be little cardiac reserve, and volatile agents may be very depressant.

Spontaneous pneumothorax may become a tension pneumothorax in a patient on positive pressure ventilation. Maintain low airway pressures and avoid overinflation of the lungs.

The risk of malignant hyperthermia may be increased; therefore, the use of intravenous agents is preferred.

In addition to minimal monitoring standards, capnography, airway pressures, arterial cannulation (but increased risk of morbidity because of weak arterial wall) and temperature are recommended.

The choice of anaesthetic technique is broad and no one agent or technique is suggested. Following careful intravenous induction while monitoring blood pressure, anaesthesia may be maintained with a volatile agent. Blood pressure may be further controlled by beta blockade, if needed. Care must be taken to maintain intravascular volumes and filling pressure.

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CROSS-REFERENCES

METABOLIC AND DEGENERATIVE BONE DISEASE

OSTEOMALACIA

Osteomalaci is a metabolic disease of the bone in which normal bone is replaced by unmineralized osteoid. When this condition occurs in children, the disease is called rickets. Clinically it can be extremely difficult to differentiate osteomalacia and osteoporosis. The finding of a low serum phosphate suggests osteomalacia, but the only certain diagnostic method is to take a bone biopsy.

Osteomalacia is caused by an inadequate level of 1,25-dihydrocholecalciferol (1,25-DHCC), an active metabolite of vitamin D. The most common cause is deficiency of vitamin D due to diet or inadequate exposure to sunlight or, less commonly, malabsorption. Severe renal disease is a potent cause of osteomalacia because 25-hydroxycholecalciferol is exclusively converted to the active 1,25-DHCC in the kidney. Osteomalacia may develop in patients on long-term therapy with drugs that induce hepatic mixed function oxidase, because this interferes with vitamin D metabolism. Clinical features include bone pain, pathological fractures and proximal myopathy.

Treatment of the underlying cause and replacement of vitamin D is important. This may be administered either as calciferol or the active metabolite 1-α-cholecalciferol. Replacement therapy must be closely monitored since there is a risk of hypercalcaemia developing. Calcium supplements are only used if the patient is hypocalcaemic.

ANAESTHETIC CONSIDERATIONS

- Abnormal drug metabolism if mixed function oxidase induced
- Potential for hypercalcaemia if on therapy with vitamin D supplements
- Great care with positioning (fractures can easily occur)
- Deformity, if occurs before epiphyseal fusion

- Hypocalcaemia – may increase nondepolarising muscle relaxant duration
- Chronic renal failure

OSTEOPOROSIS

The overall quantity of bone is reduced, whilst its shape, composition and morphology remain normal. It generally occurs in the elderly, especially women. The most common precipitating factor is menopausal withdrawal of oestrogens. Endocrinopathies, long-term corticosteroid therapy, smoking, alcohol, poor nutrition/malabsorption and immobilization can also result in osteoporosis. The net effect is a relative overactivity of the osteoclasts, leading to bone loss. Because of the bone loss, fractures occur more readily, frequently after minimal trauma.

Common sites of fractures include vertebrae (crush or wedge fractures), neck of femur, distal radius, proximal humerus and the pelvis. Multiple vertebral fractures leading to a kyphosis are common. This may be associated with marked respiratory impairment. It is unusual for vertebral fractures to be associated with serious neurological sequelae, although sciatica is common. Patients may require surgical stabilization, but any immobilization tends to worsen the condition.

PAGET'S DISEASE

Paget's disease is a metabolic disease of unknown aetiology where both environmental and genetic factors have been implicated. Several genetic loci have been linked to this disease and three genes have been identified in these loci. It is characterized by excessively rapid remodelling of bone. There is intense resorption of bone by abnormal osteoclasts and the new bone formed by osteoblasts is architecturally distorted and its mineralization is defective. The affected bones and bone marrow are initially very vascular. Eventually, the bone may become dense and hard with a reduced vascularity. These sclerotic areas are weak and fractures are common.

The incidence is ~5% over 55 years and is familial. The most frequently affected sites are the pelvis, femur, tibia, skull and spine. Because of the involvement of the skull and spine, spinal cord compression, atlantoaxial instability and brain-stem compression may develop.

Patients may be asymptomatic, but commonly bone pain or fractures are the presenting feature. Occasionally, patients present in high output cardiac failure due to the increased bone vascularity. One percent of patients develop bone sarcoma.

Calcitonin, which acts primarily as an inhibitor of bone resorption, has been used in patients with bone pain and before orthopaedic procedures to reduce the vascularity of the bone. Increasingly, the bisphosphonates (e.g. alendronate) are being used to control bone pain, their effect often far outlasting the duration of treatment. They are adsorbed onto hydroxyapatite crystals, so slowing both their rate of growth and dissolution, and reducing the rate of bone turnover.

ANAESTHETIC CONSIDERATIONS

- Careful evaluation for atlantoaxial and craniocervical instability.
- Assess lung function in patients with a kyphosis.
- Cardiac failure, if present, must be treated.
- Careful moving and positioning (fractures occur very easily).
- General or regional techniques may be used. Spinal and epidural placement can be difficult. Most dental cases are performed under general anaesthesia because extractions are difficult and the risk of postoperative bleeding is increased.
- Corticosteroid treatment may be required, depending upon previous treatment.
- Consideration should be given to the use of calcitonin before major orthopaedic procedures.

OSTEOARTHRITIS

Osteoarthritis is a common degenerative disease of the joint surface. There is damage to hyaline cartilage, with sclerosis and osteophyte formation in underlying subchondral bone. This leads to a reduced joint space. The aetiology is unclear, but may be related to joint trauma and joint overuse. Osteoarthritis is

a major cause of disability and is universally evident after age 60.

Patients usually complain of pain, worse with movement and at the end of the day and of stiffness that improves with use. Characteristically, the hip and knee joints are involved, but there may be involvement of the distal interphalangeal joints and degeneration of the spine. The middle and lower cervical spine and lower lumbar spine are the areas most likely to be involved. Spinal cord compression or nerve root compression can occur due to degenerative discs. Spinal fusion is rare.

Treatment is symptomatic, using physiotherapy and NSAIDs. Corticosteroids are not used since they are associated with a worsening of the degenerative process. Reconstructive joint surgery has much to offer but can be associated with considerable blood loss and carries a high risk of thromboembolic phenomena. The use of regional anaesthesia for these procedures, either alone or in combination with general anaesthesia, has been shown to reduce the blood loss and to decrease the incidence of deep venous thrombosis from 33% to 9%. Graduated compression stockings and thromboprophylaxis are also used in an attempt to further reduce the incidence of thromboembolism.

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CROSS-REFERENCES

Regional blocks, Chapter 29
Epidural and spinal anaesthesia, Chapter 29
Repair of fractured neck of femur, Chapter 22

RHEUMATOID DISEASE

Rheumatoid disease (RA) is a common systemic chronic inflammatory disease affecting up to 3% of women and 1% of men in the UK with an onset typically between 30 and 50 years. It is HLA DR4–linked and thought to be an autoimmune condition perhaps triggered by an infectious agent. It usually affects multiple joints symmetrically. Virtually every organ can be affected by the disease and compelling evidence exists to relate active and severe RA. Patients with RA may develop anaemia, systemic complications and other coexisting autoimmune disorders.

PATHOPHYSIOLOGY

MUSCULOSKELETAL

The destructive synovitis attacks the small joints of the hands, ankles, knees, temporomandibular joints, wrists, elbows and joints of the spinal column. The disease progresses until the inflammation eventually moves into a fibrotic phase, leaving characteristic fixed deformities. Involvement of the cervical spine is common and is associated with poor outcomes. Approximately 40%–85% of patients with RA develop neck pain and radiographic evidence of instability (atlanto-axial subluxation, and superior migration of the odontoid process) and 50% of these patients are asymptomatic. Involvement of the temporomandibular and cricoarytenoid joints is also commonly seen.

RESPIRATORY

There is a decrease in vital capacity, total lung volume and arterial hypoxaemia. Pleural disease occurs in 3%–12.5% of patients and more commonly in men. Rib-cage stiffness and interstitial lung fibrosis combine to produce a restrictive picture. Other pulmonary manifestations include the presence of rheumatoid nodules in the lungs that may rupture or cavitate and become sites of infection, pleural effusions (typically unilateral) and rarely fibrosing alveolitis. Pulmonary vasculitis should be considered as a potential cause of pulmonary hypertension. Iatrogenic pulmonary disease occurs in up to 5% of patients treated with methotrexate, and appears as a progressive interstitial fibrosis. Sulphasalazine therapy may lead to the development of eosinophilic pneumonitis.

CARDIOVASCULAR

The prevalence of cardiovascular disorders has been estimated to be up to 40%, with pericardial disease being the most common. Approximately 1%–5% have mitral valve disease, with other valves being less commonly involved. Conduction abnormalities have been reported. Pericarditis andmyocarditis can also develop. Rheumatoid vasculitis can be widespread, life-threatening and refractory to treatment. It can lead to skin ulcerations, gastric bleeding or small bowel ulcerations and neuropathies with the nerve problems causing pain, numbness or tingling.

HAEMOPOIETIC

A mild normocytic anaemia is common and tends to correlate with disease activity. It is important that other causes of anaemia are excluded, in particular bleeding from the gastrointestinal tract secondary to either steroid or NSAID therapy. The normal responses to infection may not be present due to concomitant immunosuppressive therapy. Methotrexate, sulphasalazine, gold, azathioprine and penicillamine may induce bone marrow suppression.

OTHER

Gastrointestinal symptoms are generally secondary to drug therapy, NSAIDs and steroids causing ulceration, azathioprine leading to nausea and vomiting and possibly even pancreatitis, and oral gold therapy causing irritation of the gut.

Renal and hepatic failure often occur as a result of amyloidosis or drug therapy. Subclinical renal and hepatic dysfunction is common. Renal impairment may occur secondary to gold, penicillamine, ciclosporin or NSAIDs, or rarely, amyloidosis.

Methotrexate, sulphasalazine, azathioprine, gold and ciclosporin can all cause hepatotoxicity. Methotrexate is also responsible for haematological and pulmonary side-effects, e.g. pancytopaenia and irreversible pulmonary fibrosis. Treatment with leflunamide has been associated with disturbances in the gastrointestinal tract and with the development of peripheral neuropathy. Etanercept has also been implicated in acute lung injury and polyneuropathy due to demyelinisation of nerve fibres.

Neurological complications include peripheral neuropathy (mainly sensory), mononeuritis multiplex, entrapment neuropathy (e.g. carpal tunnel syndrome) and spinal cord lesions secondary to cervical disease.

Infections of all kinds are more common in rheumatoid disease especially joint infections.

PREOPERATIVE ASSESSMENT

- Neck movement/mouth opening/dentition
- Veins/arteries/bruising
- Presence of painful joints and limitations
- Suitability for regional technique
- Complete neurological, respiratory and cardiovascular history
- Drug history
- Previous anaesthetic problems
- Investigations – see Table 8.2

PERIOPERATIVE MANAGEMENT

Consider performing the procedure under local or regional blockade if feasible. However, the involvement

Table 8.2 Preoperative investigations for patients with rheumatoid arthritis

Investigations	Result
Full blood count	Anemia normochromic, normocytic (severe, hypochromic; gastrointestinal bleed)
Urea and electrolytes	Abnormal (iatrogenic – gold, ciclosporin)
ECG	Heart block
	Ischaemic (arteritis)
	Left ventricular hypertrophy (valvular heart disease)
Chest X-ray	Rheumatoid nodules
Cervical spine X-ray	Atlantoaxial subluxation (Lateral subatlanto axial subluxation and odontoid views)
Spirometry	Restrictive flow pattern
Indirect laryngoscopy	Degree of cricoarytenoid involvement

of the spine may make epidural or spinal anaesthesia difficult if not impossible. The need of sedation (propofol and remifentanil) is often necessary because of pain due to positioning on the operating table.

If general anaesthesia is to be used, then the cervical spine and airway are the areas likely to cause most concern. The development of fibre-optic laryngoscopes has altered the management of rheumatoid patients when general anaesthesia with intubation is considered necessary.

If fibre-optic intubation is to be performed or difficulty with intubation is anticipated, then an antisialogogue should be prescribed.

CERVICAL SPINE

On induction of anaesthesia, the cervical spine will lose any protective tone around the unstable neck, and thus it is important to determine the range of comfortable neck movement before induction and limit it to this with the use of sandbags, etc. If tracheal intubation is necessary, consider awake fibre-optic intubation, especially if there is posterior atlantoaxial subluxation (neck extension potentially hazardous).

Where intubation is not required, then oropharyngeal or nasopharyngeal airways may reduce the amount of cervical manipulation required. The laryngeal mask is useful in longer procedures, although the larynx may be displaced in cervical spine disease, making placement difficult. Use manual-in-line stabilisation during airway manipulation in unconscious patients unless certain cervical spine is stable.

AIRWAY

Temporomandibular joint involvement may lead to difficulty in mouth opening and forward jaw protrusion, thus leading to difficulty in inserting a laryngoscope as well as viewing the larynx. Rarely, cricoarytenoid involvement can result in acute airway obstruction. Anticipated problems or previous difficulty should lead to early consideration of the fibre-optic laryngoscope.

ANAESTHESIA

There are no restrictions on anaesthetic agents used in rheumatoid disease, although iatrogenic, hepatic or renal disease may alter the amount of free drug available and increments should be administered with care. Great care should be taken to protect the joints during anaesthesia, with careful handling and positioning of the patient and protection of pressure points. Mechanical ventilation may be necessary in those patients with severe pulmonary disease.

POSTOPERATIVE MANAGEMENT

Analgesia is the main problem as these patients tend to be more sensitive to opioids. Patient-controlled analgesia (PCA) may be difficult for the rheumatoid patient due to hand deformities although special modifications are available. Continuous regional analgesia may provide good analgesia if appropriate.

Early physiotherapy is indicated, both to prevent chest infections in a patient who has a restrictive lung defect, and thus a propensity to develop atelectasis, and for a patient who is more difficult to mobilize due to musculoskeletal dysfunction.

Steroid cover should be continued where indicated, and there should be close monitoring of renal function, especially if preoperative dysfunction was present.

These patients tend to require multiple surgical procedures due to the relentless progression of their disease. Careful attention should be paid to previous anaesthetic notes and any problems with intubation, analgesia, etc. should be noted.

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CROSS-REFERENCES

Difficult intubation, Chapter 26
Mitral valve disease, Chapter 2
Anaemia, Chapter 7
Iatrogenic adrenocortical insufficiency, Chapter 6
Arthroscopy, Chapter 22

SCOLIOSIS

Scoliosis is a fixed, structural, lateral curvature of the
spine with associated rotation of the vertebrae. It can
be congenital or acquired.

Congenital scoliosis may present at any age and
is a result of either failure of vertebral segmenta-
tion (a *bar*) or failure of formation (a *hemivertebra*).
Congenital scoliosis is often part of a generalized
condition, such as Goldenhar syndrome or spina
bifida, and may be associated with abnormalities in
renal, cardiac, respiratory or neurological systems.
The indication for surgery is documented progres-
sion at any age.

Acquired scoliosis is mainly idiopathic. Infantile
onset idiopathic scoliosis (scoliosis before the age of
8 years) carries the most serious prognosis and if left
unchecked is likely to result in cardiopulmonary fail-
ure in middle age.

Scoliosis is a sign, not a disease. It may arise
from several different causes; although presentation,
complications and management may be similar, the
prognosis may differ greatly for different aetiologies
(Table 8.3).

CLASSIFICATION

FUNCTIONAL SCOLIOSIS

Functional scoliosis is secondary to discordant leg
length, etc. Curve disappears when patient lies down.

Table 8.3 Aetiology of structural scoliosis (relative frequencies)

Idiopathic (70%)	
Congenital	Abnormal spinal cord/vertebral development
Neuromuscular (15%)	Neuropathic: cerebral palsy, syringomyelia, poliomyelitis. Myopathic: muscular dystrophies, neurofibromatosis, Friedrich's ataxia
Mesenchymal disorders	Rheumatoid arthritis, Marfan syndrome, osteogenesis imperfecta
Metabolic bone disease	Osteoporosis, Paget disease
Malignancy	Primary and secondary tumours
Trauma/surgery	Fracture, radiotherapy, surgery
Infection	Tuberculosis, osteomyelitis

STRUCTURAL SCOLIOSIS

- *Congenital* – Associated with vertebral
  anomalies. May have abnormalities of the heart
  and genitourinary tract.
- *Idiopathic* – 60%–80% of cases; has the best
  prognosis. This is a diagnosis of exclusion.
- *Neuromuscular* – Occurs secondary to a
  neuropathy, e.g. cerebral palsy, poliomyelitis,
  Friedreich's ataxia, or myopathy, e.g. Duchenne's
  muscular dystrophy. This group includes many
  of the syndromes associated with scoliosis,
  e.g. Prader–Willi syndrome.

OTHER CAUSES

- *Mesenchymal* – Abnormalities of the tissues,
  e.g. Marfan's syndrome, Ehlers–Danlos
  syndrome
- Trauma
- *Tumours* – Intraspinal or skeletal
- *Metabolic* – Rickets, hyperphosphatasia

The degree of scoliosis is defined by the Cobb
angle, which is measured from a standing anteropos-
terior radiograph of the spine. The first line is taken

from the most tilted vertebral body above the scoliosis and extended laterally. The second line is taken from the most tilted vertebra below the scoliosis. The measured angle is at the intersection of these two lines. Although occasional patients may have a principally lumbar scoliosis, the thoracic vertebrae are more commonly involved. The larger the angle, the more severe is the scoliosis and the greater the likelihood of compromised respiratory and cardiovascular function. Patients with neuromuscular type scoliosis may have significant impairment despite lesser abnormalities.

PATHOPHYSIOLOGY

RESPIRATORY SYSTEM

Respiratory impairment usually shows a restrictive pattern, with lung volumes being related inversely to the angle of curvature. Vital capacity is the most severely affected, but total lung capacity and FRC are also reduced. Abnormalities of rib-cage development cause abnormal development of the underlying lung, with alveolar volume being compressed to, or below, FRC. There is mechanical disadvantage of muscles of respiration and reduced chest wall compliance. This results in alveolar hypoventilation and reduced P_aO_2.

There is restricted development of the pulmonary vascular bed and diversion of blood into high-resistance extra-alveolar vessels. The resulting ventilation/perfusion mismatch causes an increased alveolar to arterial oxygen gradient and exacerbates alveolar hypoventilation. An increase in P_aCO_2 occurs late and is a poor prognostic indicator. In addition, patients with more severe scoliosis have a decreased respiratory sensitivity to elevated P_aCO_2.

CARDIOVASCULAR SYSTEM

There is a relatively high incidence of congenital heart disease. Thoracic scoliosis may result in right-sided cardiac problems. The ventilation/perfusion mismatch causes an increased pulmonary vascular resistance. Low lung volumes, chronic hypoxia and abnormal development of the pulmonary vascular bed all contribute to these changes. Right atrial dilatation and right ventricular hypertrophy are late in appearance.

PREOPERATIVE ASSESSMENT

Patients with congenital or neuromuscular causes of scoliosis, such as muscular dystrophy or cerebral palsy, may be at risk for more aggressive progression of scoliosis, and therefore may warrant early surgical treatment.

Surgical correction is aimed at partial straightening of the curvature, stabilization of the spine and cessation of further progression of scoliosis. Hardware is secured segmentally to the vertebral pedicles, followed by partial straightening (distraction) of the spine with rigid rods that are manipulated to achieve the desired degree of lesser curvature.

Assessment involves determining the aetiology of the scoliosis and associated problems (Table 8.4).

Table 8.4 Suggested preoperative investigations before major spinal surgery

	Minimum investigations	Optional investigations
Airway	Cervical spine lateral X-ray with flexion/ extension views	CT scan
Respiratory system	Plain chest radiograph	Pulmonary function tests
	Arterial blood gas analysis	Pulmonary diffusion capacity
	Spirometry (FEV_1, FVC)	
Cardiovascular system	ECG	Dobutamine-stress echocardiograph
		Echocardiography
		Dypiridamole/thallium scintigraphy
Blood tests	Full blood count	Liver function tests
	Clotting profile	
	Blood cross match	
	Urea, electrolytes	
	Albumin, calcium (neoplastic disease)	

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RESPIRATORY FUNCTION

- Clinical assessment of airway and intubation may be difficult, e.g. fixed flexion deformities, large meningomyelocele, 'halo' or other neck immobilising traction.
- Exercise tolerance may be impaired.
- Verify chest expansion and ability to cough. Consider preoperative physiotherapy.
- Formal lung function tests are recommended.

CARDIAC FUNCTION

- Look for right ventricular enlargement, loud pulmonic second sound and murmur of pulmonary insufficiency.
- ECG – P wave >2.5 mm and R>S in V_1 and V_2. These changes are rare and occur late.
- Echocardiography – a more sensitive detector of cardiac abnormalities secondary to pulmonary hypertension.

INTRAOPERATIVE MANAGEMENT

See Boxes 8.2 and 8.3.

MONITORING

- Arterial cannulation is useful. It is mandatory for patients with neuromuscular scoliosis.
- *Core temperature* – There is a higher incidence of malignant hyperthermia.
- *Nasogastric tube* – Decompression of the stomach assists ventilation, and distraction of the spine may be associated with the development of paralytic ileus.
- Consider central venous pressure, oesophageal Doppler and urine output monitoring, particularly for surgery associated with high blood loss.

ANAESTHESIA – SPECIFIC CONSIDERATIONS

- Intravenous induction is appropriate. Avoid suxamethonium to prevent triggering malignant hyperthermia in susceptible patients, prevent hyperkalemia in patients with neuromuscular problems and rhabdomyolysis and myoglobinuria in patients with myopathies.
- Blood loss may be considerable and rapid. Hypotensive techniques may produce spinal cord ischaemia. Consider cell salvage or haemodilution. Use fluid warmers and warming blanket. Patients with Duchenne's muscular dystrophy and central paresis appear to bleed more.
- Postoperative pain, hypoventilation and atelectasis are severe following thoracotomy. Thoracic epidural analgesia provides good operating conditions and excellent pain relief and may be continued for several days postoperatively.
- Anaesthesia must facilitate spinal cord monitoring ('wake-up' test, sensory or motor evoked potentials). Total intravenous anaesthesia using propofol and remifentanil infusions is a suitable technique.
- Prone position is used for the posterior approach. Careful positioning is required including abdominal decompression (decreases blood loss).

NEUROLOGIC RISKS ASSOCIATED WITH SCOLIOSIS SURGERY

Neurologic impairment, particularly paraparesis or paraplegia, is an infrequent, but potentially devastating complication. As with other types of spine surgery, the spinal cord and nerve roots are at risk for mechanical injury as hardware is affixed to the vertebral pedicles. The likelihood of misdirection of hardware is probably higher in scoliosis surgery due to the abnormal curvature and rotation of the vertebrae and the risk of spinal cord injury is probably higher because the spinal cord is often situated very close to the concave wall in the scoliotic spine. Certain techniques require passing of sublaminar hardware through the epidural space, further exposing the spinal cord to potential trauma.

The greatest risk of spinal cord injury occurs during distraction of the spine. Injury may result from stretching or compression of the cord, or of the anterior spinal artery or its feeding radicular vessels causing spinal cord ischaemia and infarction.

BOX 8.2: Anaesthetic considerations for spinal surgery in children

Preoperative considerations
- Evaluation of all organs systems to pick up other congenital defects
- Evaluation of the nervous system and documentation of neurological deficit
- Evaluation for anatomical abnormalities leading to 'airway challenge'
- Specific age-related anaesthetic considerations
- Awareness of associated pathology (Arnold-Chiari malformation, CP, etc.)
- Evaluation of respiratory and cardiovascular status
- Previous anaesthetic records
- Awareness of potential for latex allergy
- Psychological status
- Premedication as appropriate

Induction of anaesthesia
- Consider fibreoptic-aided intubation if anticipating a difficult airway or for unstable spinal injuries. Consider intubation in lateral position to protect the neuroplaque
- Secure adequate venous access
- Routine monitoring — include invasive BP monitoring where appropriate

Maintenance of anaesthesia
- Airway maintenance and securing the tube well
- May require the prone position
- Use any appropriate agents for induction and maintenance
- Spinal cord monitoring is often used
- Maintenance of spinal cord perfusion
- Prevent of hypothermia
- Maintain volume status
- Awareness of potential for blood loss — blood/products organized
- Blood conservation techniques where appropriate
- Antibiotic prophylaxis after discussion with surgical team

Monitoring
- Oximetry, capnography and gas monitoring
- ECG, BP (usually invasive) and core temperature
- Monitoring of neuromuscular block

Postoperative considerations
- High dependency/intensive care nursing
- Postoperative analgesia

The anterior spinal circulation supplies the descending corticospinal tracts and the anterior horn, and infarction in this territory may result in paraparesis or paraplegia. A higher rate of neurologic complications is seen in patients with hyperkyphosis, patients with a high degree of rigid curvature, patients with congenital scoliosis, neuromuscular scoliosis, or cerebral palsy, and patients undergoing combined anterior and posterior approaches.

Neurophysiologic intraoperative monitoring including somatosensory evoked potentials, motor evoked potentials, spontaneous electromyography and triggered electromyography is standard for scoliosis surgery.

POSTOPERATIVE CARE

- ICU or HDU admission is recommended
- Postoperative IPPV may be required
- Continuous oxygen therapy
- Regular physiotherapy
- Multimodal analgesia

BOX 8.3: Anaesthetic concerns for spinal surgery in neonates

Peculiarities of the neonatal respiratory control and mechanics
- Hypercarbia stimulates respiration to a lesser extent than in adults
- Hypoxia leads to sustained respiratory depression
- Tendency to periodic breathing and increased risk of postoperative apnea up to 60 weeks after conceptual age
- Neonates are at increased risk of atelectasis
- Diaphragm is susceptible to fatigue
- Increased risk of airway obstruction and thoracoabdominal asynchrony under anaesthesia

Characteristics of neonatal circulation and myocardial function
- Limited functional reserve
- Myocardium less able to generate force
- Myocardium more dependent on extracellular calcium
- Balance in favor of the parasympathetic system

Immature hepatic function
- Require adjustment of drug dosing intervals and maintenance dosing

Immature renal function
- Tolerate fluid restriction and fasting poorly

Higher incidence of intraoperative complications
- Dislodgement of the tracheal tube
- Tracheal tube obstruction
- Bronchial intubation
- Pneumothorax
- Failure of anesthetic equipment
- Hypothermia

The aims of surgery are

- Correction of curve
- Prevention of progression of curve
- Relief or prevention of back pain
- Prevention of neurological compromise
- Prevention of respiratory compromise
- Cosmetic

Surgery may be via an anterior approach, posterior approach or both (carried out as two separate procedures several days apart or during a single session). The anterior approach requires access to the vertebrae on the convex side of the curve. This is achieved via thoracotomy and costectomy and usually precedes posterior fusion. The posterior approach requires the patient to be placed prone.

Evidence of pulmonary hypertension or right ventricular hypertrophy carries a poor prognosis. Right ventricular failure must be treated before surgery. There is no specific contraindication to local or regional anaesthesia. Placement of one or two epidural catheters by the surgeon at the end of surgery is a very efficient way to control postoperative pain. Most patients who undergo scoliosis surgery are children or young adults.

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CROSS-REFERENCES

Muscular dystrophies, Chapter 6
Restrictive lung disease, Chapter 1
Marfan's syndrome, Chapter 8
Complications of position, Chapter 30
Preoperative assessment, Chapter 25

9 Connective tissue

JOHN-PAUL LOMAS

BULLOUS AND VESICULAR SKIN DISORDERS

PEMPHIGUS AND PEMPHIGOID

These rare autoimmune conditions are characterized by bullous eruptions of skin and mucous membranes. There are several variants. In pemphigus, blisters form intradermally; in pemphigoid, lesions occur at the dermal–epidermal junction.

Pemphigus vulgaris is the most common form and occurs predominantly in patients of Mediterranean or Jewish origin, with a peak incidence between 30 and 50 years of age. It was uniformly fatal before the advent of steroid therapy. There is a familial tendency and an increased incidence of HLA-13. There may be an association with other autoimmune diseases such as thymoma, myasthenia gravis and systemic lupus erythematosus. Large, superficial, flaccid blisters occur spontaneously or in response to trauma and are found in the groin, axillae and over the trunk. Oral lesions may predate cutaneous bullae by several months. Pressure with torsion may result in blister formation in normal looking skin (Nikolsky's sign). Bullae are fragile, rupturing easily with coincident loss of large areas of skin; healing occurs without scarring. Lesions may occur on the lips and in the mouth, nose, pharynx and larynx causing difficulty in eating and hoarseness. In some variants, e.g. pemphigus erythematosus and pemphigus foliaceous, bulla formation occurs more superficially within the epidermis. These forms tend to be less severe.

Pemphigoid includes both bullous and cicatricial types. Bullous pemphigoid is clinically similar to pemphigus, but occurs predominantly in patients over 50 years of age. If untreated, it follows a chronic relapsing course and the mortality is low.

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It is self-limiting and the patient's health remains good. Large, tense bullae develop often occurring on the inner aspects of the thighs, flexor surfaces of the forearms, axillae and groin and over the lower abdomen. The mouth may be affected but, unlike pemphigus, is rarely the initial manifestation. Blister formation is thought to result from the activation of complement in association with neutrophil and eosinophil migration.

Cicatricial pemphigoid is rare and primarily affects mucous membranes. Skin is involved in 10%–30%, but rarely in the absence of mucosal lesions. It is a chronic condition and usually heals with scarring. This may lead to nasal obstruction, dysphagia and laryngeal stenosis. Blindness may complicate ocular involvement.

Treatment is with steroids and, occasionally, other immunosuppressant drugs, e.g. methotrexate, azathioprine or cyclophosphamide. Lower doses of steroids are used in pemphigoid. Gold injections and plasmapheresis may also be used in pemphigus. Immunoglobulin is an effective rescue therapy where steroid treatment fails.

ERYTHEMA MULTIFORME (EM)

An acute, self-limiting eruption of skin and mucous membranes, it is characterised by distinctive target or iris lesions. Fifty percent have no identifiable cause but, for the rest, a wide variety of triggers, including infective agents, drugs and neoplasms, has been described. Those include barbiturates, antibiotics, anticonvulsants, antipyretics and cimetidine. Other common triggers are herpes simplex, haemolytic streptococcal infection, cancer and collagen vascular disease.

The pathogenesis is not fully understood, but it may be a hypersensitivity reaction. It may present in various forms (hence its name), ranging from a mild, self-limited, skin eruption through to Stevens–Johnson syndrome (SJS) with its systemic involvement and high mortality (5%–15%) if untreated. EM may present as symmetrical target lesions (dull red macules up to 2 cm in diameter with a clear centre) on the extensor surfaces of the extremities, as a series of urticarial plaques or with vesicle or bullae formation.

SJS is a more severe form of EM, with involvement of mucosal surfaces and viscera in association with marked constitutional symptoms. There is a prodrome lasting up to 14 days consisting of fever, malaise, myalgia, arthralgia, respiratory and gastrointestinal symptoms. It is followed by the explosive eruption of bullous lesions of the mouth, lips and conjunctivae, and variable skin involvement. In severe cases the oesophagus and respiratory tract are involved. Pneumonitis, pleural effusions and bullae of the visceral pleura are seen, the latter occasionally leading to pneumothorax or even bronchopleural fistula. Myocarditis, atrial fibrillation and renal failure are complications. There may also be anaemia and fluid and electrolyte imbalance. Treatment in SJS is supportive, although steroids have also been used for SJS and in severe cases of EM.

PREOPERATIVE ASSESSMENT

HISTORY AND EXAMINATION

- Fully assess the disease together with its extent, duration, distribution and severity of lesions, especially those involving the airway.
- Assess nutritional state.
- Note drug therapy and dose, especially systemic steroids and immunosuppressants.
- Consider cholinesterase activity following plasmapheresis.

INVESTIGATIONS

- Full blood count:
  - Anaemia may occur in SJS.
  - Leucopaenia and thrombocytopaenia may result from immunosuppressant therapy.
- Urea and electrolytes:
  - Abnormalities may exist in SJS or as a result of steroid therapy.
- Assess renal function and fluid balance, particularly in SJS. Dehydration is common, especially in more severe presentations.
- Blood glucose: may be raised due to steroid therapy.
- Flow-volume loops and radiological imaging of the airway may be of use where upper respiratory tract stenosis is suspected.
- Obtain urinary porphyrin estimations in undiagnosed bullous disease.

PERIOPERATIVE MANAGEMENT

PREMEDICATION

- In children, ensure that premedication is sufficient to prevent struggling during induction, which risks new bulla formation.
- The intramuscular route may be used as new bullae formation has not been reported.
- Consider pretreatment with topical steroids of areas of skin likely to be contacted during airway manipulation.
- If using EMLA cream, do not apply self-adhesive occlusive dressing.

INDUCTION

- Suture intravenous cannulae in place.
- Avoid barbiturates, particularly in undiagnosed bullous skin disease where the possibility of porphyria should be considered.
- Preoperative steroid supplementation should be commenced.
- Intramuscular ketamine has been used successfully.

AIRWAY CONSIDERATIONS

- A major concern is the potential to cause new bullae during airway manipulation or intubation. Avoid unnecessary airway manipulation. Reduce the risk of iatrogenic skin trauma:
    - Use face-masks with a soft air cushion.
    - Pad face-masks with Vaseline gauze.
    - Place a dermal pad or Vaseline gauze under the chin to protect skin from anaesthetist's fingers.
- Endotracheal intubation is preferable to laryngeal mask use where there are oral lesions to prevent aspiration should mucosal bleeding occur.
- Use a lubricated laryngoscope blade.
- If possible, avoid the use of airway adjuncts, such as Guedel airways.
- An inhalational induction may be required where laryngeal stenosis is present; helium/oxygen mixtures may help.

- Consider the use of a hood for inhalational induction.
- Protect front of neck with Vaseline gauze if cricoid pressure must be employed.
- Laryngoscopy and oral intubation may be difficult due to preexisting airway bullae.
- Laryngeal stenosis has been reported in SJS and cicatricial pemphigoid. The use of small uncuffed tracheal tubes may be necessary.
- The use of nasotracheal tubes has not been reported.
- Secure tracheal tubes with simple loose ties rather than with adhesive tape.
- Pay meticulous attention to airway cuff pressures.

MAINTENANCE

- IPPV may be hazardous in SJS because of the risk of pneumothoraces or bronchopleural fistulae.
- Avoid nitrous oxide due to possible pulmonary involvement.
- Drug distribution will be affected by hypoalbuminaemia and renal disease in SJS.
- Intravenous ketamine and benzodiazepine infusions, in the absence of intubation, have proven useful in SJS.

REGIONAL TECHNIQUES

- Spinal and epidural techniques appear safe. No new lesions have occurred at the sites of spinal needle insertion.
- Neuraxial opioids risk pruritus and consequent new eruptions. This risk is highest with morphine/diamorphine: fentanyl may be a safer choice.
- Modifications should be made to the skin cleansing routine:
    - Apply liquid skin cleanser.
    - Do not use applicator sponges.
    - Avoid rubbing the skin.
- Avoid subcutaneous injections of local anaesthetic solutions to prevent sloughing and bullae formation at the injection site.
- Avoid self-adhesive dressings.

MONITORING

- Well-padded noninvasive BP cuffs do not appear to cause bullae, probably because direct pressure is less harmful than frictional or shearing forces.
- The pulse oximeter should be attached using a simple clip rather than adhesive or tape.
- Use ECG pads without adhesive; needle ECG electrodes may be an alternative.
- Secure all lines with sutures, not tape.
- Do not use self-adhesive electrodes for monitoring neuromuscular function.
- Avoid indwelling temperature probes and cardiac output monitors where possible.
- Glucose monitoring in those receiving steroids.

POSITIONING

- Allow patients to move themselves onto trolleys or the operating table in order to minimise skin trauma.
- Keep sheets and other linen free from creases.
- Pad heels, elbows and bony prominences using foam or gel pads.
- Take care with the positioning of a diathermy pad and do not use a self-adhesive type.
- Avoid taping eyes: use protective ointment.

EMERGENCE AND RECOVERY

- If pharyngeal suction is required, use a soft flexible catheter.
- Beware of post-intubation laryngeal oedema.
- Use protective dermal pads or Vaseline gauze under oxygen masks.
- For children, encourage the early presence of parents in the recovery room in order to reduce struggling and restlessness.

POSTOPERATIVE MANAGEMENT

- New skin lesions are a common complication. Early dermatology involvement in the postoperative period is advisable.
- Continue steroid supplements.
- Aggressively treat any postoperative pruritus to prevent new lesions and secondary bacterial infection.

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CROSS-REFERENCE

Difficult airway – management, Chapter 26

DISORDERS OF EPIDERMAL CELL KINETICS AND DIFFERENTIATION

PSORIASIS

Psoriasis is a chronic skin disorder, characterised by an accelerated epidermal turnover and epidermal hyperplasia. These are caused by increased rate of epidermal protein synthesis, rapid epidermal cell growth, shortened epidermal cell cycle and an increase in the proliferative cell population. The exact aetiology is unknown, although both genetic and environmental factors are thought to play a part. Lesions involve the extensor surfaces (elbows, knees), lumbar-sacral

area and scalp. They consist of sharply demarcated, loosely adherent, thickened, non-coherent, silver skin scales which have an increased vascularity. Lesions often follow skin trauma (Koebner's phenomenon). Psoriatic arthropathy occurs in 5%–10% of patients and resembles seronegative rheumatoid arthritis. Psoriasis is also associated with ulcerative colitis and Crohn's disease.

Psoriatic lesions become colonised by bacteria, especially *Staphylococcus aureus*. Severe psoriasis may be associated with hyperuricaemia, anaemia (chronic illness, folate or vitamin B_{12} deficiency), negative nitrogen balance, iron loss and hypoalbuminaemia.

Two forms of psoriasis produce marked systemic effects on the body: psoriatic erythroderma (see below) and generalised pustular psoriasis. The latter is rare and characterised by waves of sterile pustules over the skin of the trunk and extremities, together with fever up to 40°C lasting for several days and can be associated with facial and airway oedema. There may be associated weight loss, muscle weakness, congestive cardiac failure and hypocalcaemia.

The treatment involves topical steroids, vitamin D3 analogues, retinoids (vitamin A derivatives), coal tar or dithranol. For larger areas, ultraviolet light (with or without a psoralen), systemic retinoids, cytotoxic agents (e.g. methotrexate, azathioprine), cyclosporin A and latterly monoclonal antibodies have a role in therapy.

ERYTHRODERMA

Eyrthroderma (or exfoliative dermatitis) describes a generalized inflammatory disorder with widespread scaling and erythema of the skin. The skin is hot and oedematous, often obscuring primary lesions that present earlier in the disease process and give clues to the underlying pathology: psoriasis, eczema, drug reactions and the reticuloses are common causes. The disorder is associated with systemic effects, including cardiovascular thermoregulatory and metabolic disturbances. In the acute stages there may be marked hypothermia, pyrexia-related hypovolaemia and heart failure.

Normally, total skin blood flow is approximately 1 L/min at 37°C; however, in erythroderma, this may

Table 9.1 Common causes of erythroderma

Dermatitis
Psoriasis
Reiter's syndrome
Lichen planus
Systemic lupus erythematosus
Dermatomyositis
Leukaemia
Lymphoma
Graft-versus-host disease
Pemphigus
Pemphigoid
HIV infection
Fungal skin infections
Drug reactions

increase to 5 L/min, reaching as much as 10 L/min in the presence of pyrexia. As a result, high output cardiac failure is a risk and may be exacerbated by hypoalbuminaemia, hypercatabolism and iron- or folate-deficiency anaemia.

Acute respiratory distress syndrome and secondary infections can complicate erythroderma and worsen prognosis, with a mortality rate of up to 64%.

Treatment of erythroderma relies on treating the cause (Table 9.1).

ICHTHYOSIS

Ichthyosis describes a group of conditions that are characterized by the accumulation of large amounts of dry scales on the skin. It is a disorder of keratinization. The most common form is ichthyosis vulgaris, an autosomal dominant disease. Other common forms include X-linked ichthyosis, lamellar ichthyosis (autosomal recessive) and epidermolytic hyperkeratosis (autosomal dominant). Ichthyosis is also seen in association with neoplasia such as lymphoma, multiple myeloma and carcinomas of the lung and breast. Treatment is directed at increasing the water content of the skin (urea-containing creams), causing separation of the cells using keralytic agents (salicylic acid ointment) or by affecting epidermal metabolism (lactic acid ointment). Occasionally, methotrexate and the retinoids are used.

PREOPERATIVE ASSESSMENT

HISTORY AND EXAMINATION

- Fully assess the disease together with its extent, duration, distribution and severity of lesions.
- Assess nutritional state.
- Assess any cardiovascular dysfunction.
- Exclude hypovolaemia (due to increased transepidermal water loss or pyrexia) in erythroderma.
- Check core temperature in eythroderma and pustular psoriasis.
- Note drug therapy and dose, especially immunosuppressants and steroids.
- Meticulous assessment of the airway is needed where psoriasis is complicated by arthropathy: joint mobility may be limited and awake fibre-optic intubation may be required.

INVESTIGATIONS

- Full blood count.
  - Anaemia may be due to chronic illness or deficiencies of iron, vitamin B12 or folate.
  - Leucopaenia and thrombocytopaenia may result from immunosuppressant therapy.
- Urea and electrolytes.
  - Abnormalities in renal function and electrolyte balance may exist in erythroderma.
  - Immunosuppressants and methotrexate can result in impaired renal function.
  - Measure calcium levels if hypoalbuminaemia exists.
- Liver function tests.
  - Immunosuppressants and methotrexate can result in impaired liver function.
- ECG and chest X-ray.
  - If there is evidence of heart failure on ECG or chest radiograph, proceed to echocardiography.
- Cross-match blood in erythroderma.
  - Risk of blood loss.

PERIOPERATIVE MANAGEMENT

PREMEDICATION

- Commence perioperative steroid supplementation.
- Do not inject intramuscular premedication into areas of psoriasis.

INDUCTION

- Avoid nitrous oxide: it potentiates the cytotoxic effects of methotrexate.
- Avoid using psoriatic sites for intravenous access (risk of infection due to skin colonization).
- Suture intravenous cannulae in place. Adhesive tape is likely to denude skin (ichthyosis or psoriasis), cause bleeding (psoriasis) or cause new lesions (Koebner phenomenon in psoriasis).
- The hyperdynamic circulation in erythroderma may alter the speed of onset of intravenous and inhalational anaesthetic agents.
- Hypervolaemia, hypovolaemia, congestive cardiac failure, hypoalbuminaemia and a reduction in renal blood flow may affect the kinetics of drug distribution and excretion.

MAINTENANCE

- Hypervolaemia, hypovolaemia, congestive cardiac failure, hypoalbuminaemia and reduction in renal blood flow may affect drug kinetics.
- Increased skin blood flow and high cardiac output in erythroderma may lead to excessive bleeding during surgery.
- Use space blanket or warm air blankets with care in erythroderma because of the risk of hyperthermia. These patients have difficulty in regulating their body temperature, have an increased metabolic rate and may not be able to sweat.

REGIONAL TECHNIQUES

- Avoid using psoriatic sites for regional techniques.
- For skin-cleansing use swabs soaked in an aqueous solution of antiseptic or pour solution over skin in ichthyosis. Alcohol-based solutions may cause intense pain. Avoid skin scrubbing.
- Secure extradural catheters using bandages, as adhesive tape may cause skin loss.
- Consider the risk of postoperative pruritus with neuraxial opioids. The risk is highest with morphine/diamorphine and lowest with fentanyl.

MONITORING

- Attach ECG electrodes and pulse oximeter to unaffected skin areas.
- Consider central venous pressure or cardiac output monitoring in erythroderma where fluid replacement must be undertaken with care.
- Use ECG pads without adhesive in ichthyosis/erythroderma.
- Monitor core temperature in erythroderma.
- BP cuffs can cause the Koebner phenomenon: padding may reduce this risk.

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CROSS-REFERENCE

Preoperative assessment – specific medical conditions, Chapter 25

EHLERS–DANLOS SYNDROME (EDS)

Ehlers–Danlos syndrome (EDS) consists of a group of hereditary disorders of connective tissue characterised by hypermobile and unstable joints, extensible and weakened skin, and fragile internal organs and vascular endothelium. There are six major variants based on clinical characteristics, latterly described as nine subtypes (EDS types I to IX—Table 9.2).

In most forms the condition is inherited as an autosomal trait. Specific mutations of some of the 20 collagen genes have been described in different types of EDS. Variations in the specific types of collagen involved and their distribution in different tissues result in the diverse clinical manifestations. Type-IV (vascular) is the most severe form due to abnormalities in type-III collagen which result in marked weaknesses in blood vessels and the possibility of spontaneous vessel rupture.

Other features include cardiac conduction defects, mitral valve prolapse, pes planus, scoliosis, herniae, bladder diverticuli, pulmonary emphysema, spontaneous pneumothorax, periodontitis and loose teeth. Clotting is usually normal, but severe bruising, due to friable vessels, is common.

PREOPERATIVE ASSESSMENT

HISTORY AND EXAMINATION

- Determine the exact form of EDS and its severity.
- Note previous history of joint dislocations, abnormal bleeding or failed local anaesthetic use (e.g. dental procedures).
- Assess preexisting cardiovascular and arterial disease with emphasis on valvular abnormalities, aneurysms and arrhythmias.
- Assess jaw opening.
- Assess cervical spine mobility (may be increased) and stability.
- Check for loose teeth and periodontitis.
- Assess skin fragility: may preclude the use of adhesive tape and dressings.

Table 9.2 Variants of EDS and their clinical criteria for diagnosis

Variant	Type	Major criteria	Minor criteria
Classic	I & II	Skin hyperextensibility	Smooth velvety skin
		Widened atrophic scars	Molluscoid pseudotumours
		Joint hypermobility	Subcutaneous spheroids
			Complications of joint mobility
			Muscle hypotonia, motor delay
			Easy bruising
			Extensible and fragile tissues
			Surgical complications
			Positive family history
Hypermobility	III	Hyperextensible and/or smooth velvety skin	Recurring joint dislocations
		Generalised joint	Chronic joint/limb pain
		Hypermobility	Positive family history
Vascular	IV	This translucent skin	Acrogeria
		Arterial/intestinal/uterine fragility or rupture	Small joint hypermobility
			Tendon & muscle rupture
		Extensive bruising	Talipes equinovarus
		Characteristic facies	Early onset varicose veins
			A-V, carotid-cavernous sinus fistula
			Pneumothorax
			Pneumohaemothorax
			Gingival recessions
			Positive family history
			Sudden death of a close relative
Kyphoscoliotic	VI	Joint hypermobility	Skin hyperextensibility
		Recurrent subluxations	Tissue fragility and atrophic scars
		Congenital bilatera hip dislocations	Easy bruising
			Hypotonia
			Kyphoscoliosis
			Osteopenia/osteoporosis
Arthrochalasis	VIIA	Joint hypermobility	Sin hyperextensibility
	VIIB	Recurrent subluxations	Tissue fragility and atrophic scars
		Congenital bilateral hip dislocations	Easy bruising
			Hypotonia
			Kyphoscoliosis
			Osteopenia/osteoporosis
Dermatosparaxis	VIIC	Severe skin fragility	Soft doughy skin texture
		Sagging redundant skin	Easy bruising
			Premature rupture of foetal membranes
			Large hernias

INVESTIGATIONS

- Coagulation studies (correct any abnormalities).
- ECG (arrhythmias and conduction defects).
- Echocardiography (valvular function and assess aortic root).

PERIOPERATIVE MANAGEMENT

- For those with high risk of bleeding (vascular subtypes, positive bleeding history or major surgery), blood conservation strategies are advised in particular intraoperative cell salvage.
- Cross-match blood (large volumes may be required).

PREMEDICATION

- Warn the patient about the risks of anaesthesia.
- Avoid intramuscular premedication (risk of haematoma formation).
- Bacterial endocarditis prophylaxis if mitral valve disease is present.
- Consider cardiac pacing in the presence of conduction defects.

MONITORING

- A blood pressure cuff may produce bruising and haematoma formation. Invasive blood pressure monitoring runs the risk of vascular wall dissection, particularly in the subtypes of EDS with vascular fragility.
- Where invasive pressure monitoring is essential, perform Allen's test and use ultrasound guidance to minimise vessel trauma.

INDUCTION

- Cannulation of vessels may be difficult:
  - Lax mobile skin makes vessel fixation difficult.
  - Loss of the normal sensation when a vessel wall is pierced is common.
  - Vessels may be fragile.
  - Risk of haematoma formation with all vessel punctures, especially arteries and central veins (avoid puncture of a vessel where direct pressure cannot be easily used to control bleeding, e.g. subclavian).
- Securing cannulae may be difficult due to mobile skin.
- Subcutaneous extravasation may undetected due to skin laxity.

MAINTENANCE

- Care with insertion of nasogastric tubes (risk of haemorrhage).
- Avoid systemic hypertension (risk of haemorrhage and aneurysmal rupture).
- Treat intraoperative haemorrhage aggressively with blood products as guided by point-of-care coagulation tests. The use of desmopressin (DDAVP) has been shown to improve the bleeding time and reduce transfusion requirements.
- Spontaneous pneumothorax should be considered in the event of cardiorespiratory instability. Avoid nitrous oxide.

POSITIONING

- Lax and fragile skin demands that extra care be taken when moving and positioning. Avoid shearing forces.
- The movement and positioning of joints should also prompt vigilance: brachial plexus damage can result from hyperextension.
- Avoid tourniquets: haematoma formation and compartment syndrome.

AIRWAY

- Take care during mask ventilation to ensure the temporomandibular joint is not dislocated.
- Take special care during intubation due to the risk of cervical spine damage. Where there is preexisting C-spine instability, awake fiber-optic intubation may be the safest approach.
- Mucosal haemorrhage can be problematic with repeated attempts at intubation.
- Modify the hypertensive response to laryngoscopy to reduce the risk of aneurysm rupture.

- Carefully monitor cuff pressures to minimise damage to tracheal mucosa.
- Limit airway pressures due to the risk of pneumothorax.

REGIONAL TECHNIQUES

- Regional techniques are said to be inadvisable in patients with easy bruising because of risk of haematoma formation. However, spinals and epidurals (caudal and lumbar) have been employed without complication.

OTHER CONSIDERATIONS

- Pay meticulous attention to haemostasis.
- Risk of major blood loss is significant.
- Ligaments and skin may not hold sutures (risk of wound dehiscence).

POSTOPERATIVE CARE

- Ensure good PONV prophylaxis as spontaneous eosophageal rupture has been reported as a result of vomiting in vascular EDS.
- Monitor for signs of bleeding and cardiovascular instability.

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CROSS-REFERENCE

EPIDERMOLYSIS BULLOSA (EB)

Epidermolysis bullosa (EB) encompasses a group of rare hereditary diseases characterised by blistering of skin, either spontaneously or following minimal mechanical trauma. Direct pressure to the skin is less likely to cause damage than frictional or shearing forces. Separation of the outer layer of the epidermis with accumulation of fluid within the space forms a large blister. Mucous membranes, particularly the mouth, pharynx and oesophagus, may also be affected. Over 20 different subtypes of EB exist, but are classified into four major groups depending on the plane within the skin where separation occurs:

- *Dystrophic (EBD)* – Split below the lamina densa
- *Junctional (EBJ)* – Split within the lamina lucida
- *Simplex (EBS)* – Intra-epidermal split
- *Kindler (EBK)* – Variable level of split formation

Treatment may involve systemic corticosteroids or drugs with collagenase activity, e.g. phenytoin and monocycline. Patients with EB often require repeated surgery for repair of syndactyly, oesophageal dilatation, skin grafting, dental surgery, removal of skin cancer or change of dressings. Infections of bullae are common.

DYSTROPHIC EB

Dystrophic EB may be transmitted as either an autosomal dominant or recessive disorder with an incidence of 1 in 50,000 to 1 in 300,000. It is characterised by low amounts of type-VII collagen, possibly due to excessive collagenase activity. Blister formation occurs beneath the lamina densa of the epidermal basement membrane. The disease may start at birth or in early infancy, and is characterised by extensive skin bullae with subsequent scarring. The bullae are large, flaccid and may become infected or haemorrhagic. In the hand, scar formation eventually results in sydactyly. Flexural contractures also occur. Involvement of the mucous membranes of the mouth, pharynx and oesophagus may lead to feeding difficulties, microstomia, fixation of the tongue to the floor of the mouth, and oesophageal stricture. Anaemia is common and poor nutrition may lead to growth retardation.

JUNCTIONAL EB

Junctional EB describes a group of autosomal recessive conditions leading to blister formation within the lamina lucida. Half of patients die in the first 2 years of life. Patients who survive infancy develop many of the complications of EBD. Enamel hypoplasia gives the teeth a characteristic pitting appearance.

SIMPLEX EB

Some forms are inherited as autosomal dominant disorders, others as autosomal recessives. EBS may be generalized or localized and is the least disabling form.

KINDLER SYNDROME

Characterised by generalised blistering from birth, it was previously classed as a poikilodermatous photosensitivity disorder. Immunofluorescence antigen mapping has now recognised the disease as a variant of EB.

PREOPERATIVE ASSESSMENT

- Fully assess the form of EB, together with the extent and duration.
- Note drug therapy, especially systemic corticosteroids and phenytoin.
- Assess venous access.
- Carefully assess the airway.
- Establish any dysphagia and oesophageal stenosis (which may impact on postoperative analgesia planning) and any reflux (which may impact on conduct of anaesthesia).

INVESTIGATIONS

- *Full blood count* – Anaemia and thrombocytosis are common. Some severe forms of EB can result in a transfusion dependent anaemia.
- *Urea and electrolytes* – Abnormalities exist in severe EB; assess renal function.
- Serum iron, folate and vitamin B_{12} levels.
- *Liver function tests* – Especially if receiving phenytoin/minocycline.
- *Albumin* – Hypoalbuminaemia is common.
- Phenytoin levels.

PERIOPERATIVE MANAGEMENT

PREMEDICATION

- Keep the patient calm during induction (struggling may risk new EB lesions).
- If using EMLA cream do not apply self-adhesive occlusive dressing.
- Avoid intramuscular injections for fear of inducing new EB lesions; however, it seems unlikely that intramuscular injections cause such problems.
- Prophylaxis against gastric aspiration if oesophageal complications exist.

INDUCTION

- Venous access may be difficult and central venous cannulation may be necessary. Suture cannulae in place: do not use adhesive tape.
- An inhalation induction may be needed if venous access is difficult.
- Intramuscular ketamine has proven useful.
- Suxamethonium appears to be safe (no new EB lesions have been reported after muscle fasciculations; hyperkalaemia response is not seen, despite muscle atrophy).
- Thiopental appears safe, despite fears of associated porphyria.
- For children, encourage the presence of parents in the anaesthetic room to reduce the chances of struggling causing new lesions.

AIRWAY CONSIDERATIONS

- Laryngoscopy and oral intubation may be difficult due to poor dentition, limited mouth opening and adhesion of tongue to floor of mouth. Consider fibre-optic intubation and have adjuncts including video-laryngoscopes immediately available.

- Concern has been expressed over the possibility of causing new EB lesions during airway manipulation or intubation. Although pharyngeal lesions do occur, the hazards of both intubation and laryngeal mask used appear to have been overstated, since there are no reports of laryngeal or tracheal lesions following tracheal intubation or tracheostomy. However, spontaneous EB lesions are possible in all of these sites.
- Use a face-mask with a soft air cushion and pad with Vaseline gauze.
- Place protective dermal pads or Vaseline gauze under chin to protect skin from anaesthetist's fingers.
- If possible, avoid the use of airway adjuncts, e.g. Guedel airways.
- Use a smaller endotracheal tube or laryngeal mask than the size predicted by standard formulae.
- Rapid sequence induction when there are oesophageal symptoms.
- Protect front of neck with Vaseline gauze when applying cricoid pressure.
- New head and neck lesions are often associated with difficult or failed intubation.
- Avoid nasal airways or tubes or lubricate well and use with great care.
- Lubricate the laryngoscope blade well.
- Use small uncuffed tracheal tubes if possible, always taking into account the surgical field and the need to prevent airway soiling.
- Secure tracheal tubes with simple loose ties and not adhesive tape.
- Treat haemorrhage from mucous membrane lesions using sponges soaked in epinephrine (1:200,000).
- Avoid nasogastric tube if possible.

MAINTENANCE

- Drug disposition will be affected by decreased muscle bulk, hypoalbuminaemia and any renal disease.

REGIONAL TECHNIQUES

- Although regional anaesthesia has traditionally been avoided, many blocks have been used without complication (e.g. brachial plexus, spinal, epidural [lumbar and caudal], femoral nerve, digital, wrist and lateral cutaneous nerve of thigh).
- Neuraxial opioids risk pruritus and consequent new eruptions. This risk is highest with morphine/diamorphine: fentanyl may be a safer choice.
- Modifications should be made to the skin cleansing routine:
    - Apply liquid skin cleanser.
    - Do not use applicator sponges.
    - Avoid rubbing the skin.
- Avoid subcutaneous injections of local anaesthetic solutions to prevent bullae formation at the injection site.
- Avoid self-adhesive dressings.

MONITORING

- Well-padded BP cuffs do not appear to cause bullae, probably because direct pressure is less harmful than frictional or shearing forces.
- Use ECG pads without adhesive; needle ECG electrodes may be an alternative.
- Secure arterial lines with sutures not tape.
- Glucose monitoring in those receiving steroids.

POSITIONING

- Protect the eyes using non-petroleum-based ointment and avoid adhesive tape (bullae may lead to corneal ulceration and globe perforation, thick lubricating ointment may cause visual disturbance and rubbing during emergence).
- Inform operating-room staff of the need for special care during patient positioning.
- Allow patients to move themselves onto trolleys or the operating table.

- Keep sheets and other linen free from creases.
- Pad heels, elbows and bony prominences using foam or gel pads.
- Take care with the positioning of a diathermy pad and do not use a self-adhesive type.
- Tourniquets do not appear to be hazardous if the skin is protected by sufficient padding.

EMERGENCE AND RECOVERY

- Pharyngeal suction must be performed extremely carefully using a soft flexible catheter.
- Use protective dermal pads or Vaseline gauze under oxygen masks.

POSTOPERATIVE CARE

Common problems or complications include new skin lesions, and regurgitation leading to aspiration pneumonitis.

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CROSS-REFERENCES

GLYCOGEN STORAGE DISEASE

Glycogen consists of chains of 6–12 glucose units joined at carbon atoms 1 and 4. The chains are joined by 1:6 linkages to form branching structures. The principal storage sites are liver and muscle. Liver glycogen is concerned with maintenance of blood glucose; muscle glycogen comprises the energy store for muscle itself and does not contribute to blood glucose homeostasis. Enzymes synthesise and break glycogen down to its constituent glucose units (liver glycogen) or to pyruvate and lactate in muscle. Muscle glycogen is principally used in anaerobic metabolism via the relatively inefficient glycolytic pathway and in situations where sudden bursts of activity are required and there is no time for increases in cardiac output and delivery of oxygen. Lactate diffuses into the circulation and may then be resynthesised to glucose by the liver. Adrenergic stimulation also causes muscle and blood lactate to rise in shocked states.

Glycogen storage diseases (GSD) and their associated enzyme deficiencies, with a few exceptions, show an autosomal recessive inheritance. Diagnosis is by liver or muscle biopsy and genetic/enzyme studies.

GSD TYPE I: VON GIERKE DISEASE

- A deficiency of glucose 6-phosphatase (or a phosphatase translocase/transporter for one subtype) in the liver, kidney and intestine. This enzyme hydrolyses glucose 6-phosphate to glucose and phosphate resulting in an inability to mobilise liver glycogen to maintain blood glucose.
- Presents as short stature; prominent rounded abdomen due to liver enlargement with fat deposits in cheeks and buttocks. The kidneys also enlarge due to storage of glycogen which can cause Fanconi syndrome.
- Patients develop hypoglycaemia, hyperlipidaemia, a tendency to acidosis (both ketoacidosis and lactic acidosis) and attacks of hyperuricaemia/gout.
- There is also a prolonged bleeding time due to platelet glucose-6-phosphatase deficiency.
- Frequent or nasogastric feeding is needed to maintain blood glucose. Avoid carbohydrates which must be converted to glucose 6-phosphate (e.g. fructose).

- A portacaval shunt enables absorbed glucose to bypass the liver and has been used as treatment; however, whilst growth often improves, the metabolic abnormalities remain unresolved. This dichotomy is also true of liver transplantation, another therapeutic approach that has been attempted in cases where metabolic control is poor or hepatocellular carcinoma develops. With liver transplantation, patients are also at risk of complications from the liver transplant itself (such as malignancy transformation) and immunosuppression.

GSD TYPE II: POMPE DISEASE

- Alpha-1-4-glucosidase (acid maltase) deficiency in the liver, muscle and cardiac tissue.
- This lysosomal enzyme normally breaks the 1–4 linkage in glucose chains resulting in excessive quantities of glycogen in the liver, heart, muscle, tongue and central nervous system (especially anterior horn cells).
- Whilst blood sugar, lipid and ketone concentrations and response to glucagon and adrenaline are normal, the prognosis is poor due to cardiac disease from hypertrophic cardiomyopathy with left ventricular outflow tract obstruction.

GSD TYPE III: CORI DISEASE

- A deficiency of amylo 1,6-glucosidase (debranching) enzyme in the liver and muscles.
- Clinical features are similar to GSD Type I but milder; patients are able to mobilise glucose from outer chains of the glycogen molecule.
- Liver enlargement, growth retardation, hypoglycaemia, elevated blood lipid concentrations and muscle fatigue are common clinical features. Hypoglycaemic brain injury can occur prior to diagnosis leaving long-term disability.
- Treatment consists of frequent feeds with a high protein diet as the gluconeogenic pathway is intact.

GSD TYPE IV: ANDERSEN DISEASE

- A deficiency of amylo-1,4 to 1,6-transgluosidase (branching) enzyme in the liver.
- Patients are normal at birth, but fail to thrive.
- Subsequent hepatosplenomegaly, muscle hypotonia and rapidly progressive liver cirrhosis develops.
- Problems are those of severe liver disease and few sufferers live beyond 5 years.

GSD TYPE V: MCARDLE DISEASE

- Lack of muscle phosphorylase which normally removes glucose units from glycogen as glucose 1-phosphate for metabolism to pyruvate and lactate; therefore, unable to maintain glucose supply.
- Three distinct subtypes:
  - Rapidly fatal neonatal form.
  - Mild form with congenital myopathy.
  - Benign classic with myalgia and dark urine.
- The latter two subtypes have a good prognosis and usually present in the second decade of life with fatigue and muscle cramps.

GSD TYPE VI: HERS DISEASE

- Reduced hepatic glycogen phosphorylase E.
- Clinically mild form of GSD Type I: hepatomegaly and mild hypoglycaemia.

GSD TYPE VII: TARUI DISEASE

- Phosphofructokinase deficiency in muscle.
- Clinically similar to GSD Type V though can also affect the liver, platelets and fibroblasts.

GSD TYPE VIII/IX

- Disorder of the phosphorylation activation system in the liver.
- Clinically similar to GSD Type III but without muscle involvement.
- There is an X-linked variant as well as an autosomal recessive inherited phenotype.
- Other types are extremely rare.

PREOPERATIVE ASSESSMENT

Specifically enquire about:

- Bleeding tendencies, epistaxis: can indicate platelet dysfunction (particularly in GSD Type I due to platelet glucose-6-phosphatase deficiency).
- Obstructive respiratory symptoms: an enlarged heart causing bronchial obstruction.

EXAMINATION

- Hepatomegaly or hepatosplenomegaly may be present. This will require particular care if the prone position is required.
- Evidence of active respiratory disease should lead to postponement of elective surgery.

INVESTIGATIONS

- *Haematology profile* – Check platelet count and function in GSD Type I.
- *Urea and electrolytes including calcium* – Renal dysfunction in GSD Type I and hepatorenal syndrome in most types.
- *Liver function and coagulation profile* – Particularly in GSD Type V where there is early hepatic cirrhosis.
- *Spirometry and chest X-ray* – Reduced functional residual capacity, atelectasis and a propensity to chest infections.
- *ECG and echocardiogram* – Look for ischaemic changes, cardiomyopathy and associated left ventricular outflow tract obstruction which can be severe.

PERIOPERATIVE MANAGEMENT

A multi-professional approach involving special-ists in metabolic medicine is crucial in preventing preoperative morbidity and mortality and avoiding complications.

- Minimise fasting times. Consider continuous enteral glucose via a nasogastric tube to up the commencement of the fasting period.

- Parenteral glucose is required during the fasting period and is continued intra-operatively: 0.4 g/kg/h for a child, 0.5 g/kg/h for an infant and 0.6 g/kg/h for a neonate (4, 5 and 6 mL/kg/hr of 10% dextrose, respectively).
- Monitor blood glucose and lactate levels throughout the fasting period.
- Platelet transfusion may be required despite a normal count where there is prolonged bleeding time or platelet dysfunction.
- Administration of DDAVP may also be useful to normalise platelet function.

MONITORING

- Regular blood glucose, lactate and acid-base measurements are required: an arterial line is recommended. This also prevents the theoretical risk of muscle damage due to frequent cuff inflation during longer surgeries.
- Continuous glucose monitoring device may be appropriate.
- Point of care tests of ketosis, particularly in the subtypes affecting the liver.
- Monitor temperature (risk of hyperthermia). Hypothermia and shivering can cause muscle damage in some types of GSD.
- Consider cardiac output monitoring or transoesophageal echocardiography if there is preoperative evidence of cardiac dysfunction.
- A urinary catheter is advisable: oliguria can herald rapidly developing rhabdomyolysis and prompt early intervention for hyperkalaemia and consequent arrhythmias.

INDUCTION AND AIRWAY

- Macroglossia is reported in Type II GSD and may make laryngoscopy difficult.
- Maintain coronary perfusion pressure and avoid myocardial depressants where possible.
- Avoid suxamethonium where possible due to the theoretical risk of myoglobin release, hyperthermia and hyperkalaemia.
- Avoid drugs with predominantly renal metabolism (e.g. neuromuscular blockers) Type I GSD.

POSITIONING

- Avoid tourniquets where possible, particularly in GSD Types II, V and VII.

MAINTENANCE

- Avoid lactate-containing crystalloids.
- Bicarbonate may be required to correct intraoperative acidosis.

ANALGESIA

- *Avoid NSAIDs* – Effect on platelet function.

EMERGENCE AND RECOVERY

- Continue pH, lactate and glucose measurement until 4 hours after oral feeding has been reestablished.
- Have a low threshold for suspecting hypoglycaemia if seizures occur.
- Impaired recovery of neurological function should prompt serum ammonia testing: case reports of hyperammonaemia with GSDs exist.
- Dark urine should warn for the potential of myoglobinuria which can occur within a few hours of muscle injury.

REGIONAL ANAESTHESIA

- Case reports exist of neuraxial techniques being used without complications. Avoid shivering and maintain coronary perfusion pressure.

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INFLAMMATORY MYOPATHIES

This includes a group of four chronic conditions characterised by the subacute onset of proximal weakness. While the exact mechanism is unclear, there is strong evidence to suggest autoimmune pathogenesis.

DERMATOMYOSITIS

The weakness is accompanied by characteristic violaceous skin lesions, often involving the upper eyelid. It can present at any age and is associated with other multisystem connective tissue diseases such as systemic lupus erythematosus, rheumatoid arthritis, Hashimoto's thyroiditis and systemic sclerosis. There is an increased risk of cancer during the first 3–5 years following diagnosis.

NECROTISING AUTOIMMUNE MYOSITIS

Predominantly a disease of adults, it can have an acute (over days) or subacute (over weeks) clinical course. The degree of inflammation, muscle damage and therefore weakness is severe. It can occur in isolation, following viral infections or as a result of statin therapy. There is an association with cancer and other connective tissue disorders such as scleroderma.

INCLUSION-BODY MYOSITIS (IBM)

IBM has a more insidious onset and less symmetrical pattern of symptoms than the other inflammatory myopathies. There is earlier involvement of distal muscle groups causing a different symptom pattern. Dysphagia is common – over half of patients are affected. It is a complex disorder with autoimmune and degenerative components, similar to the molecular biochemistry seen in Alzheimer disease. Proteinaceous deposits induce an inflammatory and

degenerative process within the myocyte. Unlike other inflammatory myopathies, all steroid and immuno-suppressive therapies are ineffective in IBM.

POLYMYOSITIS

A diagnosis is made once other causes of subacute proximal weakness have been excluded. Apart from the other inflammatory myopathies, the important diagnoses to exclude are

- Fasciitis
- Drug-induced myopathies (e.g. statins, antiretrovirals)
- Inflammatory dystrophies (e.g. Becker's muscular dystrophy)
- Metabolic myopathies
- Fibromyalgia

MANAGEMENT

A combination of clinical history, creatine kinase, electromyography, muscle biopsy, autoantibody measurement and magnetic resonance imaging is used to differentiate between these conditions. Treatment is usually with oral steroids or, in difficult cases, immunosuppressive drugs, e.g. azathioprine, cyclophosphamide, methotrexate or ciclosporin. Immunoglobulin and monoclonal antibodies can be used when these therapies have failed. IBM is classically resistant to therapy, though immunoglobulin can improve dysphagia.

MUSCLE

Chronic inflammation leads to proximal weakness, muscle tenderness, myalgia and, eventually, atrophy and fibrosis of skeletal muscle. Distal muscle involvement is a late sign apart from inclusion-body myositis where it can be an early presenting symptom. Characteristically there is a rise in serum enzymes derived from muscle, e.g. creatine phosphokinase (CPK), and their levels usually parallel disease activity. Myoglobin may be released leading to myoglobinaemia and myoglobinuria. The risk of malignant hyperthermia is unknown, but recent *in vitro* studies have shown that muscle from some patients produces

significant contracture to caffeine or halothane, but not both (i.e. malignant hyperthermia equivocal).

RESPIRATORY

Approximately 10%–40% of patients have associated interstitial pulmonary disease, predominantly fibrosis. Pulmonary compromise can also occur because of intercostal and diaphragmatic weakness or aspiration pneumonia. Vocal cord dysfunction may exist.

CARDIAC

Cardiac disease is a major cause of death. The principal manifestations are arrhythmias, conduction defects, myocarditis, cardiomyopathy and cor pulmonale. Steroid-induced hypertension may occur.

GASTROINTESTINAL

Poor coordination of swallowing, nasal regurgitation and pooling of secretions in the vallecula and cricopharyngeal spaces may predispose to pulmonary aspiration. Oesophageal reflux, delayed gastric emptying and decreased intestinal motility also occur.

PREOPERATIVE ASSESSMENT

- Review of associated cardiovascular, respiratory and gastrointestinal disease and assessment for other associated autoimmune disorders.
- Review of drug therapy and its possible complications.
- Treat any chest infection.
- ECG (tachyarrhythmias, conduction abnormalities, ventricular hypertrophy, cor pulmonale).
- Chest X-ray (pulmonary fibrosis, ventricular dilatation).
- Lung function tests (decreased vital capacity, FRC and tidal volume despite normal chest X-ray. Patients who have no pulmonary symptoms may exhibit abnormal pulmonary function tests).
- High resolution computerised tomography (interstitial lung disease).
- Full blood count (anaemia, leucopaenia, thrombocytopaenia).

- Urea and electrolytes (establish baseline renal function).
- Creatine phosphokinase (establish baseline level).
- Urinary myoglobin (elevated levels may prompt more judicious use of intravenous fluid).

PREMEDICATION

- Continue systemic steroid cover.
- Avoid intramuscular injections.

PERIOPERATIVE MANAGEMENT

- Avoid malignant hyperthermia trigger agents.
- Controlled ventilation should be used in the presence of preoperative ventilatory compromise.
- It is wise to avoid suxamethonium because of the potential risk of malignant hyperthermia and hyperkalaemia.
- Nondepolarising neuromuscular blocking agents appear to be safe although reports exist of prolonged paralysis following vecuronium in polymyositis and atracurium in eosinophilic myositis. A myasthenic-like response has been reported, but this may be related to associated malignancy.
- Use small doses of short-acting relaxants preferably after a test dose.

AIRWAY

- The use of a tracheal tube is strongly recommended, especially if there is risk of aspiration.
- Rapid sequence induction should be used if pharyngeal, oesophageal or gastric symptoms are present.

MONITORING

- Routine AAGBI monitoring.
- Neuromuscular monitoring.
- Glucose monitoring in those receiving steroids.
- Urinary catheter: adequate urine output should be maintained where there is preoperative myoglobinuria.

EMERGENCE AND RECOVERY

- Extubation and weaning should follow measurement of lung volumes and careful exclusion of residual neuromuscular block.
- Extubate awake or in the lateral position (risk of aspiration).
- Consider noninvasive ventilation in the immediate postoperative period.

POSTOPERATIVE CARE

- Continue steroid supplementation.
- Avoid intramuscular analgesics.
- Postoperative physiotherapy.
- The following complications might be expected from knowledge of the disease:
  - Prolonged recovery from neuromuscular blockade, requiring IPPV
  - Lung atelectasis
  - Postoperative pneumonia
  - Postoperative respiratory failure
  - Aspiration if pharyngeal muscles are weak
  - Arrhythmias
  - Cardiac failure

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MUCOPOLYSACCHARIDOSES

A group of rare familial disorders caused by deficiencies of lysosomal enzymes required to metabolise the mucopolysaccharide (MPS) constituents of connective tissue, now known as glycosaminoglycans. This causes accumulation of MPS in organ systems resulting in anatomical and biochemical dysfunction.

The disorder is divided into eight phenotypes based on defined enzymatic deficiencies. All have autosomal recessive inheritance with the exception of the X-linked MPS II (Hunter syndrome) (Table 9.3).

SYSTEMS AFFECTED

AIRWAY AND CERVICAL SPINE

Airway obstruction may occur due to macroglossia and adenotonsillar hypertrophy. There may be narrowing of the hypopharynx and trachea with laryngomalacia and tracheomalacia. Abnormalities of the cervical spine are common, including atlanto-axial instability and cervical cord compression.

RESPIRATORY

Obstructive and restrictive lung disease may coexist. Kyphoscoliosis and chest wall deformities result in a predisposition to lower respiratory tract infections. Patients are often at closing capacity during tidal breathing and are prone to sleep apnoea. Chronic right ventricular failure may further affect respiratory function.

CARDIAC

Valvular heart disease is common. Myocardial and coronary vessel involvement leads to hypertrophic cardiomyopathy, congestive cardiac failure, myocardial ischaemia and infarction. Arrhythmias and conduction block may occur. Pulmonary hypertension and cor pulmonale from chronic respiratory disease cause further compromise.

Table 9.3 Phenotypes and major anaesthetic considerations of the mucopolysaccharidoses

MPS type I

Hurler syndrome

    Multi-system chronic and progressive presentation

    Wide range of symptom severity

    Major anaesthetic considerations: macroglossia, kyphoscoliosis, odontoid hypoplasia, mitral incompetence, cardiomegaly

MPS type IS

Scheie syndrome (formally MPS type V)

    A less severe form of Hurler syndrome

    Characterised by a normal lifespan

    Major anaesthetic considerations: macroglossia, prognathia, short neck, aortic incompetence

MPS type I H-S

Hurler–Scheie syndrome

    Intermediate form of MPS type I, with a disease severity between that of Hurler and Scheie syndromes

    Major anaesthetic considerations: macroglossia, micrognathia, short neck, mitral and aortic incompetence

MPS type II

Hunter syndrome

    Predominant male (X-linked recessive)

    Similar to MPS type I but usually milder symptoms

    Major anaesthetic considerations: hydrocephaly, short neck, ischaemic cardiomyopathy

MPS type III

Sanfilippo syndrome

    Predominance of CNS involvement

    Major anaesthetic considerations: macroglossia, vertebral abnormalities

MPS type IV

Morquio syndrome

    Greater skeletal involvement manifesting in spinal deformities

    Major anaesthetic considerations: kyphoscoliosis, odontoid hypoplasia, short neck, C1/C2 instability, aortic regurgitation

(*Continued*)

Connective tissue

Table 9.3 (Continued) Phenotypes and major anaesthetic considerations of the mucopolysaccharidoses

MPS type VI
Maroteaux–Lamy syndrome
  Characterised by a period of normal development
  Facial features and hepatosplenomegaly predominate
  Major anaesthetic considerations: macroglossia, kyphoscoliosis, odontoid hypoplasia, mitral and aortic valve lesions

MPS type VII
Sly syndrome
  Spectrum of mild (developing symptoms aged 4 or older) to severe (presenting neonatally as hydrous fetalis)
  Major anaesthetic considerations: contractures, thoracic gibbus, odontoid hypoplasia, mitral and aortic valve lesions, aortic dissection

CENTRAL NERVOUS SYSTEM

Learning difficulties, developmental delay, hydrocephalus, raised intracranial pressure and visual and hearing impairment may all be seen.

GASTROINTESTINAL TRACT

Hepatosplenomegaly is common but liver and splenic functions are usually unaffected. Umbilical and inguinal herniae are common.

PREOPERATIVE ASSESSMENT

Seek information about previous operations and anaesthetic problems, physical and mental disability, respiratory or cardiovascular symptoms and symptoms of obstructive sleep apnoea.

Examination – pay particular attention to:

- Skeletal abnormalities (craniofacial, contractures, etc.)
- Skin and peripheral veins
- Upper airway and orofacial abnormalities
- Cervical spine abnormalities and kyphoscoliosis
- Proximal and distal airways obstruction
- Evidence of chronic lung disease

- Secretion and sputum production
- Active pulmonary infection
- Cardiac valve incompetence or stenosis
- Cardiomegaly, left ventricular hypertrophy
- Cor pulmonale
- Univentricular and biventricular failure
- Arrhythmias
- Raised intracranial pressure, papilloedema, visual and hearing impairment

INVESTIGATIONS

- *Spirometry* – Decreased vital capacity, FRC, TLC
- *Arterial blood gases* – Hypoxaemia, hypercapnia
- *Chest X-ray* – Pneumonia, atelectasis, subglottic narrowing
- *ECG* – LVH, RVH, arrhythmias, conduction block
- *Echocardiogram* – Decreased ejection fraction, valve lesions, dyskinesia
- *Cardiac catheterisation* – Valve pressure gradients, coronary artery occlusion
- *CT scan of brain* – Hydrocephaly, increased ICP
- *Cervical spine X-rays* – Decreased ossification, atlanto-occipital instability
- *MR scan of cervical spine* – Decreased ossification, atlanto-occipital instability
- *Haematology profile* – Increased white cell count, anaemia, polycythaemia
- *Urea and electrolytes* – Diuretic and digoxin effects
- *Liver function tests* – Liver dysfunction
- *Somatosensory evoked potentials* – Early cord compression
- *CSF pressure* – Communicating hydrocephalus

PREPARATION

- Treat any active respiratory infection.
- Chest physiotherapy.
- Optimise cardiac failure and arrhythmias.
- Full discussion with parents, and consideration of parental accompaniment to anaesthetic room.
- Difficult airway equipment prepared (ETT size not predictable from age).
- ENT backup in case of emergent tracheostomy.
- ICU availability.
- Topical anaesthesia for IV cannulation.

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- Benzodiazepine if premed needed (pulse oximetry after administration).
- Antisialogogue (glycopyrrolate).
- Topical anaesthesia where fibre-optic intubation likely including nasal vasoconstrictor.
- Consideration of antibiotic prophylaxis.

PERIOPERATIVE MANAGEMENT

In addition to standard anaesthesia monitoring, somatosensory evoked potentials should be monitored in patients with a high risk of compression of the cervical spinal cord (MPS types I, IV, VI and VII), particularly during airway manipulations or spinal surgery. Intra-arterial blood pressure measurement is recommended where there is cardiac involvement.

INDUCTION AND AIRWAY

- Difficult laryngoscopy and intubation is common and becomes progressively more so with increasing age.
- Oral access may be reduced due to anatomical distortion and temporomandibular stiffness.
- Fibre-optic intubation is often required either with the patient lightly sedated or through a laryngeal mask.
- The lateral position may be helpful for those with sleep apnoea.
- Take care to avoid neck extension during manipulation, particularly where there are C-spine risk factors.
- Nasal airway adjuncts are preferable: oral airways may cause obstruction by pushing the epiglottis backwards.
- Neutral head position to avoid neck overextension.
- Kyphoscoliosis may impair position.

MAINTENANCE

- Spontaneous breathing techniques are generally inadvisable unless the procedure is of short duration or tracheal intubation proves impossible.
- Caution with opioid analgesics because of increased sensitivity.
- Local or regional techniques are beneficial.

- On completion, treat as difficult extubation. Nasopharyngeal airways and airway exchange catheters are useful adjuncts as obstruction is common.
- Delayed emergence is a recognised problem.
- Postoperative monitoring in ICU should be considered.

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POLYARTERITIS NODOSA

Polyarteritis nodosa (PAN) is a rare systemic disease in which a necrotising arteritis affects the small- and medium-sized arteries. The consequence of this vasculitis depends on the site and number of vessels involved and may range from localised lesions with clinically insignificant effects to life-threatening organ failure. Typically, there is aneurysm formation in the medium-sized arteries, and both haemorrhage and infarction in major organs. Prognosis in PAN is significantly influenced by the size of vessel affected and by the presence of renal involvement: most patients die as a result of renal failure. While the prognosis has improved in recent years, the 5-year mortality for PAN is up to 46% for those with severe disease.

The histology is a fibrinoid necrosis of the media of affected arteries associated with infiltration of the intima and media by polymorphs. It occurs more often in women, with a peak incidence in the 40–50 year age group and can be related to malignancies and allergic drug reactions. A subset of PAN cases are associated with acute hepatitis B virus (HBV) infection.

Cutaneous PAN is a variant with restricted involvement of the skin, exclusively involving the limbs and generally restricted to below the knees.

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It is usually treated with immunosuppressant drugs (e.g. cyclophosphamide), systemic corticosteroids and management of any underlying disease, particularly in HBV-associated PAN. Plasmapheresis is occasionally used.

PREOPERATIVE ASSESSMENT

- Assess the extent and duration of the illness. Is there an acute arteritis or is it in remission? This will affect perioperative risk and outcome.
- Determine hepatitis B and C status.
- Cardiovascular symptoms: angina, heart failure, cardiomegaly, pericarditis.
- Respiratory symptoms: asthma, pneumonia, haemoptysis, pleurisy.
- Renal: renal failure, hypertension.
- CNS: peripheral neuropathy, CVA.
- Airway: acute pharyngeal oedema.
- Drug history: systemic corticosteroids, immunosuppressants, cardiac drugs.

INVESTIGATIONS

- *ECG* – Cardiac ischaemia, myocardial infarction, arrhythmias, pericarditis.
- *Echocardiography* – Assess cardiac contractility, pericardial effusions.
- *Chest X-ray* – Pulmonary infection, haemorrhage, pulmonary infiltrates (pulmonary eosinophilia), pulmonary fibrosis, cardiac failure.
- *Angiography* – Used to aid diagnosis and monitor disease progression.
- *Urea and electrolytes* – Usually normal until disease is very severe, assess renal function (creatinine and potassium).
- *Full blood count* – Anaemia is common; leucocytosis is a frequent finding, leucopaenia or pancytopaenia may result from immunosuppressant drug therapy.
- *ESR and CRP* – Endovascular procedures are ideally performed when inflammation is controlled.
- *Liver function tests* – Albumin and clotting factors as indicators of liver function, check hepatitis B and C status.

- *Plasma cholinesterase levels* – Reduced by plasmapheresis, inhibited by cyclophosphamide.
- *Nerve conduction studies* – To record preoperative neuropathies.

PREOPERATIVE PREPARATION

- Optimise cardiovascular and respiratory systems.
- Chest physiotherapy.
- Antibiotics.
- Control hypertension.
- Treat angina (e.g. nitrates).
- Patients with severe cardiovascular disease may require invasive haemodynamic monitoring.

PERIOPERATIVE MANAGEMENT

- Scrupulous anti-infection measures are essential because of the high risk of sepsis.
- Aneurysmal repairs are prone to large volume blood loss: ensure cross-matched blood is available and consider the use of intraoperative cell salvage.
- Perioperative steroid cover will be needed for the patient receiving systemic steroids.
- Anxiolytic premedication may be advantageous to reduce the risk of tachycardia and hypertension from catecholamines. Avoid anticholinergic premedication (risk of tachycardia).

INDUCTION

- Avoid drugs and techniques likely to cause tachycardia, hypertension, hypotension or reduced myocardial contractility.
- Use a cardiostable technique (e.g. high dose opioid) to limit the hypertensive response to laryngoscopy.
- Preoxygenate.
- Pharyngeal oedema, although rare, has been reported.

MAINTENANCE

- Avoid drugs and techniques likely to cause tachycardia, hypertension, hypotension or reduced myocardial contractility.

- Maintain good tissue blood and oxygen supply.
- Maintain fluid balance, especially in patients with renal involvement.
- Drug handling is altered in renal disease. Avoid drugs which are primarily excreted by the kidneys.
- Hypoalbuminaemia will affect drug distribution.
- Avoid vasoconstrictor drugs, if possible, because of the risk of vascular occlusion.
- When plasmapheresis has been recent, remember that the action of ester drugs (e.g. suxamethonium) will be prolonged.
- The action of suxamethonium may be prolonged by cyclophosphamide.

REGIONAL TECHNIQUES

- Avoid use of epinephrine-containing local anaesthetic solutions because of the risk of vascular occlusion.
- Document existing sensorimotor neuropathies prior to use of regional techniques.

MONITORING

- CM5 lead for ECG to detect myocardial ischaemia and arrhythmias.
- Radial artery cannulation may be inadvisable in the presence of aneurysmal disease because of the risk of vascular occlusion.
- Cardiac output monitoring or transoesophageal echocardiography may be helpful intraoperatively if there is severe cardiac disease.
- Glucose monitoring in those receiving steroids.

POSTOPERATIVE MANAGEMENT

- Monitor cardiovascular/renal function.
- Be vigilant for signs of systemic sepsis.
- Continue systemic steroid supplementation.
- Posterior reversible encephalopathy syndrome (PRES), a reversible syndrome of headache, altered mental functioning, seizures and loss of vision has been reported. Neuroimaging in these patients reveals extensive bilateral white-matter oedema predominantly in the posterior regions of the cerebral hemisphere.

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PSEUDOXANTHOMA ELASTICUM

Pseudoxanthoma elasticum (PXE) is a systemic disorder of elastic and collagen tissue in which there is progressive calcification and degeneration of elastin fibres and the supportive collagen matrix in skin and mucous membranes. Similar processes may affect the arterial system, leading to arterial occlusion and ischaemia. Ocular and myocardial involvement may also occur. The condition is caused by a mutation in the ATP-binding cassette subfamily C member 6 (ABCC6) gene, inherited in either an autosomal dominant or recessive pattern. Diagnosis is usually confirmed by skin biopsy. The prevalence of PXE is estimated to be between 1 in 50,000 and 1 in 200,000 adults. The disease occurs at any age and, although average life expectancy is normal, premature death in childhood is a recognised risk. Acceleration of symptoms may occur during pregnancy. The majority of patients with PXE who undergo anaesthesia and surgery have an uncomplicated course and outcome. Those with severe cardiovascular involvement are at greater risk, as are those suffering complications such as gastrointestinal haemorrhage, requiring emergency surgery.

SYSTEMS AFFECTED

SKIN

There is a variable tendency to loosening of the skin, particularly in the neck, face, axillae, abdomen and groin (including the genitalia). Most affected individuals show evidence of these changes by their second decade, but some may have minimal involvement even in later life. The loose skin becomes thickened

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and 'pebbled', sometimes being described as 'peu d'orange'. A characteristic skin deposition of yellowish lesions, mainly in the antecubital fossae, gives a xanthoma-like or 'cobblestone' appearance and the disorder its name. Similar deposition and calcification may occur in mucous membranes, lower lip, rectum and vagina.

EYE

PXE produces well-recognised ocular manifestations in the form of retinal streaks extending radially from the optic disc over the fundus. The 'angioid streaks' are the result of reduced elasticity of Bruch's membrane, and may lead to scarring and retinal or vitreous haemorrhage. Visual impairment usually presents in the second decade of life with characteristic preservation of peripheral vision.

VASCULAR

Arterial involvement may lead to a reduction or absence of peripheral pulses, often with associated medial calcification. Distal ischaemic problems are uncommon, presumably because the slow process allows time for collateral circulation to develop. Renovascular arterial occlusion may cause severe hypertension, even in young patients. Vascular aneurysms and arteriovenous malformations also occur.

AIRWAY AND RESPIRATORY SYSTEM

Laryngeal involvement has been reported. This may cause loss of elasticity of the laryngeal structures leading to difficult tracheal intubation. Respiratory complications may occur secondary to cardiac involvement.

HEART

Calcification within the endocardium may affect conduction, and predisposes to arrhythmias. Mitral valve thickening and incompetence/prolapse have been reported. Coronary arterial lesions may result in severe angina, even in the young; myocardial ischaemia and infarction may result. Coronary artery bypass grafting has been necessary in teenage sufferers and a fatal myocardial infarction has been reported in a 6-week-old infant.

GASTROINTESTINAL SYSTEM

Acute gastrointestinal haemorrhage is common, often presenting in children and young adults. This is due to gastric and duodenal micro-aneurysms and arteriovenous malformations compounded by impaired normal healing responses to minor abrasions of the mucosa. Peptic ulceration and oesophagitis also occur.

CENTRAL NERVOUS SYSTEM

Stroke, cerebral haemorrhage and calcification of the falx cerebri are recognised.

PREOPERATIVE ASSESSMENT

Patients may require surgery for the ocular, cardiovascular, peripheral vascular or gastrointestinal complications. Increasingly, patients present for the surgical treatment of disfiguring cutaneous manifestations.

Assess the extent of the disease, taking note of the degree of involvement of the cardiovascular and respiratory systems:

- Angina, myocardial infarction
- Dyspnoea
- Syncopal attacks (arrhythmias)
- Arterial pulses
- Blood pressure
- Heart murmurs
- Assess venous access, both peripheral and central

INVESTIGATIONS

- ECG
- Chest X-ray
- Urea and electrolytes (baseline test of renal function)

Temporary cardiac pacing may be indicated in patients with conduction defects.

PERIOPERATIVE MANAGEMENT

- BP monitoring, although essential, may be difficult.
- Noninvasive methods are likely to be unreliable in the presence of occlusive arterial disease.

- Invasive BP monitoring may be difficult because of arterial occlusion or vessel calcification.
- Care should be taken to avoid upper gastrointestinal trauma from the use of an oesophageal temperature probe or cardiac output monitor.

ANAESTHETIC TECHNIQUE

There appear to be no specific contraindications to the use of any particular anaesthetic drug. However, in planning the anaesthetic technique, the following points should be borne in mind:

- Venous access, both central and peripheral, may be difficult due to the loose and thickened skin: antecubital fossae are often unsuitable; neck involvement may prevent use of the internal jugular veins; groin involvement may prevent use of the femoral veins; venous cut-down or inhalational induction may be necessary.
- Risks of undiagnosed cardiac ischaemia and dysrhythmias.
- Risk of major haemorrhage.
- Risks of hypertension, e.g. subendocardial ischaemia, intracranial haemorrhage and in particular retinal haemorrhage. Avoiding the hypertensive response to laryngoscopy is critical.
- Consideration of the relative risks of GI haemorrhage with the use of postoperative NSAIDs.
- Bacterial endocarditis prophylaxis may be required.
- Care should be taken to avoid upper gastrointestinal trauma from the use of a nasogastric tube.
- Thickened loose skin may make regional techniques difficult.

AIRWAY CONSIDERATIONS

- Tracheal intubation may be difficult because involvement of the laryngeal ligaments and cartilages may cause laryngeal rigidity.
- Equipment for both video laryngoscopy and fibre-optic intubation should be available.
- Rigid bronchoscopy has been necessary to assist with endotracheal intubation.

OBSTETRIC ANAESTHESIA

There is limited evidence for the management of parturients. Most women with PXE have normal pregnancies and the disease has no known effects on the foetus. Heavy straining may result in retinal bleeding so consideration should be given to a shortened second stage of labour and epidural analgesia. During caesarean section, care should be taken to avoid swings in blood pressure during the establishment of regional block with a theoretical benefit to epidural over spinal anaesthesia.

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SCLERODERMA

Scleroderma, sometimes known as systemic sclerosis, is a chronic multisystem connective tissue disease. Inflammation then fibrosis of small vessels, particularly arterioles, occurs as a result of an excess production of collagen, fibrinonectins and glycosaminoglycans in connective tissue. This results in end-organ damage to many organs, in particular skin, gastrointestinal tract, lungs, heart and kidneys.

Cases of scleroderma are classified as limited cutaneous or diffuse cutaneous based on the degree of skin involvement. Limited cutaneous scleroderma is usually restricted to the hands, arms and face and preceded by Reynaud's phenomenon. Diffuse systemic sclerosis progresses rapidly, affects a larger area of skin and involves more extensive systemic organ damage.

There is evidence of an autoantibody response and the types of antibody present may be used to

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categorise different subgroups. Females are affected more than males (3–14 times). Onset is usually between the ages of 20 and 50 years. Symptoms may be exacerbated by pregnancy

CLINICAL FEATURES

- Shiny, waxy, taut skin with loss of skin folds.
- Perioral contractures which limit mouth opening and can lead to poor dental hygiene.
- Oesophageal involvement (dysphagia, reflux and stricture formation) in about 80% of cases.
- The heart is involved in approximately 50%–80% of patients leading to conduction defects, cardiomyopathy, pericarditis, cardiomegaly, arrhythmias, reduced ventricular contractility and pericardial effusions.
- Pulmonary fibrosis resulting in decreased compliance and decreased vital capacity. Pulmonary hypertension can develop, even in the absence of clinical signs and symptoms, and is associated with a poor prognosis.
- Weakness of the intercostal muscles and diaphragm has been reported.
- Skin of the chest wall may be involved thus limiting normal chest expansion.
- Renal disease may lead to hypertension. Hypertensive crises may follow withdrawal of angiotensin converting enzyme inhibitors.
- Lower gut involvement may result in malabsorption, obstruction or perforation.
- Other findings include peripheral neuropathies, Sjogren syndrome, non-pitting oedema, telangiectasia and diminished sweating.
- Raynaud disease is common.

The CREST syndrome, a variant of scleroderma, is the combination of calcinosis, Raynaud phenomenon, oesophageal involvement, sclerodactyly and multiple telangiectasia of the skin, lips, oral mucosa and gut.

PREOPERATIVE ASSESSMENT

- Assess the extent and duration of the illness.
- Assess venous access.
- Assess mouth opening: contractures are common.
- Assess state of dentition (often poor).
- Look for mucosal telangiectasia in the mouth.
- Assess neck mobility.
- Assess degree of cardiac and respiratory symptoms (e.g. exertional dyspnoea or the dry cough of pulmonary fibrosis); examine for cardiac dysrhythmias, a pericardial effusion and signs of pulmonary hypertension. Cardiopulmonary exercise testing may be useful.
- Check preoperative blood pressure.
- Ask about dysphagia and oesophageal reflux.
- Ask about symptoms of Raynaud phenomenon.
- Check for evidence of chronic renal failure.
- Examine for peripheral neuropathies.
- Check drug history: in particular for systemic corticosteroids and immunosuppressant drugs.

INVESTIGATIONS

- ECG and echocardiography.
- 24-h ECG recording may be indicated. The presence of a normal resting ECG does not exclude involvement of the heart in scleroderma: up to 40% of such patients may have abnormalities on 24-h rhythm analysis. There may be diastolic dysfunction, asymmetric septal hypertrophy or pericardial effusion.
- *Chest X-ray* – Look for signs of pulmonary fibrosis.
- *Cervical spine X-ray* – Assess associated spinal disease and neck mobility.
- *Urea and electrolytes* – Baseline test of renal function.
- *Full blood count* – Look for anaemia, leucocytosis or thrombocytopaenia.
- *Lung function tests* – Decreased FVC and/ or impaired DLCO in early scleroderma is predictive of lung disease. Pulmonary fibrosis may lead to decreased lung compliance and reduced total lung capacity or vital capacity. Impaired gas transfer may occur: DLCO is abnormal in 70% of cases of progressive systemic sclerosis.
- *Coagulation studies* – These may be abnormal due to malabsorption.
- *Thyroid function tests* – Hypothyroidism may occur in scleroderma.

- Antiphospholipid antibodies: presence confers a high risk of vascular thrombosis and may necessitate perioperative anticoagulation.

PERIOPERATIVE MANAGEMENT

- Xerostomia and poor dental hygiene may be helped by frequent preoperative mouthwashes.
- Steroid cover may be needed.
- Use prophylactic H2 blockers and prokinetic drugs (but beware of inducing arrhythmias), when oesophageal disease is present.
- Vitamin K may be required where malabsorption has led to a bleeding tendency.
- Temporary cardiac pacing may be indicated in selected patients with conduction defects.
- Pulmonary hypertension should be optimised.

INDUCTION

- Venous access may be difficult due to vasoconstriction; cut-down or central vein cannulation may be necessary. Arterial compression following inadvertent cannulation is likely to be difficult in the neck due to taught skin: ultrasound or the femoral site should be considered.
- An inhalational induction or fibre-optic intubation technique may be necessary.

AIRWAY

- Many patients have severe reflux disease and are at risk of aspiration.
- A rapid sequence induction may not be practicable if intubation difficulties are anticipated. Cricoid pressure may be ineffective if the oesophagus is fibrosed.
- Consider awake fibre-optic intubation. Intubation should be performed very gently to avoid injury to the mouth. Use oral intubation in preference to the nasal route as nasopharyngeal telangiectasia may lead to bleeding.
- In very severe cases, consider tracheostomy under local anaesthesia.

MAINTENANCE

- Maintain temperature at 37°C (risk of Raynaud phenomenon). Warm all intravenous fluids; warm theatre; use a warming blanket. Reduced lung and chest-wall compliance may make ventilation difficult.
- Dry eyes makes eye care especially important: eyelids should be taped shut; use bland ointments or artificial tears to avoid corneal scarring.
- Avoid hypotension and maintain fluid balance, especially in patient with renal involvement.
- Avoid vasoconstrictor drugs if possible.
- Reduced lung compliance runs the risk of barotrauma: lung protective ventilation strategies are required.

REGIONAL TECHNIQUES

- Nerve blocks, including brachial plexus and sciatic nerve, have been used successfully, but may be associated with prolonged duration of block.
- Raynaud phenomenon may follow wrist blocks.
- Avoid use of epinephrine-containing local anaesthetic solutions.
- Unilateral local anaesthetic stellate ganglion blocks have worsened Raynaud phenomenon on the contralateral side.
- Ischaemic effects of tourniquets make the use of intravenous regional anaesthesia unwise.

MONITORING

- Limb contractures may make blood pressure measurement difficult. Radial artery cannulation or sampling should be avoided if possible (if it is essential, then careful assessment of collateral flow [Allen's test] is necessary or use a larger artery).
- Temperature should be carefully monitored.
- Monitor blood glucose intraoperatively for those on steroids.
- Rotate pulse oximeter site during longer cases to prevent digital ischaemia.
- Oesophageal strictures may make placement of cardiac output monitors difficult.

POSITIONING

- Consider the risks of pressure effects on fibrotic skin during prolonged operations.
- Position the patient awake if possible.

EMERGENCE AND RECOVERY

- Due to the high risk of regurgitation, ensure the patient is fully conscious prior to extubation.

POSTOPERATIVE MANAGEMENT

- Common problems/complications include:
  - Respiratory failure (due to postoperative respiratory tract infection, aspiration or respiratory muscle weakness).
  - Increased postoperative analgesic requirements (possibly due to increased numbers of sensory nerve fibres in skin).
  - Prolonged sensory blockade with peripheral and central neuraxial techniques.
- Continue systemic steroid supplements.
- Maintain normothermia.

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SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease affecting most body systems and characterised by abnormal immune function. It is a complex disorder with genetic, hormonal and environmental pathogenesis and is associated with a wide variety of autoantibodies. Increased numbers of hyperactive B lymphocytes, together with impaired T-cell regulation, lead to the production of IgG antibodies which are specific for exogenous and endogenous antigens. The serum in SLE contains a variety of autoantibodies directed against nuclear material (anti-DNA antibodies in particular), and histological examination of affected tissue shows evidence of immune complex deposition.

SLE is more common in females than males (9:1), and the usual onset is in the third and fourth decades. It is an episodic disease with periods of prolonged remission punctuated by life-threatening exacerbations. Usually the onset involves arthralgia, fever, weight loss, rash, anaemia and leucopaenia; involvement of other organs produces a wide clinical spectrum of disease (Table 9.4). The presence of lupus nephritis indicates a poor long-term prognosis although most deaths are a result of cardiovascular complications: the risk of myocardial infarction is 50 times greater in SLE than the age/sex-matched general population. Many patients with SLE have subclinical atherosclerosis but the mechanisms underlying the increased cardiovascular risks are not fully understood.

Secondary antiphospholipid syndrome affects up to 30% of patients with SLE. An abnormal circulating anticoagulant (lupus anticoagulant) prolongs the activated partial thromboplastin time but also risks the formation of arterial and venous thromboses and predisposes to recurrent pregnancy loss.

Treatment of SLE is with a combination of NSAIDs, antimalarial agents, corticosteroids, immunosuppressant drugs (e.g. cyclophosphamide, methotrexate, azathioprine) and modulators of B-cell function. Cardiovascular and thrombotic risk is treated with aspirin and, in antiphospholipid syndrome, systemic anticoagulation.

PREOPERATIVE ASSESSMENT

- Assess the extent and duration of the illness. Discover whether the patient has acute disease or is in remission, as this will affect perioperative risk and outcome.

Table 9.4 Clinical features of SLE

- Bones/joints
  - Flitting arthralgia
  - Polyarthritis (wrists, elbows, knees)
  - Aseptic necrosis of large joints
- Skin
  - 'Butterfly' rash in malar region
  - Photosensitivity
  - Raynaud phenomenon
- Cardiovascular system
  - Atherosclerosis (often subclinical)
  - Pericarditis/pericardial effusion
  - Myocarditis
  - Endocarditis
  - Leg ulcers
  - Limb gangrene
  - Necrotic finger pulp lesions
  - Arterial thrombosis
- Respiratory system
  - Pleurisy/pleural effusions
  - Alveolar haemorrhage
  - Interstitial lung disease
- Renal system
  - Lupus nephritis:
    - Proteinuria
    - Haematuria
    - Abnormal urinary segments
  - Nephrotic syndrome
  - Hypertension
  - Acute and chronic renal failure
- Muscular system
  - Myalgia
  - Myositis
  - Wasting/weakness
- Nervous system
  - Seizures
  - Psychosis
  - Peripheral sensorimotor neuropathy
  - Hemiplegia
  - Cranial nerve palsies

(*Continued*)

Table 9.4 (Continued) Clinical features of SLE

- Gastrointestinal system
  - Peritonitis
  - Gastrointestinal haemorrhage
  - Vasculitic ischaemia
  - Chronic mesenteric ischaemia
- Haematological system
  - Anaemia
  - Leucopaenia
  - Thrombocytopaenia
  - Bleeding diathesis (circulating anticoagulants)
- Other
  - Pregnancy-induced hypertension

- Assess systemic involvement:
  - Cardiovascular (tachycardia, heart failure, cardiomegaly, pericardial effusion, valve lesions)
  - Respiratory (pleurisy, dyspnoea)
  - Renal (renal failure, hypertension)
  - CNS (peripheral neuropathy, CVA)
  - Raynaud phenomenon
- Check drug history:
  - NSAIDs
  - Antimalarials
  - Systemic corticosteroids
  - Other immunosuppressant drugs
  - Cardiac drugs
  - Anticoagulant and antiplatelet therapy

INVESTIGATIONS

- *ECG* – Myocarditis, dysrhythmias, pericarditis.
- *Echocardiography* – Cardiac contractility, pericardial effusions.
- *Chest X-ray* – Pleural effusion, interstitial lung disease, alveolar haemorrhage, pericardial effusion, cardiac failure.
- *Urea and electrolytes* – Baseline test of renal function.
- *Full blood count* – Anaemia, leucopaenia (immunosuppressant, SLE), thrombocytopaenia (immunosuppressant).

- *Lung function tests* – Signs, symptoms and chest X-ray may not reflect degree of pulmonary involvement; pulmonary fibrosis will cause a restrictive defect; diffusing capacity may be reduced.
- *Coagulation* – Prolonged APTT or KCCT due to anticardiolipin antibody and lupus anticoagulant. PT may be normal.
- *Plasma cholinesterase levels* – Reduced by plasmapheresis and cyclophosphamide.

PERIOPERATIVE MANAGEMENT

- Preoperative optimisation of cardiovascular and respiratory systems. In particular:
  - Control of hypertension
  - Treatment of angina
  - Treatment of arrhythmias
  - Treatment of heart failure
  - Drainage of pericardial/pleural effusions
- Patients with severe cardiovascular disease may require perioperative invasive haemodynamic monitoring.
- Perioperative steroid cover will be needed for the patient receiving corticosteriods.
- Take antithrombotic measures perioperatively in patients with antiphospholipid syndrome:
  - Antiembolism stockings
  - Subcutaneous low molecular weight heparin (or consideration of heparin bridging therapy where there is a history of thrombotic events)
  - Avoid dehydration (minimise starvation times, IV fluids)
- Blood transfusions can exacerbate SLE.

INDUCTION

- Avoid drugs and techniques likely to cause tachycardia, hypertension, hypotension or reduced myocardial contractility.
- Use a cardiostable anaesthetic and attenuate the hypertensive response to laryngoscopy.

AIRWAY CONSIDERATIONS

- There are rare reports of a narrowed airway due to laryngeal inflammation in SLE.

MAINTENANCE

- Avoid drugs and techniques likely to cause tachycardia, hypertension, hypotension or reduced myocardial contractility.
- The action of suxamethonium may be prolonged by cyclophosphamide.
- Avoid hypotension and maintain fluid balance, especially in the patient with renal involvement.
- Drug metabolism is altered in renal disease; avoid drugs which are metabolised/excreted by the kidneys.
- Hypoalbuminaemia in nephrotic syndrome will affect drug distribution.
- Avoid vasoconstrictors in Raynaud disease.

REGIONAL TECHNIQUES

- Be absolutely sure of the patient's coagulation status before embarking upon regional anaesthetic techniques.
- Document existing sensorimotor neuropathies prior to use of regional techniques.

MONITORING

- Urinary catheter and urine output monitoring is essential where there is pre-existing renal dysfunction.
- CM5 lead ECG is useful to detect myocardial ischaemia and arrhythmias.
- Radial artery cannulation may be inadvisable in the presence of Raynaud phenomenon.
- Transoesophageal echocardiography may be helpful intraoperatively in severe cardiac disease.
- Neuromuscular junction monitoring is essential if neuromuscular blockers are used.
- Blood glucose monitoring for patients receiving systemic steroids.

CONSIDERATIONS IN OBSTETRIC ANAESTHESIA

- Regional anaesthesia is the preferred technique for caesarean section.
- Isolated elevation of partial thromboplastin time secondary to lupus anticoagulant is not contraindication to regional anaesthesia.

POSTOPERATIVE MANAGEMENT

- Common problems/complications include:
  - Deterioration in cardiovascular function
  - Renal failure
  - Arterial or venous thromboses
- Continue systemic steroid supplements.

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URTICARIA AND ANGIO-OEDEMA

Urticaria is a well demarcated, usually pruritic, skin reaction characterised by erythematous, raised, palpable lesions, often with pale centres, which blanch on pressure. Urticarial lesions result from a transient increase in capillary permeability causing focal oedema of the superficial part of the dermis. Angio-oedema describes a condition in which there is circumscribed, non-pitting subepithelial oedema, sometimes with erythema. It may involve the eyelids, lips, tongue, larynx, pharynx, respiratory tract, gastrointestinal tract, renal system and, occasionally, the central nervous system. This may result in airway obstruction, pleural effusions, abdominal pain, vomiting, diarrhoea, hemiplegia and seizures.

Urticaria and angio-oedema often accompany each other; however, in those cases of angio-oedema due to a deficiency of the C1-esterase deficiency, urticaria does not occur. Individual attacks of urticaria and angio-oedema usually last no longer than 48 h. If episodes of urticaria/angio-oedema occur for more than 2 months, the condition is termed 'chronic'.

Many common forms of urticaria and angio-oedema (Table 9.5) result from the antigen-induced release of biologically active substances from mast cells which are found in organs rich in connective tissue (e.g. skin, respiratory tract, etc.). Urticaria/angio-oedema may also be caused by drugs which cause direct mast cell degranulation or which activate the arachidonic acid or complement pathways. In all these cases, release or activation of mediators (e.g. histamine, heparin, tryptase, bradykinin, chemotactic factors, prostaglandins, leucotrienes, platelet activating factor [PAF], adenosine, oxygen radicals)

Table 9.5 Aeitology of urticaria and angio-oedema

- Idiopathic
- Foodstuffs and additives
- Drugs:
  - Antibiotics
  - Muscle relaxants, e.g. tubocurarine
  - Opiates, e.g. morphine
  - Heparin
  - Protamine
  - Antihypertensives, e.g. ACE inhibitors
  - NSAIDs
  - Antifibrinolytics, e.g. alteplase
  - Psychotropic drugs
- Insect bites and stings
- Physical:
  - Dermatographism
  - Cold ('cold urticaria')
  - Heat
  - Vibration
  - Exercise
  - Delayed pressure urticaria
- Infections
- Collagen vascular disease
- Endocrine disease
- Vasculitis
- Malignancy
- Hereditary and acquired angio-oedema
- Miscellaneous

causes altered vascular permeability, smooth muscle contraction and chemotaxis of leucocytes. This results in a spectrum of signs and symptoms ranging from simple urticaria or angio-oedema to fulminant anaphylaxis which can be categorised as IgE-mediated or non-IgE mediated (anaphylactoid reactions).

Some forms of angio-oedema result from a functional deficiency of the inhibitor of the first component of the complement cascade (C1), and this is known as C1 esterase inhibitor (C1EI). Hereditary angio-oedema (HAO) is an autosomal dominant disorder characterised by recurrent spontaneous episodes of oedema of the skin and the mucous membranes of the respiratory tract and gut. Minor trauma, concomitant illness or peri-oral surgery (e.g. dentistry or tonsillectomy) may precipitate an attack. Attacks may increase during pregnancy (when C1EI levels are low) or during menstrual bleeding and are non-responsive to antihistamines. The major serious complication of an acute attack of HAO is upper airway obstruction, although oedema of the bowel wall may be mistaken for an acute abdomen and result in unnecessary, and risky, surgery. Low levels of C1EI also occur as an acquired disorder (Table 9.6). Acquired C1EI deficiency can be distinguished from HAO by the absence of complement abnormalities in other family members, late age of onset and by the reduced level of C1 seen in the acquired form.

Table 9.6 C1-esterase inhibitor (C1EI) deficiency

Idiopathic hereditary C1EI deficiency
- *Type 1 (85%)*
  - Impaired synthesis
  - Mostly autosomal dominant
- *Type 2 (15%)*
  - Normal or high C1EI level but dysfunctional protein
  - Heterogeneous genetic groups
  - Mostly autosomal dominant

Acquired C1EI deficiency
- B-cell lymphoproliferative disorders
- Connective tissue diseases
- Monoclonal gammopathies
- Antibodies to C1EI

LONG-TERM TREATMENT OF URTICARIA/ANGIO-OEDEMA

CHRONIC URTICARIAL/ANGIO-OEDEMA

- Identify and avoid precipitating factors.
- Avoid drugs that may aggravate disease, e.g. salicylates, NSAIDs, opiates.
- Treat with H1-receptor antagonists, e.g. chlorpheniramine.
- Add a beta-adrenergic agonist, e.g. terbutaline.
- Add an H2-receptor antagonist, e.g. ranitidine.
- Consider use of a tricylcic antidepressant, e.g. doxepin (this acts against both H1- and H2-receptors).
- Corticosteroids are used only in very severe disease.
- Adrenaline is used for severe attacks of anaphylaxis.

C1EI DEFICIENCY (HEREDITARY AND ACQUIRED)

- Androgens, e.g. stanozolol; stimulate hepatic synthesis of C1EI.
- Antifibrinolytic agents, e.g. tranexamic acid or epsilon aminocaproic acid (EACA); these inhibit plasmin activation (plasmin is a potent catalyst for complement activation).
- Purified C1EI concentrate in severe and recurrent attacks where oral prophylaxis is inadequate or not tolerated.

PREOPERATIVE ASSESSMENT

- Specifically ask about:
  - Atopy
  - Hypersensitivity
  - Drug reactions
  - Family history
  - Previous episodes of urticaria/angio-oedema, including frequency, duration, effect, etc.
  - Drug therapy (see above)

INVESTIGATIONS

- C1EI function in HAO.
- Radioallergosorbent (RAST) tests.
- Skin testing may be useful, but results are often unreliable and there is a small risk of severe anaphylaxis during test.

PERIOPERATIVE MANAGEMENT

- Avoid precipitating drugs or factors in chronic urticaria/angio-oedema.

PREMEDICATION

- In all cases of urticaria/angio-oedema, suitable premedication should be used to allay anxiety: stress may precipitate an acute episode.
- In chronic urticaria/angio-oedema the following agents may be useful as a part of the premedication:
  - H1 antagonists
  - H2 antagonists
  - beta-Adrenergic agonists
  - Corticosteroids
- In HAO attempts should be made to increase C1EI levels preoperatively using:
  - Androgens
  - Antifibrinolytics
  - Purified C1EI concentrate
  - Fresh frozen plasma (FFP) in an emergency where access to other treatments is limited

If the patient is not receiving long-term therapy with androgens or antifibrinolytics, these should be administered for several days prior to surgery. Although they start to act within 24 h, they require 1–2 weeks to reach maximum effect. Purified C1EI concentrate should be administered preprocedure; the timing depends upon the preparation used. Alternatively, the administration of 2 units of FFP given in the immediate preoperative period will restore the C1EI to a safe level (40% of normal) for between 1 and 4 days. These measures also seem appropriate for the acquired form of C1EI deficiency, although there is little evidence of their efficacy.

PERIOPERATIVE MANAGEMENT

INDUCTION/MAINTENANCE

- Venepuncture has precipitated forearm angio-oedema in a patient with HAO.
- Use the most 'immunologically benign' agents, particularly in atopic patients. Avoid histamine-releasing drugs, where possible.
- Avoid airway manipulation and intubation, where possible.
- In cold urticaria:
  - Warm intravenous fluids.
  - Warm the laryngoscope blade.
  - Use a warming blanket and warm air circulating cover.
  - Humidify respiratory gases.

MONITORING

- Monitor core temperature, especially in cold and cholinergic (heat) urticaria.

POSITIONING

- In pressure urticaria use extra protection for bony prominences, tourniquets, etc.

REGIONAL TECHNIQUES

- These may allow the avoidance of tracheal intubation.

CARDIOPULMONARY BYPASS

- Cardiopulmonary bypass (CPB) has been undertaken successfully in patients with certain forms of urticaria/angio-oedema.

MANAGEMENT OF ACUTE ATTACKS OF URTICARIA AND ANGIO-OEDEMA

- Always have facilities available to treat anaphylaxis or airway obstruction, e.g. nebulised adrenaline, intubation equipment and tracheostomy facilities.
- Treat acute attacks of chronic urticaria/angio-oedema with adrenaline, steroids and

antihistamines and fluid resuscitate with crystalloid.

- Treat attacks of angio-oedema due to C1EI deficiency with recombinant C1 inhibitors (conestat alfa and icatibant). Alternatives are purified C1EI concentrate (1000–1500 plasma units) and, in the absence of other treatments, FFP though this can sometimes precipitate worsening clinical features.
- There is unlikely to be any response during an acute attack of HAO to adrenaline, steroids or antihistamines.
- Monitor coagulation status in HAO.
- Remember that C1EI levels will fall due to haemodilution if patients with C1EI deficiency undergo CPB or in the event of crystalloid volume replacement during a period of acute blood loss.
- CPB in a patient with cold urticaria has led to a rise in arterial histamine levels during rewarming.

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PART

2

SURGICAL PROCEDURES

Abdominal surgery

10

BRIAN J POLLARD AND GARETH KITCHEN

ABDOMINAL TRAUMA

- Intra-abdominal injuries may carry a high morbidity and mortality. Diagnosis may be difficult or delayed and severity underestimated.
- A high index of suspicion of abdominal trauma is required in all patients who have sustained serious or high energy injury.
- The principal cause of death is uncontrolled bleeding, particularly from bursting injuries of the liver or spleen.
- Blunt trauma is common in road traffic accidents secondary to seat-belt injuries. The spleen is the most vulnerable organ. Liver, pancreas, bowel, kidneys and bladder are injured by greater forces.

- Penetrating trauma occurs in 20% in the UK. It may be due to low velocity projectiles, e.g. knives, hand gun bullets or high velocity projectiles, e.g. rifle bullets or shrapnel from bombs or blasts. Impaled objects or weapons must only be removed under controlled circumstances in theatre. Approximately 90% of gunshot wounds sustain visceral injuries.
- Patients often require exploratory laparotomy and repair of damaged viscera.
- The concept of 'damage control surgery' has been gaining popularity. The least possible is done for the patient at the first operation. The patient is transferred to ICU to correct hypothermia, acidosis and coagulopathy, before definitive surgery is undertaken within the next 24–48 hours.

- Some abdominal injuries (e.g. liver and spleen) may not require surgery and improved outcomes have been shown with conservative management.

INITIAL MANAGEMENT

- Injuries to the abdomen cannot be managed in isolation and require a multidiciplinary team approach.
- Full assessment and resuscitation is essential before definitive investigations are carried out and the decision to embark on surgery is made.
- The ATLS scheme is recommended to achieve stabilization in a consistent and systematic order and life-threatening injuries treated according to priority.

PRIMARY SURVEY

- An **ABCDE** approach is followed.
    - **A**irway: assess patency and manage as appropriate, with tracheal intubation if necessary. The cervical spine is immobilized.
    - **B**reathing: assess and where necessary commence IPPV. Life-threatening chest injuries are excluded and treated.
    - **C**irculation: insert IV access and control external haemorrhage by compression. Assess circulation and blood volume. Remember that in young patients blood pressure is often maintained until the final phase of shock, when catastrophic and sometimes irretrievable falls occur.
    - **D**isability and **E**xposure are assessed and managed as appropriate.
- Choice of fluid for resuscitation is controversial. ATLS recommends warmed crystalloid solutions. Request cross-matched blood immediately. With the exception of imminent exsanguination, there is little indication for the use of O-negative or type-specific blood. Cross-matched blood should be available within 20 minutes.
- Excessive fluid resuscitation in penetrating trauma prior to haemostasis may be detrimental. In patients who have sustained

major vascular injuries, increasing blood pressure leads to clot disruption and increased bleeding.

SECONDARY SURVEY

- Once stabilized, a full 'top-to-toe' examination is carried out.
- Constant reassessment and vigilance is required for on-going life-threatening problems which require immediate treatment.

INVESTIGATIONS

- Full blood count, cross-match, coagulation
- Plasma electrolytes, creatinine, amylase
- X-ray c-spine, chest, abdomen, pelvis
- ECG
- Following erect chest and abdominal X-ray, as indicated, proceed to:
    - Deep peritoneal lavage
    - CT scan of abdomen
    - Ultrasound scan of abdomen
        - Focussed abdominal sonography in trauma (FAST)
    - Laparoscopy
    - Laparotomy
- In the 'FAST' protocol ultrasound scanning is performed simultaneously with initial assessment and resuscitation. The urinary bladder is filled with saline, then ultrasound is used to look for free fluid in the pericardium, perisplenic and perihepatic areas (including Morrison's pouch) and paracolic gutters. In the absence of fluid, no further immediate radiology is indicated. A positive scan indicates a CT in a stable patient, or laparotomy in an unstable patient. Solid organ trauma is not assessed by ultrasound in this protocol.
- Great care should be exercised when deciding to move a potentially unstable patient from the emergency department to a remote environment, e.g. CT scanner.
- Indications for immediate laparotomy:
    - Unexplained shock
    - Rigid silent abdomen
    - Evisceration

- Radiological evidence of free intra-peritoneal gas
- Radiological evidence of ruptured diaphragm
- All gunshot wounds

LAPAROTOMY FOR ABDOMINAL TRAUMA

- Resuscitate and correct hypovolaemia prior to induction as far as possible. When haemorrhage is ongoing, proceed cautiously with anaesthesia.

MONITORING

- ECG
- Arterial line
- Pulse oximeter
- Central venous line
- Temperature
- Urine output
- Transoesophageal Doppler

ANAESTHETIC TECHNIQUE

- Two large bore cannulae are essential. Vasopressors and inotropes should be prepared in advance and intravenous fluids should be running, through a rapid infuser and blood available.
- Induce anaesthesia in theatre with the surgeon scrubbed ready. Severe cardiovascular decompensation may occur due to the vasodilatory and myocardial depressant effects of anaesthetic drugs plus the loss of a tamponade effect from the abdominal muscles at the institution of neuromuscular blockade.
- Rapid sequence induction with care to protect the potentially unstable cervical spine. Suxamethonium or rocuronium are the relaxants of choice.
- Choice of anaesthetic drugs will depend upon the patient. Far more important than the actual drug given is the dose. The intravascular volume is reduced in hypovolaemic patients;

therefore, usual doses will result in higher than expected plasma concentrations. Etomidate (0.1–0.3 mg/kg) is a popular choice due to its cardiovascular stability. Ketamine (0.3–0.7 mg/kg) may also be used; its sympathomimetic effects help to maintain blood pressure, though may lead to a false sense of security in the patient who already has a maximal endogenous sympathetic drive. Avoid ketamine in patients with head injuries because of possible effects on intra-cranial pressure.
- An alternative technique is high dose opioid combined with a benzodiazepine. This has minimal cardiac depressive action but may result in a decrease in endogenous catecholamine output and the patient will require postoperative ventilation.
- Intubation may be difficult in patients with facial injuries, cervical spine immobilization and blood in the airway.
- Maintenance is with cautious amounts of volatile agent in an air/oxygen mixture. Avoid nitrous oxide to prevent bowel distension. Once haemodynamic stability has been achieved, anaesthesia may be deepened.
- Use an intermediate acting relaxant.
- A nasogastric tube may be used to aspirate gastric contents.
- Prophylactic broad-spectrum antibiotics according to hospital policy.
- Once control of bleeding has been achieved, the patient's cardiovascular status should improve. Further fluid resuscitation should be guided by on-going losses, central venous pressure, transoesophageal Doppler and urine output. Blood should be taken at intervals for arterial gases, haemoglobin concentration, coagulation screen and electrolytes.
- Warming is essential. Hypothermia results in coagulopathy, reduced metabolism of citrate and lactate, hypocalcaemia, increased incidence of cardiac arrhythmias, metabolic acidosis and cardiac arrest. In the longer term, hypothermia impairs immune function, increases the risk of septic consequences and impairs wound healing.

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MASSIVE TRANSFUSION

- Coagulopathy is common after about 1 blood volume transfusion, due to dilutional thrombocytopaenia, ↓ coagulation factors (~40% of normal), DIC/fibrinolysis.
- Hypothermia causes inhibition of enzyme function and increased fibrinolytic activity.
- Massive transfusion results in metabolic acidosis, hyperkalaemia and impaired oxygen carrying capacity (↓ 2,3-DPG).
- Intra-operative red cell salvage (Cell Saver) provides warm blood with normal levels of 2,3-DPG. It is contraindicated if the blood is contaminated with intestinal contents.
- Coagulopathy is corrected with fresh frozen plasma, cryoprecipitate and platelet transfusions. They should be requested early if their use is anticipated. Recent evidence from the battlefield and in civilian practice suggests FFP:blood ratio should be at least 1:1.
- There may be a role for the use of antifibrinolytic and platelet activating drugs, e.g. tranexamic acid, DDAVP.

POSTOPERATIVE MANAGEMENT

- Admit to HDU or ICU.
- Those who have sustained massive blood loss and undergone transfusion are often hypothermic, coagulopathic, acidotic and require drugs to support their cardiovascular system. They will benefit from a period of ventilation in the ICU.
- Oxygen, fluids and analgesia are prescribed. An opioid infusion or patient-controlled analgesia system is appropriate. Once clotting has returned to normal the patient may benefit from the insertion of an epidural.

LIVER TRAUMA

- Liver injuries range from trivial to fatal.
- Comprise 45% of abdominal trauma; 30%–40% are due to penetrating injuries; 60% are associated with other injuries, especially life-threatening head injuries.

- Graded I–V, from minor lacerations to avulsion from the IVC.
- May be treated by insertion of packs which are then removed 24–48 hours later, following a period of stabilization, correction of coagulopathy and hypothermia, and transfer to a specialist centre. By this stage haemorrhage will often have stopped.
- Patients may be investigated by angiography, permitting bleeding vessels to be embolized before further surgery to remove packs, or occasionally in a stable patient it is the sole intervention.
- In the most severe liver injuries (Grade V) with simultaneous damage to the hepatic veins or vena cava, caval-atrial or caval-caval bypass may be required. Survival from such injuries is less than 10%.
- Stable patients, with a liver injury detected on CT scan, are best managed conservatively in a specialist centre. Transfusion requirements are reduced and there are fewer abdominal complications. Approximately 34%–51% of adult blunt hepatic injuries can be managed conservatively, although in Grade V injury, only 10% are stable enough.
- Conservative management of solid abdominal visceral injuries is not a passive process; continuous assessment is required. The patient may need emergency laparotomy at any time for potentially exsanguinating haemorrhage. ICU monitoring is mandatory.

SPLENIC INJURY

- Splenectomy is a relatively simple surgical procedure which has saved many lives. It is not necessary for all injuries and may occasionally be associated with overwhelming post-splenectomy sepsis.
- Most splenic injuries are managed conservatively. Approximately 50% of spleens can be repaired by partial resection, ligation of bleeding vessels and packing and enveloping the spleen in an absorbable mesh bag.
- If haemostasis is obtained, the incidence of further haemorrhage is <2%.

- In stable patients conservative treatment is now advocated. Careful patient selection is required, and laparotomy undertaken for haemodynamic instability, signs of peritonism or transfusion requirements over 2 units.
- Angiography and embolization techniques have also been employed.
- Observation in ICU or HDU is essential, with continual readiness for an emergency operation.
- Patients who have undergone splenectomy are protected by daily administration of oral penicillin for 2 years and immunised against pneumococcus and haemophillus influenzae.

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CROSS-REFERENCES

Major trauma, Chapter 30
Blood transfusion, Chapter 30

BARIATRIC SURGERY

The incidence of obesity is increasing in the developed world and morbid obesity has become a significant health problem. Many patients are younger adults but any age can be encountered. The most commonly accepted definition of morbid obesity is a body mass index of 40 kg m^2 or greater. A further category, the super-obese, is used for those with a BMI of greater than 55 kg m^2. The initial treatment options include dietary and lifestyle changes. Surgery is available as a later treatment option. The morbidly obese patient may present for gastric banding or bypass surgery. They may also present for surgery unrelated to their weight. In all cases, management strategies are similar.

PATIENT CHARACTERISTICS

- Any age
- Males and females equally affected
- Increased incidence of
  - Diabetes mellitus
  - Cardiovascular disease
  - Respiratory disease
  - Sleep apnoea syndrome

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Full blood count and coagulation screen if indicated.
- Electrolytes and liver function tests.
- Chest X-ray and ECG where indicated.
- Careful assessment of mouth, jaw opening and airway.
- Any further investigations according to coexisting medical conditions.

ANAESTHETIC TECHNIQUE

- Premedication is not usually necessary.
- Antibiotics as per hospital protocol.
- Consider prophylaxis against gastric acid aspiration.
- Routine standard monitoring.
- Invasive direct blood pressure monitoring is recommended.
- Neuromuscular blockade monitor.
- Adequate intravenous access.
- Discuss fibre-optic intubation with patient if this technique is indicated.
- Get the patient to position themselves on the operating table before starting and induce anaesthesia in the operating theatre.
- Ensure that the working load of the operating table is correct for a patient of that weight.
- Laparoscopic gastric surgery is often performed with the surgeon standing between the patient's legs and with some head-up tilt. Position a prop (padded roll bar) under the buttocks before starting so that the patient cannot slide down the table when tilted head-up.

INDUCTION AND MAINTENANCE

- Regional blocks can be challenging. Ultrasound guidance may help. Longer block needles are available.
- Full preoxygenation is important.
- Induction: propofol is recommended.
- Tracheal intubation.
- Relaxants: any short or intermediate acting agent is suitable for intubation followed by intermittent boluses guided by neuromuscular monitoring.
- Maintenance: air/oxygen with a volatile agent. Desflurane is commonly used due to its rapid onset and recovery. A propofol infusion (TIVA or manually controlled) is also suitable. If TIVA is used, the ideal body weight or the corrected body weight should be used and not the actual body weight.
- Controlled ventilation (higher pressures will be needed and PEEP is recommended).

- Analgesia: remifentanil is useful. Intravenous paracetamol and a non-steroidal anti-inflammatory drugs are useful if there is no contraindication.
- Additional opioid will be required at the end of the procedure if remifentanil has been used.
- Antiemetic prophylaxis: commonly a 5HT3 inhibitor and dexamethasone.
- Local anaesthetic infiltration around incisions and laparascopic port sites.

RECOVERY: EARLY

- Oxygen via face mask.
- Nurse in the semi-recumbent position.
- Intravenous fluid until the patient is drinking adequately.
- Analgesia:
  - This depends on the operation and expected analgesic requirements.
  - Simple oral analgesics may be adequate.
  - Intravenous opioids via PCA may be used.
  - A continuous epidural infusion or patient controlled epidural analgesia is an option.

RECOVERY: LATE

- Complications may be secondary to hypoventilation, poor pain control, or immobility. Basal atelectasis, deep vein thrombosis and hypoventilation may occur. Physiotherapy, early mobilization and thromboembolic prophylaxis are recommended.

Corrected body weight $= IBW + [0.4 \times (TBW - IBW)]$

Where

IBW = Ideal body weight and TBW = Total body weight

(Servin's correction)

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CROSS-REFERENCES

COLORECTAL SURGERY

COMMON LOWER GI TRACT SURGICAL PROCEDURES

- Appendicectomy
- Anterior resection (L/R hemicolectomy)
- Hartmann's procedure
- Total colectomy
- Panproctocolectomy (abdominoperineal resection)
- Ileorectal pouch formation
- Stoma formation/closure
- Fistula surgery
- Haemorrhoidectomy
- Other perianal surgery, e.g. fissure, haematoma, abscess, carcinoma

Procedures vary in complexity. Most involve laparotomy, excision of the affected section of bowel, anastamosis of the remaining segments, ± stoma formation. Adjacent organs may be involved (e.g. bladder, uterus) requiring a multidisciplinary approach, prolonged anaesthesia and major blood loss. Laparoscopic techniques are now being used for many procedures. Advantages of improved diagnosis, reduced wound size and tissue trauma must be weighed against the problems associated with laparoscopic surgery (pneumoperitoneum, increased operating time) and the anaesthetist must be prepared for conversion to laparotomy at any stage.

Enhanced recovery after surgery (ERAS) protocols allow early mobilisation and discharge following colorectal surgery, with rapid restoration of GI tract function. Such programmes consist of a package including:

- Admission and discharge planning.
- Teaching patients stoma care prior to hospital admission.
- Maintaining GI tract function by continuing feeding until shortly before surgery (high-calorie preoperative drinks).
- Low neurohumoral stress response (epidural analgesia, active warming).
- Stroke volume optimisation.
- Avoidance of or immediate postoperative removal of the nasogastric tube.
- Forced early mobilisation and diet.
- Day 1 removal of epidural catheter.
- Early discharge.

PATIENTS

- All age groups.
- Physiological status depends on:
  - Underlying pathology
  - Concurrent medical conditions
- Patients with malignant disease may have significant weight loss, anaemia, bowel obstruction, electrolyte imbalance and metastatic disease.
- Often elderly with comorbidity, e.g. ischaemic heart disease, hypertension, pulmonary disease, diabetes mellitus or arthritis.

- Patients with inflammatory bowel disease are usually younger but have frequently undergone multiple surgical procedures and may present with severe sepsis due to bowel perforation or 'toxic megacolon'. Steroid therapy may cause immunosuppression and signs of sepsis may be masked.
- Patients of all age groups and levels of fitness present for perianal surgery. Pregnant women can present for urgent haemorrhoidectomy.

PREOPERATIVE ASSESSMENT

- Routine history and examination with special attention to concurrent disease.
- Specific to gastrointestinal pathology:
  - Anaemia
  - Weight loss
  - Diarrhoea and vomiting
  - Bowel obstruction
  - Sepsis
  - Medications (including steroids)
  - Metastatic disease

INVESTIGATIONS

- Full blood count, coagulation, group and save or cross-match blood (depending upon nature of surgery and local transfusion policy)
- Urea, creatinine and electrolytes, glucose, amylase, liver function
- ECG
- Chest X-ray where indicated
- Other investigations, e.g. echocardiogram, pulmonary function tests, as indicated by history and examination

PERIOPERATIVE MANAGEMENT

- Continue regular medication until the day of surgery, with consideration given to postoperative intravenous regimes.
- Prescribe intravenous fluids for patients given 'bowel prep' or those with diarrhoea or vomiting.
- Commence diabetics on an appropriate intravenous insulin regime.

- Routine monitoring.
- Large bore IV access in major cases where blood loss is anticipated.
- Arterial line in cases involving large fluid and blood losses, in patients with significant ischaemic heart disease or arrhythmias.
- CVP line may be indicated.
- Transoesophageal Doppler for fluid management and cardiac output optimisation if indicated.
- Following induction of anaesthesia a nasogastric tube and urinary catheter are inserted.
- Positioning: common positions include Lloyd–Davies, lithotomy, Trendelenburg, prone.
- Temperature: use warming mattresses, forced air warmers and warmed intravenous fluids. Monitor core temperature.
- Give prophylactic antibiotic at induction along with steroid cover if indicated.

ANAESTHETIC TECHNIQUE

- In emergency procedures and in unfasted patients, use rapid sequence induction. For elective major abdominal surgery, general anaesthesia with tracheal intubation and controlled ventilation is recommended.
- Choice of induction agent depends upon the physiological status of the patient and the length of the procedure. In septic, hypovolaemic or haemorrhaging patients and those with significant cardiovascular disease, etomidate may be preferred for its greater cardiovascular stability.
- Maintenance is usually with a volatile anaesthetic agent. Avoid nitrous oxide to prevent bowel distension. Total intravenous anaesthesia has been employed successfully in major abdominal surgery.
- Muscle relaxation is required: an intermediate acting agent, e.g. atracurium or vecuronium, is usually employed. Monitor neuromuscular block.
- Fluid therapy: commence intravenous infusion of crystalloid solution via a fluid/blood warmer prior to induction. Monitor fluid balance throughout surgery, taking into account

blood loss, evaporative losses from exposed bowel, urine output, trends in central venous pressure. Use crystalloids, colloids and blood as appropriate. Major blood losses may occur requiring blood transfusion. Coagulopathy may ensue requiring administration of blood products such as FFP and platelets.

- Studies have shown an increase in tumour recurrence, increased infection rates and a poorer outcome in cancer patients who had received perioperative blood transfusions. Transfusion should therefore be avoided if possible.
- Analgesia may be provided with intravenous opioids. Nonsteroidal anti-inflammatory drugs and intravenous paracetamol have an opioid sparing effect. Alternatively, regional anaesthetic techniques may be employed.

ANAESTHESIA FOR LAPAROSCOPIC COLORECTAL SURGERY

Laparoscopic and laparoscopically assisted colorectal procedures are becoming increasingly popular. Benefits in terms of reduced surgical stress response and faster recovery need to be weighed carefully against problems associated with laparoscopic procedures and increased operating time. A Cochrane review in 2005 found short-term advantages including less pain, reduced ileus, preservation of normal pulmonary function, shorter length of stay, less wound complications and better quality of life in the first month after surgery. The overall conclusion, however, was that the evidence was insufficient to conclude that laparoscopic colorectal surgery was superior to open colorectal surgery when optimal perioperative management is applied.

ANAESTHETIC TECHNIQUE

- General anaesthesia with endotracheal intubation and muscle relaxation.
- Hypercapnoea and acidosis due to CO_2 absorption from pneumoperitoneum, prolonged procedure, and compromised ventilation in steep head-down

position may cause tachyarrhythmias and hypertension.
- Steep head-down position for several hours:
  - Increased venous return, increased cardiac output: caution in patients with congestive cardiac failure or valvular heart disease.
  - Risk of endobronchial intubation, reduced FRC and total lung volume: resulting in atelectasis.
  - Increased risk of gas embolism.
  - Cerebral oedema.
  - Facial and airway oedema.
  - Cephalad spread of epidural.
- Difficult access to IV lines due to positioning of arms at patient's sides to allow surgical access and problems with noninvasive blood pressure measurement (surgeon leaning on cuff): use extensions on lines and consider direct arterial pressure monitoring.
- Conversion to open procedure (laparotomy) may be necessary at any time.

POSTOPERATIVE ANALGESIA

Neuraxial blockade reduces the surgical stress response and may contribute to overall reduction in complications associated with major colorectal surgery in particular postoperative ileus. This benefit has also been shown in patients undergoing laparoscopic surgery.

Opioid PCA is a popular alternative. The minimally invasive approach results in less pain and less risk of ileus, and the volumes of narcotic analgesics are modest compared with open procedures.

REGIONAL BLOCKS

Some procedures are suitable for regional anaesthesia alone, for example, perianal surgery and refashioning of stomas. Major abdominal procedures are less suited to regional techniques alone; however, in some high-risk patients this may be the preferred technique.

A combination of light general anaesthesia and epidural blockade (Table 10.1) is now a popular

Table 10.1 Benefits of epidural blockade

- General
  - Good operating conditions
  - Good postoperative analgesia
- Reduced neuroendocrine stress response:
  - ↓ O_2 consumption
  - ↓ Catecholamine release
  - ↓ Postoperative negative nitrogen balance (↓catabolism)
  - ↓ Hyperglycaemia
  - ?↓ Immunosuppression
- Improved GI function
  - ↓ Duration of postoperative ileus (compared to opioids)
  - ↑ Gut mucosal perfusion by ↑ mesenteric blood flow
  - Faster recovery of gut function
  - Note that segmental blockade of dermatomes T5-T12 is required to ↓ sympathetically mediated peristaltic inhibition, preserving vagal and sacral parasympathetic outflow
- Improved respiratory function
  - ↑ Postoperative oxygenation
  - ↓ Pulmonary infections
- Cardiovascular benefits:
  - ↓ DVT
  - ↓ Blood loss

technique in major abdominal surgery (both open and laparoscopic).

- There is little convincing evidence to support concerns that epidural blockade may provoke breakdown of bowel anastomoses. Epidural blockade results in a relative increase in parasympathetic tone which may result in increased bowel activity. This, combined with hypotension, which sometimes accompanies epidural anaesthesia, may compromise mesenteric blood flow and affect anastomotic healing. Reduced gut blood flow in the presence of epidural analgesia is a direct function of arterial pressure and can be restored by raising arterial pressure. The balanced view overall favours the use of epidural analgesia, although this remains a matter of patient choice.

- Supplementing the local anaesthetic agents with opioid, clonidine, etc. may give a prolonged analgesic effect with minimal motor block.
- TAP block, bilateral ilio-inguinal/ilio-hypogastric nerve block and single-shot spinal block may also be effective.

PERIANAL SURGERY

- Procedures are usually short but often intensely stimulating and extremely painful postoperatively. They are often carried out on a day-case basis and, therefore, require a technique that allows the patient to be pain-free and fit for discharge within a few hours of surgery.
- Surgical stimulation to the anus can cause severe pain and may precipitate reflex responses, including laryngeal spasm, tachyarrythmias and bradyarrhythmias.
- Anaesthetic techniques aim to provide sufficient depth of anaesthesia and adequate analgesia to avoid these responses and yet allow rapid emergence.
- A spontaneously breathing GA technique using a laryngeal mask or face mask, and intravenous induction with propofol and a potent analgesic, e.g. fentanyl or alfentanil, is commonly used. Patient positioning dictates airway management. Lithotomy and steep head-down tilt increase the risk of aspiration of gastric contents and ventilatory compromise and may necessitate tracheal intubation and controlled ventilation. Patients may also be managed in the lateral position. Certain procedures require the patient to be positioned prone and 'jack-knifed', and then tracheal intubation is required, with positive pressure ventilation.
- Spinal and caudal extradural anaesthesia have both been used successfully in perianal surgery. The benefits include: avoidance of general anaesthesia, good postoperative analgesia and attenuation of the stress response. Regional techniques may be less suitable for day-case surgery where urinary retention may prove a concern and early mobilization is required.

A low spinal ('Saddle Block') aiming to anaesthetise S2–S5, can be performed in the sitting position (difficult in elderly and patients with severe perianal pain). A caudal extradural block may be used alone or in combination with GA. Local blocks, e.g. posterior perineal nerve blockade and local infiltration, have also been used for certain procedures, as sole anaesthetic as well as in combination with GA to provide postoperative analgesia.

POSTOPERATIVE MANAGEMENT

- Patients who have undergone major surgery or who have significant concurrent medical problems should be managed on HDU with invasive monitoring.
- Oxygen, intravenous fluids and appropriate analgesia should be prescribed and consideration given to antithrombotic prophylaxis.
- Antibiotics are prescribed according to local guidelines and provision made for restarting essential medication.
- Day-case patients should be assessed according to unit policy and discharged to the care of a competent adult with suitable analgesia. Clear instructions for follow-up should be given along with contact telephone numbers should problems occur.

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HEPATIC RESECTION SURGERY

Historically liver resection was associated with high mortality rates, due to uncontrolled bleeding and liver failure. This has improved significantly in recent decades with a 3% mortality rate for patients undergoing hepatic resection without significant parenchymal disease.

The liver is the only organ in the body that can regenerate, and in young healthy patients with normal liver parenchyma it is safe to remove four segments or 50%–60% of the liver.

INDICATIONS

- Isolated hepatic metastases
- Hepato-biliary tumours (benign and malignant)
- Donation of tissue for transplantation
- Trauma

SURGICAL TECHNIQUES

INITIAL

- Liver mobilisation from peritoneal attachments
- Cholecystectomy
- Expose vascular anatomy

RESECTION

- During resection blood loss is significantly reduced by temporary occlusion to the blood supply.
- Most commonly this involves the 'pringle manoeuvre' which involves total inflow occlusion of the portal vein and hepatic artery.
- More rarely total hepatic vascular occlusion is required; this involves clamping of the supra and infra hepatic vena cava.
- Both these techniques cause cardiovascular disturbance and significantly decrease cardiac output by decreasing preload and increasing afterload.

HAEMOSTASIS

- Ensuring haemostasis can be aided surgically with argon beam coagulation and fibrin glues.
- Adequate assessment of coagulation and appropriate use of clotting products can help the surgeon significantly.

PREOPERATIVE ASSESSMENT

The majority of patients have normal liver parenchyma, and should be assessed as for any other major intra-abdominal procedure. Patients with even moderate liver dysfunction have high surgical mortality rates and may not be offered surgery. The appropriate surgical selection is significant in the improved mortality rates in recent years.

PERIOPERATIVE MANAGEMENT

MONITORING

- Routine minimal monitoring
- Arterial line
- CVP line
- Cardiac output (e.g. oesophageal Doppler)
- Core temperature

ANAESTHETIC TECHNIQUE

Tracheal intubation and controlled ventilation are required. There is no evidence to suggest specific use of particular agents, although nitrous oxide should be avoided due to the risk of air embolism. Standard analgesia involves thoracic epidural placement. The risk of epidural haematoma has not proved significant providing adequate monitoring of postoperative coagulation and administration of fresh frozen plasma is considered prior to catheter removal.

The anaesthetist can also aid in the reduction of blood loss by controlling the CVP – CVP <5 cm H_2O will significantly reduce blood loss. This needs to be carefully weighed against the effects on the cardiovascular system. A low CVP is achieved by treating the postinduction hypotension with head down position, vasoconstrictors and avoiding significant fluid transfusion. After the surgical resection phase, the circulating volume can then be restored. In addition to bleeding, surgical compression of the vena cava can lead to sudden bouts of hypotension, consequently it is imperative to maintain good communication with the surgeon. Tranexamic acid can also be added to reduce blood loss.

POSTOPERATIVE CARE

Extubation is possible in most patients; however, they should be managed in the ICU due to the risk of postoperative bleeding and hepatic dysfunction. Most patients have some degree of liver function impairment and paracetamol should be avoided.

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CROSS-REFERENCES

Chronic liver disease, Chapter 4
The jaundiced patient, Chapter 4

HERNIA REPAIR

A hernia is a protrusion of the whole or part of a viscus from its normal position through an opening in the wall of its containing cavity. Surgical repair involves excision of the hernia sac and closure of the defect. Occasionally bowel resection and anastamosis are required. If hernia contents become constricted or twisted, strangulation occurs. Bowel obstruction may result indicating the need for urgent surgery. Laparotomy and bowel resection may be required.

TYPES OF HERNIA

- Inguinal
- Femoral
- Umbilical
- Paraumbilical
- Epigastric
- Incisional
- Obturator
- Spigelian
- Lumbar
- Gluteal
- Sciatic
- Perineal
- Hiatus
- Diaphragmatic

INGUINAL

- The most common; 75%–80% of abdominal wall hernias.
- Either gender and any age group.
- Repair early to reduce risk of strangulation and minimise stretching of abdominal wall musculature, hence reducing recurrence rate.
- Repaired by various techniques of herniorraphy and in infants, herniotomy.
- 3% recur and require further repair – a longer and more complex procedure.

- Laparoscopic repair is presently the most common technique of repair.
  - Extraperitoneal approach; problems of pneumoperitoneum avoided.
  - Complex procedure with significant risk of complications.
  - Reduced length of hospital stay and analgesic requirements.
  - Patient experience (pain, rehabilitation) superior to open approach.
  - Longer operative time than open repair.

FEMORAL

- Protrusion of the peritoneum into the potential space of the femoral canal.
- Sac may contain small bowel or omentum.
- More common in multiparous women; rare in children.
- Strangulate more readily than inguinal hernias (femoral canal is small).
- Often present with symptoms of small bowel obstruction.
- Repair at the earliest opportunity even when asymptomatic.

UMBILICAL AND PARA-UMBILICAL

- True umbilical hernias only occur in infancy. The most extreme form is exomphalos, where the midgut fails to return to the abdominal cavity in early foetal life. Urgent surgical repair is necessary and prognosis poor. A small hernia can occur in the first few days of life; repair is rarely required.
- Paraumbilical hernias are acquired and associated with: middle age, obesity, multiparous women. They occur at a small defect in the linea alba. There is a high risk of strangulation and repair is recommended even in relatively unfit and obese patients.

INCISIONAL

- Late complication of 10%–15% of abdominal wounds, 1–5 years postoperatively due to breakdown of a previous repair in muscle and fascia.

- Predisposing factors:
  - Obesity
  - Distension
  - Poor muscle quality
  - Inadequate closure technique
  - Postoperative wound infection
  - Multiple operations through same incision
- Usually asymptomatic but may strangulate when surgical repair is indicated.

PATIENTS

- All ages.
- May have concurrent medical problems especially in the elderly.
- Specific problems:
  - Chronic cough (COPD, asthma)
  - Chronic constipation
  - Obesity
  - Pregnancy
- Normally, a day-case procedure (50% in the UK; 70% in the US). Inpatient treatment is required for those with significant comorbidities.
- Repair under local anaesthesia is increasingly common.
- Occasionally patients present as an emergency with bowel obstruction.

PREOPERATIVE MANAGEMENT

- Routine history and examination, with particular attention to concurrent disease.
- Baseline investigations as dictated by local policy.
- Additional investigations as indicated by history and examination.
- Premedication is not normally indicated.
- Oral analgesia may be prescribed in advance as part of a multimodal, pre-emptive analgesic regime.
- IV fluids may be needed for patients with a history of vomiting, dehydration, hypovolaemia or electrolyte imbalance.

ANAESTHETIC MANAGEMENT

- All strangulated hernias with symptoms of bowel obstruction should be treated as an 'acute abdomen'. A general anaesthetic with rapid sequence induction is the technique of choice.
- Standard monitoring with IV access ± intravenous fluids.
- Antibiotics according to hospital policy.
- General anaesthesia may be preferred in anxious patients, children, obese patients and those with difficulty lying flat.
- For inguinal and femoral hernia repair with no risk of vomiting or regurgitation, a spontaneously breathing technique using a laryngeal mask may be appropriate.
- For abdominal wall and large incisional hernias and those involving bowel, muscle relaxation is required; tracheal intubation and IPPV is needed.
- Extubate with minimal coughing and bucking to protect the hernia repair.
- Avoid nitrous oxide in cases with bowel obstruction.
- Use a multimodal analgesia regime tailored to the type of hernia repair and postoperative pain expected. A short-acting opioid and NSAID and infiltration of the wound with local anaesthetic may be satisfactory in inguinal and femoral hernia repairs.
- A field block or TAP block will reduce intraoperative anaesthetic requirements and produce good quality postoperative analgesia.
- In cases where a large laparotomy wound is explored, an epidural may be beneficial, especially if the patient has concurrent pulmonary disease.

REGIONAL ANAESTHESIA

- Regional and neuraxial techniques are appropriate for elective hernia repair provided an intra-peritoneal approach is not required.
- Reduces stress response and provides good postoperative analgesia.
- Not recommended in emergency cases with bowel obstruction and hypovolaemia.
- Useful in high risk elderly patients.
- Patient must be able to lie flat.
- Sedation may be required. Oxygen should be administered by facemask or nasal cannulae.

LOCAL ANAESTHESIA

- May be used as the sole anaesthetic for patients considered unfit for other anaesthetic techniques. Intraoperative pain has been reported as a problem. In combination with general anaesthesia will reduce anaesthetic requirements and provide postoperative analgesia.
- Usually performed as a field block by the surgeon.
- Not suitable for the anxious, obese and uncooperative patients, or those with complicated hernias.
- All patients require standard monitoring and IV access.

POSTOPERATIVE MANAGEMENT

- Analgesia is best provided by a multimodal approach. A combination of opioids, paracetamol, NSAIDs and local blocks or wound infiltration with local anaesthetic is recommended.
- PCA may be preferable for patients undergoing large or bilateral hernia repairs.
- Those with significant cardiovascular or respiratory disease undergoing major abdominal wall hernia repairs benefit from an epidural.
- Oxygen and IV fluids should be prescribed and thromboprophylaxis considered.
- Chest physiotherapy is recommended for all patients with respiratory disease.
- Assess day-case patients in accordance with hospital policy before discharge. Provide analgesia and information regarding follow-up and contacts should problems arise.

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CROSS-REFERENCES

OBSTRUCTION OR PERFORATION

PATIENT CHARACTERISTICS

- Any age group; older age groups may have significant comorbidities.
- Systemically unwell with significant pain.
- Major fluid loss into the bowel or peritoneal cavity possible with peritonitis.
- Hypovolaemic with developing sepsis.
- Associated with underlying pathologies such as malignancy.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Look for comorbidities particularly of the cardiorespiratory system.
- Assess hydration and serum electrolytes.
- There may be evidence of septic/hypovolaemic shock.
- Full blood count and coagulation screen.
- Urea, electrolytes and creatinine.
- Cross-match blood according to local policy.

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- Arterial blood gases (metabolic acidosis/high lactate suggests poor tissue perfusion and the need for preoperative resuscitation).
- ECG and chest X-ray if indicated.

PREOPERATIVE PREPARATION

These patients usually require urgent, but not emergency surgery. Fully resuscitate prior to theatre. Emergency surgery may be needed if there is bowel ischaemia or perforation. Consider preoperative optimisation with invasive monitoring in the HDU. All patients require a nasogastric tube, urinary catheter and adequate analgesia. Consider thromboembolic prophylaxis.

MONITORING

- Routine anaesthetic monitoring.
- Core temperature.
- Invasive arterial and central venous pressure monitoring in all but the most straightforward of cases.
- Monitor cardiac output in high risk cases.

ANAESTHETIC TECHNIQUE

General anaesthesia is appropriate and can be supplemented with an epidural to provide postoperative analgesia. Regional anaesthesia, with a continuous spinal or epidural technique, can be used in selected cases. Excellent postoperative analgesia results but the following points should be considered:

- There may be a coagulopathy.
- Profound hypotension may result since patients are often hypovolaemic and may have severe sepsis.
- Placement of an epidural catheter in the presence of systemic sepsis may be inadvisable.

Establish large bore IV access and commence fluid administration prior to induction. Aspirate the nasogastric tube and leave it open to drain freely. Use a rapid sequence induction. There is potential for cardiovascular collapse on induction if the patient is inadequately resuscitated.

Choice of agents is wide but consider drugs with a rapid recovery profile since drug kinetics may be altered in patients who are unwell, particularly in the elderly. Avoid nitrous oxide since it can diffuse into an already dilated bowel.

Patients with an obstructed or perforated viscus may require very large fluid volumes intraoperatively, including blood transfusion. Fluid warmers and active body warming are mandatory. Patients may require inotropic or vasopressor support.

POSTOPERATIVE MANAGEMENT

All but the most straightforward cases should be managed in the HDU or ICU. Significant fluid loss and fluid shifts are likely to continue in the postoperative period, due to redistribution and sequestration. There is potential for deterioration, most commonly due to sepsis or respiratory complications.

Analgesia can be provided with epidural, opioid infusion or opioid PCA.

Care should be taken using NSAIDs if upper gastrointestinal tract perforation or renal compromise is present.

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CROSS-REFERENCES

Epidural, spinal CSE, Chapter 29
Preoperative risk assessment, Chapter 25

OESOPHAGECTOMY

Oesophagectomy is usually performed for malignant disease of the oesophagus and/or stomach. Several surgical approaches exist for oesophageal surgery, which are determined by the anatomical position of the tumour and by surgical preference:

- 'Ivor–Lewis' oesophagectomy: upper midline laparotomy followed by right thoracotomy (tumours in the middle and lower third of the oesophagus).
- Left thoracoabdominal oesophagectomy: tumours of the lower third and cardia.
- Three-stage oesophagectomy: upper midline laparotomy, right thoracotomy and left- or right-sided cervical incision (tumours of the upper and middle third).
- Pharyngo-laryngo-oesophagectomy: tumours of the hypopharynx and cervical oesophagus. Usually an ENT or multi-speciality procedure.
- Transhiatal oesophagectomy: oesophagectomy without thoracotomy – rarely used.
- Thoroscopic surgery is becoming more popular.

Most cases are either performed open, or by a combination of laparoscopy and thoracotomy. Some centres now practice a fully 'minimally invasive' technique – laparoscopy (head-up tilt), thoracoscopy/VATS (prone) and a neck incision for removal of the specimen and anastomosis.

PATIENT CHARACTERISTICS

- Elderly.
- High prevalence of tobacco and alcohol consumption.
- High incidence of ischaemic heart disease and COPD.
- May be malnourished secondary to dysphagia and malignancy. Surgery on malnourished patients carries a high morbidity and mortality.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Adequate staging of the tumour is essential.
- Assess and optimise comorbidities particularly of the cardiorespiratory systems.
- Assess nutritional status.
- Full blood count and coagulation if indicated.
- Urea, electrolytes and creatinine; liver function if indicated.
- ECG.
- Chest X-ray.
- Lung function tests and arterial blood gases if respiratory disease and one-lung-ventilation required.
- Echocardiography should be considered.
- Additional tests of global cardiorespiratory function, including the shuttle walk test or CPET.

PREOPERATIVE PREPARATION

Enteral or parenteral nutrition may be commenced preoperatively with possible addition of immuno-modulatory substances. Patients with poor respiratory function may benefit from admission to allow preoperative physiotherapy and treatment of infection. Thromboembolic prophylaxis is required.

PERIOPERATIVE MANAGEMENT

MONITORING

- Routine anaesthetic monitoring.
- Urinary catheter.
- Core temperature.
- Invasive arterial pressure.
- Central venous pressure. Insert lines ipsilateral to the side of the thoracotomy.
- Cardiac output in high-risk cases.

ANAESTHETIC TECHNIQUE

The most common technique is GA supplemented by epidural analgesia. Agents with rapid recovery profiles are useful to avoid prolonged postoperative respiratory depression. Unlike the volatile agents, propofol does not inhibit hypoxic pulmonary

vasoconstriction, and may therefore confer a small benefit. A left-sided double-lumen endobronchial tube or a single-lumen tube with an endobronchial blocker is inserted to allow one-lung ventilation, which may be prolonged. Active patient warming is required.

SPECIAL POINTS

With multilevel approaches to the oesophagus, such as the Ivor–Lewis operation, several intraoperative changes of patient position are required. This risks displacement of the double-lumen tube, loss of invasive lines and patient injury.

- Tube position needs to be checked after each change of position.
- Surgery can be prolonged and involve significant fluid and blood loss.
- Manipulation of mediastinal structures can impede cardiac filling and produce arrhythmias.

POSTOPERATIVE MANAGEMENT

Early extubation and admission to the HDU is undertaken if possible. Some cases will need ventilation in the ICU. Adequate analgesia is vital to avoid respiratory complications. Analgesia can be provided by an epidural, paravertebral catheter, intercostal nerve blocks or an opioid-based technique. Complete analgesia can sometimes be difficult to achieve because the surgery involves many dermatome levels. A multimodal approach is advisable.

- Aggressive physiotherapy.
- Thromboembolic prophylaxis.
- Early institution of enteral nutrition, often via a surgically sited jejunostomy, reduces the incidence of postoperative complications.
- A high index of suspicion is required for surgical complications, the most important of these being anastamotic breakdown.

OUTCOME

Oesophagectomy has an operative mortality of approximately 10%.

Outcome is improved by performing oesophagectomies in specialist centres with experienced teams.

COMPLICATIONS

Respiratory complications occur in 25% of patients.

Cardiovascular complications (including arrhythmias, especially atrial fibrillation [AF]) occur in approximately 10%, and thromboembolic complications in <10%.

Anastamotic leaks occur in approximately 10% and chylothorax in 3%. These have a high mortality if not aggressively managed. Early SIRS or sepsis, unexplained infiltrates on chest X-ray, etc. should always raise the possibility of an anastomotic leak.

One-year survival is approximately 50%, with a 5-year survival of 20%.

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OPEN AND LAPAROSCOPIC CHOLECYSTECTOMY

The incidence of gallstones is approximately 12% in men and 24% in women in the UK.

Standard treatment for symptomatic gallstones is now laparoscopic cholecystectomy. There are relatively

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few absolute contraindications, and most surgeons proceed with laparoscopic surgery even after acute cholecystitis, previous surgery and in patients with common bile duct stones. A minority of patients with impacted duct stones may benefit from ERCP and sphincterotomy.

The main indications for open cholecystectomy are

- Patients unfit to tolerate pneumoperitoneum
- Intraoperative conversion to an open technique
- Previous upper abdominal surgery (relative)
- Cholecystectomy performed at the same time as another open procedure

TYPICAL PATIENT CHARACTERISTICS

- Age 40–50 years
- Female:Male ratio 5:1
- History of hypercholesterolaemia or haemolytic disease
- Obesity

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Full blood count and electrolytes
- Liver function tests
- Chest X-ray and ECG where indicated
- Further investigations according to comorbidities
- In acute situations patients may be unwell, jaundiced and/or dehydrated
- Pay attention to renal function
- Correct hypovolaemia
- If coagulation is deranged consider 10 mg IV vitamin K ± fresh frozen plasma
- Antibiotics as per hospital protocol

ANAESTHETIC TECHNIQUE

- Routine standard monitoring
- Occasionally invasive monitoring dependant on any comorbidity or intercurrent condition
- Rapid sequence induction in the acute state
- Majority are performed electively

INDUCTION AND MAINTENANCE: GENERAL

- IV induction, muscle relaxation, tracheal intubation
- Air/oxygen with volatile agent, controlled ventilation
- Opioid, intravenous paracetamol and NSAID
- Antiemetic prophylaxis: 5HT3 inhibitor and dexamethasone

OPEN CHOLECYSTECTOMY

- Historically, morphine was avoided as it was thought to cause contraction of the sphincter of Oddi. This is not now considered relevant. Pethidine is an alternative and has smooth muscle relaxant properties. Hyoscine may occasionally be requested by the surgeon where duct exploration or intraoperative cholangiography proves difficult.
- Thoracic epidural, intercostal or field blocks.
- If a midline incision is employed, rectus sheath block may be an alternative.

LAPAROSCOPIC CHOLECYSTECTOMY

- TAP block and/or local infiltration to port sites.
- This can be a deceptive operation which can finish very suddenly – watch the screens and be aware of surgical progress!
- Be aware of potential complications (Table 10.2).

RECOVERY: EARLY

- Oxygen via face mask
- Intravenous fluid until the patient is drinking adequately
- Analgesia
  - Open procedures: opioid PCA, regional block, continuous or patient controlled epidural, paravertebral blocks or intercostal blocks
  - Laparoscopic procedure: paracetamol, codeine, NSAID

Table 10.2 Perioperative complications of laparoscopic cholecystectomy

- Cardiovascular collapse: raised intra-abdominal pressure leads to decreased venous return, increased systemic vascular resistance and reduced cardiac output. Further compromised by reverse Trendelenburg (head up) position.
- Gas embolism: sudden fall in $ETCO_2$, cardiovascular collapse, hypoxaemia. CO_2 is rapidly absorbed leading to resolution.
- Direct surgical trauma: vascular or visceral injury (increased risk after gas insufflation of stomach by IPPV, reduced by insertion of nasogastric tube).
- Dysrhythmias: bradycardia on inflation related to vagal stimulation from peritoneal stretch. Prophylactic anticholinergics recommended.
- Hypercarbia: ventilation can be difficult particularly in obese patients.
- Haemorrhage: difficult to access.
- Pneumothorax, mediastinal emphysema: presents with cardiovascular collapse.
- Hypothermia: due to large volumes of gas used. Monitor temperature and actively warm.
- PONV

RECOVERY: LATE

- Open procedure:
  - Complications secondary to poor pain control, obesity or immobility. Basal atelectasis, pneumonia DVT. Optimising pain control, physiotherapy, early mobilization and thromboembolic prophylaxis can reduce these.
  - Operative mortality is <1%. Factors increasing postoperative mortality are advancing age and acute surgery.
- Laparoscopic procedure:
  - Delayed recovery due to conversion to open technique or bile duct injury.
  - Mortality rate <0.4%.

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CROSS-REFERENCES

Day case surgery, Chapter 25
Truncal blocks, Chapter 29
Epidural, spinal, CSE, Chapter 29

PANCREATIC SURGERY

Indications:

- Necrotising acute pancreatitis
- Chronic pancreatitis
- Trauma
- Neoplasia

ACUTE PANCREATITIS

Conservative management is the first line, centred on supportive management with ICU support. Serial radiological imaging of the pancreas, including CT guided drainage of any collection. Antibiotics are reserved for proven infection.

Surgery is reserved for cases of 'failed' conservative management. The main indication is necrotising pancreatitis – necrosectomy plus lavage. This procedure has reduced the mortality to 24%.

Minimally invasive radiological assisted necrosectomy has been described with good results. Surgery (usually laparoscopic) may also be required for complications – persistent pseudocyst, pancreatic abscess and haemorrhage. Epidural analgesia is an excellent method of pain control in the acute episode.

TYPICAL PATIENT CHARACTERISTICS

- Age 40–70 years
- History of gallstones and associated risk factors
- Alcohol
- Trauma
- 40% idiopathic
- Drugs: ACE inhibitors, thiazide diuretics, steroids
- Lipid abnormalities
- Hypercalcaemia

PRESENTATION

- Acutely unwell complaining of epigastric pain.
- Dehydrated and often jaundiced.
- Pleural effusions and acute lung injury can develop.
- Septic shock and multi-organ failure are indicators of necrotising pancreatitis developing.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Full history and examination (patient may be in ICU).
- Full blood count, electrolytes, liver function tests, coagulation screen, arterial blood gases.
- Chest X-ray and ECG.
- Additional investigations according to coexisting medical conditions.
- If coagulation is deranged, consider IV vitamin K. Fresh frozen plasma (FFP) is reserved for cases of active bleeding.
- Hypovolaemia should be fully corrected.

ANAESTHETIC TECHNIQUE

PREINDUCTION

- Routine monitoring
- Arterial line, central venous line, cardiac output
- Rapid sequence induction if not intubated already (gastric stasis likely)

INDUCTION AND MAINTENANCE

- Thoracic epidural where coagulation permits.
- IV induction, muscle relaxation, tracheal intubation.

- Insert nasogastric tube if not already present.
- Air/oxygen mix with a volatile agent, controlled ventilation.
- Opioid analgesic or epidural.
- Extubate or transfer to ICU.

RECOVERY

- HDU or ICU.
- Supplemental oxygen.
- Surgery causes release of inflammatory mediators which can lead to initial clinical deterioration.
- Start enteral feeding early via a naso-jejunal feeding tube.
- Postoperative complications include colonic necrosis, fistula formation and bleeding.
- Respiratory and renal complications are common.

CHRONIC PANCREATITIS

- Increasing in incidence: 27.4 cases per million.
- Indications for surgery are
  - Palliation of pain
  - Exclude carcinoma
  - Bypass or remove the complications of the disease
- Endoscopic techniques are increasingly being used as an alternative to traditional pancreaticojejuonstomy.

TYPICAL PATIENT CHARACTERISTICS

- Alcohol: 60%–70% of cases in western world
- Tropical pancreatitis
- Obstructive pancreatitis
- Hypercalcaemia
- Hereditary
- Biliary tract disease
- Idiopathic

PRESENTATION

- Chronic abdominal pain radiating to back.
- Weight loss due to endocrine and exocrine insufficiency.
- Diabetes and associated complications.
- Jaundice is rare.

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PREOPERATIVE ASSESSMENT, INVESTIGATION, OPTIMISATION

- History and examination
- Full blood count and coagulation screen
- Electrolytes, liver function tests, blood glucose
- Chest X-ray and ECG
- Further investigations according to coexisting medical conditions
- Record alcohol intake and treatment withdrawal if needed
- Stabilise diabetes and convert to a sliding scale or infusion
- Consider multivitamins and vitamin K

PANCREATIC NEOPLASIA

Pancreatic neoplasms comprise a spectrum of exocrine and endocrine tumours, the majority being malignant. Two-thirds arise in the head of the pancreas. The incidence is 10 per million, with 10% 1-year survival. Surgery is by Whipples pancreaticoduodenectomy or a variant of this.

TYPICAL PATIENT CHARACTERISTICS

- Male:female ratio 1.25:1
- Smokers
- Occupational exposure to β-naphthylene and benzidine
- Hereditary
- Diabetes mellitus
- Chronic pancreatitis
- Gastrectomy

PRESENTATION

- Obstructive jaundice, weight loss, abdominal pain
- Anorexia, nausea, vomiting
- Impaired glucose tolerance

PREOPERATIVE ASSESSMENT, INVESTIGATION, OPTIMISATION

- Full blood count and coagulation screen
- Electrolytes, liver function tests
- Chest X-ray and ECG
- Further investigation according to coexisting medical conditions
- Correct hypovolaemia
- Stabilise diabetes and commence a sliding scale or infusion
- Remember that endocrine tumours are often part of a more complex syndrome
- Preoperative stenting to relieve jaundice is common, but consider vitamin K 10 mg where the liver function tests remain deranged, even in the presence of normal coagulation

ANAESTHETIC TECHNIQUE FOR WHIPPLE'S PANCREATICODUODENECTOMY

Classically performed as an open procedure. In some specialist centres may be performed laparoscopically in suitable patients. The anaesthetic technique for the laparoscopic variant is essentially similar to that for the open procedure, with the additional consideration of the effects of pneumoperitoneum. The position adopted involves steep lateral tilt and head-up tilt with the real possibility of haemodynamic compromise. A prolonged procedure should be expected.

INDUCTION AND MAINTENANCE

- Routine monitoring
- Arterial line, central venous line, cardiac output
- Thoracic epidural if coagulation is satisfactory
- IV induction, muscle relaxant, tracheal intubation
- Air/oxygen mix with volatile agent, controlled ventilation
- Epidural preferable, but if not possible, a remifentanil infusion is effective

INTRAOPERATIVE COMPLICATIONS

- *Hypothermia* – Active warming – fluid and blankets
- *Hypovolaemia* – Large fluid losses plus potential large blood losses – maintain central venous pressure and monitor haematocrit, transfuse as necessary

- *Hyperkalaemia* – Surgical manipulation leading to portal vein or hepatic artery obstruction and hepatic ischaemia and intracellular potassium leak – monitor potassium
- *Hypo/hyperglycaemia* – Monitor closely, in insulinoma constant infusion of dextrose infusion has been shown to reduce rebound hyperglycaemia
- *Hypoxia* – Basal atelectasis or development of acute lung injury – increase FiO_2 add PEEP – consider facial CPAP or continued ventilation postoperatively
- *Renal dysfunction* – Hepatorenal syndrome-monitor renal output, maintain circulating volume and mean blood pressure, consider mannitol or a loop diuretic

RECOVERY: EARLY

- Oxygen via face mask
- HDU care
- IV fluid then early introduction of enteral (jejunal) feed
- Analgesia: epidural, IV paracetamol, PCA or opioid infusion
- DVT prophylaxis
- Monitor blood glucose: manipulation with insulin and dextrose

RECOVERY: LATE

- Morbidity up to 40%; mortality 5%
- Common complications are basal atelectasis, pneumonia, DVT, anastomotic leak
- Optimise pain control, physiotherapy, early mobilization and thromboembolic prophylaxis
- Incidence of postoperative diabetes is dependent on size of pancreatic resection but is quoted at 50%

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CROSS-REFERENCES

Prolonged anaesthesia, Chapter 28
Diabetes mellitus, Chapter 6
The jaundiced patient, Chapter 4

PHAEOCHROMOCYTOMA

Catecholamine secreting tumours of chromaffin tissue.

- Usually benign; may be malignant, solitary or multiple.
- Most common within the adrenal gland, but can exist in paraganglionic sites base of the bladder to the base of skull.
- About 13% are associated with multiple endocrine neoplasia (MEN) type 2, either 2A associated with thyroid carcinoma and parathyroid hyperplasia or more rarely 2B associated with Marfan like features and mucosal neuroma.
- Associations with neurofibromatosis and von Hippel Lindau syndrome exist.
- 10% are extra-adrenal, multiple, bilateral or malignant.
- Occasionally, they develop in childhood, more boys than girls and are more often in extra adrenal sites.

PATIENTS

Common presentations include:

- Hypertension: may be paroxysmal (50%) or continuous.
- Headache.
- Sweating.
- Cardiovascular problems ranging from arrhythmias to catecholamine cardiomyopathy.
- Headache, palpitations, sweating and hypertension are 90% predictive of phaeochromocytoma.

- Hyperglycaemia and glycosuria; some patients may require insulin.
- Ectopic tumours in the neck may be provoked by head movement, and those at the base of the bladder by micturition.

Presentation depends upon the relative proportion of epinephrine and norepinephrine. Predominantly norepinephrine-secreting tumours present with hypertension, headache and pallor, while epinephrine-secreting tumours present with tachycardia and arrhythmias. Some patients develop a hypermetabolic state similar to that seen in severe hyperthyroidism.

Diagnosis includes 24 hour or overnight urine collection for met-epinephrine and nor-met-epinephrine. Plasma catecholamines are less reliable. Tumour is localised by CT, MR and ^{131}I *m*-iodobenzylguanidine (MIGB) scans. Radiological contrast media may precipitate a crisis. Genetic analysis for abnormal RET oncogene may be performed.

SURGICAL PROCEDURE

The usual surgical approach is laparoscopic resection of the adrenal gland. Extra-adrenal tumours may require a variety of surgical approaches.

PREOPERATIVE MANAGEMENT

Multisystem preoperative assessment is necessary, with special emphasis on the cardiovascular system. All patients should have echocardiography, since cardiomyopathy is often symptomless and unsuspected.

PREOPERATIVE BLOCKADE

Advice is varied but a reduction in mortality from 50% to 0%–3% was coincident with the introduction of preoperative alpha and beta blockade. Both selective and nonselective alpha blockers have been used. This does not prevent the release of catecholamines but obtunds the physiological response.

Published recommendations range from nothing to prolonged alpha +/- beta blockade. Calcium channel blockers, magnesium and metyrosine may also have a role.

RECOMMENDATIONS FOR PREOPERATIVE BLOCKADE

Alpha blockade using phenoxybenzamine (commencing at 10 mg orally twice daily) titrated to BP. Increase dosage until symptoms abate and the BP and HR are controlled as assessed on 24-hour recording; maximum BP 140/90 and HR 100 bpm. This allows restoration of normal circulating volume, and in patients with cardiomyopathy a period of cardiac rest which may allow some recovery.

Side-effects include:

- Stuffy nose
- Postural hypotension (initial treatment as an in-patient)
- Drowsiness and blurred vision (the patient should not drive)
- Tachycardia: control with a beta blocker (propranolol or atenolol) introduced after alpha blockade is established.

At least 7–10 days of established blockade is required, but many patients benefit from a longer period of treatment, particularly those with cardiomyopathy or ST–T wave changes. Plasma volume may increase thus preventing severe hypotension after tumour removal.

PREMEDICATION

- An anxiolytic is useful.
- Avoid atropine because of tachycardia.
- DVT prophylaxis.
- Discontinue blockade on the evening before surgery.

PERIOPERATIVE CARE

- Sodium nitroprusside infusion for control of hypertensive surges.
- Norepinephrine for postresection hypotension.
- Epinephrine in case of decreased cardiac output associated with abrupt fall in circulating catecholamines.
- Bolus doses of propranolol for control of tachycardia.

- Maintenance of temperature in a vasodilated patient.
- Warming: IV fluids, warming blanket, forced air warmer, humidify inspired gases.

MONITORING

- Routine minimal monitoring
- Arterial line
- CVP line
- Cardiac output (e.g. oesophageal Doppler)
- Core temperature

ANAESTHETIC TECHNIQUE

Thoracic epidural block plus general anaesthesia is recommended. Since these patients already have peripheral vasodilation, it is most unusual for the epidural to cause hypotension. For laparoscopic surgery, general anaesthesia alone is sufficient. Avoid histamine-releasing drugs (e.g. morphine, atracurium). Most anaesthetic drugs have been used successfully.

CONTROL OF ARTERIAL PRESSURE AND HEART RATE

Marked swings in blood pressure and heart rate may be seen during manipulation of the tumour. Control with nitroprusside and a short-acting beta blocker.

Alpha blockade leads to increased plasma volume, but even then it is usually necessary to administer additional IV fluids during the dissection phase if hypotension following tumour devascularization is to be prevented.

After ligation of all venous drainage from the tumour, arterial pressure commonly declines. This may be associated with a high cardiac output and very low systemic vascular resistance, requiring norepinephrine infusion, low cardiac output requiring inotropic support or both. An adequate period of preoperative blockade, combined with generous intraoperative fluid loading may obviate the need for postresection catecholamines.

POSTOPERATIVE CARE

Transfer to HDU or ICU.

BLOOD PRESSURE

Arterial pressure usually stabilizes remarkably quickly, and vasoactive medication can be rapidly withdrawn. Occasionally hypertension persists for several days and in a small minority it may persist indicating residual tumour or underlying essential hypertension.

BLOOD GLUCOSE

High catecholamine levels increase blood glucose and block insulin secretion. Tumour removal and loss of catecholamines may result in hypoglycaemia. Persisting beta blockade will mask the signs of hypoglycaemia. Monitor blood glucose every 30 minutes for the first 6 h and correct promptly.

STEROIDS

If bilateral adrenalectomy has been carried out, steroid replacement will be required.

ANALGESIA

Abrupt withdrawal of catecholamines and residual concentrations of blocking drugs may cause marked somnolence. Patients may be sensitive to the sedative actions of opioids; conventional doses may cause dangerously deep sedation. Patient-controlled epidural analgesia avoids this problem.

UNEXPECTEDLY ENCOUNTERED PHAEOCHROMOCYTOMA

Rarely, a patient will exhibit severe hypertension with a tachycardia and possibly arrhythmias and pulmonary oedema during surgery for an unrelated condition. If an undiagnosed phaeochromocytoma is suspected, commence alpha blockade with phentolamine. Surgery should be completed as rapidly as possible and no attempt made to resect the tumour unless this is essential to the patient's immediate survival. Then, diagnosis, localization and preparation may be undertaken as usual.

Acute nonoperative presentation may include hypotension as heart failure supervenes; in these circumstances diagnosis may be difficult.

Presentation in pregnancy is associated with increased risks to both mother and foetus; both early intervention and conservative management until delivery has been reported.

OUTCOME

- In adequately prepared patients mortality is 0%–3%.
- Mortality is up to 50% in patients diagnosed during operation.

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CROSS-REFERENCES

Iatrogenic adrenocortical insufficiency, Chapter 6
Multiple endocrine neoplasia, Chapter 6
Hypertension, Chapter 2
Prolonged anaesthesia, Chapter 28
Intraoperative hypertension, Chapter 30

Gynaecological surgery

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FERTILITY SURGERY

**AMY HOBBS, SOPHIE KIMBER CRAIG
AND PATRICK ROSS**

There are many different procedures performed for fertility assessment, treatment and management, including:

- Gamete/oocyte/embryo transfer
- Oocyte/sperm retrieval
- Donor insemination
- Oocyte donation
- Techniques for people with cancer wishing to preserve fertility

Some investigative procedures looking for the cause of female infertility will involve laparoscopic or minor gynaecological procedures (e.g. tubal surgery or ablation of endometriosis).

Most fertility procedures are carried out as day cases under sedation. There has been a move away from laparoscopic procedures towards transvaginal ultrasound-guided egg retrieval.

PREOPERATIVE

Women are of childbearing age, ASA 1 or 2, and rarely have conditions associated with advancing age. They should have had their thyroid function checked during preparation for treatment.

Fertility treatment should be recognised as being stressful for the woman and her partner. Many women are anxious and may have additional psychosocial issues relating to their infertility adding to their concerns about the procedure. This will also often be a repeat procedure for many women. It is very important, therefore, to establish expectations and work to allay any fears she has about the process and the management of her pain.

INTRAOPERATIVE

- Monitoring according to AAGBI standards
- IV access
- Position in lithotomy or Lloyd–Davies
- Day case procedure
- Paracetamol for analgesia
- Antiemetics

Most cases are performed under sedation due partly to concerns about the drugs used in GA and the length of the exposure time which might have a detrimental effect on subsequent success rates.

Good analgesia and sedation are likely to reduce the time taken for the procedure as this will prevent patient movement, which makes the technical process easier for the surgeon. The following agents can all be used, often in combination:

- Propofol (bolus or target-controlled infusion)
- Midazolam
- Remifentanil infusion
- Alfentanil or fentanyl
- Ketamine

Most of these agents can be detected in the follicular fluid but their influence on the success rates of the fertility treatment has not been shown to be deleterious. Ketamine has a number of unpleasant side effects which, particularly in the day case setting, make it the least appealing choice.

Spinal blocks can be used as an alternative to sedation techniques. The use of short-acting agents (e.g. lidocaine, prilocaine) will mean a quicker return to normal function for the woman postprocedure. Paracervical block can also be used to provide anaesthesia locally. This should be combined with sedation to ensure better patient comfort throughout.

POSTOPERATIVE

Ensure adequate analgesia (paracetamol ± weak opioids). Avoid NSAIDs. Provide appropriate antiemetic treatment.

REFERENCE

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CROSS-REFERENCES

HYSTERECTOMY

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Hysterectomy is a common procedure which may be total (cervix removed) or subtotal (cervix preserved). It may be combined with salpingo-oophorectomy. Radical hysterectomy also includes removal of part of the vagina and pelvic lymph nodes. Indications for hysterectomy are malignancy, fibroids, endometriosis and prolapse.

The procedure may be approached abdominally, vaginally or laparoscopically. Vaginal hysterectomy is associated with the best outcomes although some cases are not suitable for this approach. Patients recover quicker after laparoscopic hysterectomy compared to abdominal hysterectomy but the former is associated with a higher risk of urinary tract injury.

PREOPERATIVE ASSESSMENT

- Common presentation at age 40–50 years.
- A range of comorbidities is possible: thorough preoperative assessment is required.
- Anaemia is often present. Optimise preoperatively. Group and save blood.
- A bulky uterus or mass may compress ureters resulting in renal dysfunction.
- Patients with endometrial cancer may have received chemotherapy. Look for neutropaenia and thrombocytopaenia.
- Anxiolysis may be required.
- Compression stockings should be worn unless contraindicated.

INTRAOPERATIVE MANAGEMENT

- Routine AAGBI minimal monitoring.
- Invasive monitoring in selected patients or if complex surgery is planned.
- Consider cardiac output monitoring in complex cases.
- PONV is common so appropriate anti-emetics must be provided.
- Mechanical prophylaxis against VTE.

- Maintain body temperature.
- Antibiotic prophylaxis according to hospital guidelines.

VAGINAL HYSTERECTOMY

This approach requires the lithotomy position so precautions should be taken to avoid nerve injuries and in positioning.

Either GA or neuraxial block may be employed. If GA is used, patients may breathe spontaneously or be mechanically ventilated but anticipate potential respiratory changes when the patient is moved into the Trendelenberg position. Multimodal analgesia with paracetamol, NSAIDs, opiate and local anaesthetic infiltration should be provided. Some surgeons use local with adrenaline to reduce bleeding. Vigilance is required as adrenaline may be absorbed systemically, causing arrhythmias, tachycardia and hypertension.

If neuraxial block is used, adding intrathecal opiate will aid postoperative analgesia. A block height to at least T8 is required. It is important not to position the patient for surgery too soon after siting the block as both lithotomy position and Trendelenberg position will increase cephalad spread. Hypotension should be managed with vasopressors. Some, but not all, patients may require supplemental sedation.

Postoperative pain is less than with abdominal hysterectomy. Paracetamol, NSAIDs and oral opiates, antiemetics and VTE prophylaxis should be prescribed.

ABDOMINAL HYSTERECTOMY

Surgery will be performed with the patient in the Trendelenberg position, which may be steep. A transverse Pfannenstiel incision is the common approach but occasionally a midline incision is used.

Either GA or neuraxial block may be employed. If GA is used, muscle relaxation and controlled ventilation is usually required. Trendelenberg positioning will cause diaphragmatic splinting; therefore, adjustment of ventilation parameters may be required. Multimodal analgesia with paracetamol, NSAIDs, opiate and local blocks should be provided. Some centres use epidural analgesia for the peri- and postoperative periods. A transversus abdominis plane (TAP) block may reduce opiate requirements, at least in the first 24 hours, and this should be considered for patients with transverse incisions. Rectus sheath block provides analgesia for midline incisions. Some hospitals offer continuous local analgesic infusions.

If neuraxial block is used, intrathecal opiate should be added. A block height to T4 is required. Hypotension is common and should be addressed. Positioning patients in the Trendelenberg position too soon after siting the block may result in significant cephalad spread.

Blood loss is usually moderate but transfusion is occasionally required, particularly in those with preoperative anaemia.

Pain can be severe but is reduced in those who have had a block. Gabapentin can provide good analgesia in addition to paracetamol, NSAIDs and simple opiates. Patient controlled analgesia is often required. Antiemetics and VTE prophylaxis should also be prescribed.

LAPAROSCOPIC HYSTERECTOMY

The patient will be in lithotomy position with steep Trendelenberg, so complications of both must be expected. Additionally, the risks of pneumoperitoneum must be considered. Large bore venous access is recommended in anticipation of any intraoperative cardiovascular complications or intraperitoneal injury.

A uterine manipulator is usually inserted through the cervix, which may provoke profound vagal stimulation and bradycardia. The uterus may be removed vaginally (laparoscopically assisted vaginal hysterectomy) or through an abdominal incision (total laparoscopic hysterectomy).

GA is required in almost every case. If GA is deemed too high risk, the surgical approach should be discussed with the surgeon and patient. Open surgery performed under neuraxial block is possible. Muscle relaxation, tracheal intubation and controlled ventilation are essential. Frequent adjustments to parameters should be made to ensure adequate ventilation, as the effects of pneumoperitoneum and steep Trendelenberg positioning will alter lung mechanics and carbon dioxide can accumulate from insufflation.

Multimodal analgesia with paracetamol, NSAIDs, opiate and local infiltration should be provided.

Evidence suggests that TAP blocks do not provide analgesic benefit after laparoscopic hysterectomy.

Blood loss is usually moderate but transfusion is occasionally required, particularly in those with pre-operative anaemia.

Postoperative pain is less than with abdominal hysterectomy and is usually moderate. Shoulder tip pain is common. Paracetamol, NSAIDs and oral opiates, antiemetics and VTE prophylaxis should be prescribed.

RECOVERY

Enhanced recovery pathways are used at many hospitals for hysterectomy, allowing consistency in perioperative care and thorough physiological and psychological patient preparation. Length of stay is reduced and patient satisfaction improved without increasing readmission rates. Common components include:

- Preoperative patient counselling and optimisation of comorbidities
- Use of nerve blocks and PCA in preference to epidural analgesia
- Use of cardiac output monitoring to guide intraoperative fluid management
- Minimising use of drains and nasogastric tubes
- Early commencement of oral diet postoperatively
- Early removal of urinary catheter postoperatively
- Appropriate use of patient warming, antiemetics and venous thromboembolism prophylaxis

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CROSS-REFERENCES

HYSTEROSCOPY AND LASER SURGERY

AMY HOBBS AND SOPHIE KIMBER CRAIG

Hysteroscopy allows direct visualisation of the uterine cavity, tubal ostia, cervix and vagina. The most common indication is abnormal bleeding. Simple hysteroscopy is an outpatient procedure not requiring an anaesthetic.

Hysteroscopy is often combined with a therapeutic procedure, such as endometrial ablation, myomectomy, dilatation and curettage (D&C), or sterilisation. Many patients are treated as day cases.

Recognised risks include vagal stimulation, uterine perforation and haemorrhage. Other risks depend on the nature of the surgery.

DISTENDING MEDIUM

A distending medium is required to view the operating field (Table 11.1). Irrigating fluid is most common as it allows debris and blood to be washed away.

Intracervical injection of 8 mL of dilute vasopressin solution (0.05 U/mL) has been shown to reduce distending media absorption during resectoscopic surgery but is rarely used.

Table 11.1 Hysteroscopic distending media

Distending medium	Procedure type	Complications	Thresholds
Carbon dioxide	Diagnostic	Gas embolism	Flow 40–60 mL/min
		Hypercapnoea	Maximum pressure 100 mmHg
		Shoulder tip pain	
0.9% Sodium chloride	Diagnostic, bipolar diathermy, morcellation, laser	Fluid overload	Plan completion at 750 mL fluid deficit
			Maximum fluid deficit 2500 mL
Compound sodium lactate	Diagnostic, bipolar diathermy, morcellation, laser	Fluid overload	Plan completion at 750 mL fluid deficit
			Maximum fluid deficit 2500 mL
5% Mannitol	Monopolar diathermy	Fluid overload	Plan completion at 750 mL fluid deficit
		Hyponatraemia	
			Maximum fluid deficit 2500 mL
3% Sorbitol	Monopolar diathermy	Fluid overload	Plan completion at 750 mL fluid deficit
		Hypotonic hyponatraemia with TUR syndrome	Maximum fluid deficit 1000 mL
1.5% Glycine	Monopolar diathermy	Fluid overload	Plan completion at 750 mL fluid deficit
		Hypotonic hyponatraemia with TUR syndrome	Maximum fluid deficit 1000 mL

DIATHERMY RESECTION OF FIBROIDS

Use of diathermy has been associated with thermal burns, causing damage to organs adjacent to the surgical site or where the neutral plate has been improperly applied.

Avoid diathermy if possible in patients with pacemakers as it can inhibit or permanently damage them. Bipolar diathermy should be used whenever possible.

MORCELLATION OF FIBROIDS

The morcellator finely cuts up fibroids before removal by suction. Although quicker than other methods of myomectomy, it has been associated with bowel injury and is not currently recommended by NICE.

LASER ABLATION OF FIBROIDS

Laser is used less commonly than other thermal ablative treatments in modern gynaecology practice. Safe use of lasers in theatres requires strict adherence to

rules established by the MHRA. Specific safety features include:

- Eye protection for patient and all staff
- Theatre entrances locked, windows shut and safety signs displayed
- Reflective surfaces covered
- Availability of appropriate non-water-based fire extinguisher

PREOPERATIVE

- Patients of any age and ASA grade.
- Anaemia is common; perform full blood count with other tests as per hospital guidelines.
- Some patients presenting for hysteroscopy under anaesthetic have been unable to tolerate the procedure as an outpatient and may be very anxious, requiring anxiolytic premedication.

INTRAOPERATIVE

Regional or general anaesthesia may be used depending on patient and surgical factors. If prolonged

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surgery is predicted, with significant irrigation volumes, regional anaesthetic may be preferred to allow early identification of TUR syndrome. Intrathecal opiate is rarely required and the use of prilocaine instead of marcaine may be considered to allow earlier discharge.

Standard AAGBI monitoring, with additional invasive monitoring in high risk patients.

Careful monitoring of fluid balance is essential, including calculation of the deficit between irrigated and collected fluid. Guidelines for maximum deficits are shown in Table 11.1.

Maintain normothermia.

Lithotomy, with or without Trendelenberg, is usually required. Take appropriate care with positioning and precautions against associated risks.

At least one antiemetic should be given intraoperatively.

DVT prophylaxis with compression stockings and/or pneumatic compression devices is required.

POSTOPERATIVE

Postoperative pain is usually mild to moderate – paracetamol and NSAIDs are usually adequate.

PONV is common and antiemetics should be prescribed.

Mechanical and/or pharmacological prophylaxis against VTE should be prescribed for the postoperative period.

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CROSS-REFERENCES

LAPAROSCOPY

AMY HOBBS AND SOPHIE KIMBER CRAIG

Laparoscopy may be required for diagnostic (e.g. investigation of pain, dye tests) or therapeutic (e.g. tubal ligation) procedures. Patients are usually relatively fit and can often be managed as day cases.

The initial trocar is not inserted under direct vision. Trocar damage to peritoneal structures, including the aorta and vena cava, has been reported. Carbon dioxide is initially insufflated to achieve a pressure of 20–25 mmHg, but the distension pressure should be reduced to 12–15 mmHg once all ports are placed.

Unique to gynaecology is the uterine cannula, inserted through the cervix, allowing manipulation of the uterus and injection of dye. This may provoke vagal stimulation.

PREOPERATIVE

- Full blood count is often required, with other tests only as clinically indicated.
- Many patients are nervous and an anxiolyic may be appropriate.

INTRAOPERATIVE

- GA is the usual technique. If a neuraxial block is used, a block height up to T4 is required.
- Routine monitoring to AAGBI recommendations with additional invasive monitoring in high-risk patients.
- Large bore IV access due to the potential for cardiovascular instability and risks of damage to intraperitoneal structures.
- Maintain normothermia.
- Lithotomy or Lloyd–Davies position with Trendelenberg.
- Tracheal intubation is recommended. Several studies have demonstrated success with second generation laryngeal masks but only in the fittest, lowest risk women.
- Short or intermediate acting induction agent and muscle relaxant is appropriate.

- Maintain anaesthesia with a volatile agent or TIVA.
- Multi-modal analgesia with paracetamol, NSAIDs and local anaesthetic should be employed. Long acting opioids may be required if significant visceral pain is anticipated.
- Antiemetics should be given.
- Use DVT prophylaxis with compression stockings and/or pneumatic compression devices.
- Laparoscopy may pose particular risks to patients, including:
  - Aorta and vena cava compression
  - Hypercapnoea
  - Diaphragmatic splinting
  - Splanchnic hypoperfusion
  - Raised intracranial pressure and cerebral oedema
  - Parasympathetic stimulation (augmented by cervical dilatation), causing profound bradycardia and rarely asystole
  - Gas embolism

POSTOPERATIVE

- Pain usually controlled by simple analgesics but some patients require opiate analgesia.
- Shoulder tip pain, due to irritation of the diaphragm by carbon dioxide, is common and patients should be warned about this in advance. The incidence is reduced if as much gas as possible is removed from the peritoneal cavity. Peritoneal inflammation can persist for up to three days.
- PONV can be expected in up to 50%. Rescue antiemetics must be prescribed.
- Mechanical and/or pharmacological prophylaxis against VTE should be prescribed for the postoperative period.

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CROSS-REFERENCES

MINOR GYNAECOLOGY PROCEDURES (ELECTIVE AND URGENT)

AMY HOBBS AND SOPHIE KIMBER CRAIG

These are usually day-case procedures and include:

- Endometrial biopsy
- Dilatation and curettage
- Termination of pregnancy
- Minor vulval surgery (e.g. incision and drainage or marsupialisation of Bartholin's cyst, biopsy)

Surgery involving the uterus requires cervical dilatation. The indications are varied, but include investigation or management of vaginal bleeding. They all require lithotomy positioning.

PREOPERATIVE

- Women of child-bearing age through to postmenopause.
- Patients of ASA 1 or 2 may be managed as day-case procedures; those with significant comorbidities may need an overnight stay.
- Perform a full preoperative assessment.
- Full blood count (anaemia is common) with additional tests as per hospital policy.
- Patients may be receiving iron therapy or tranexamic acid.

- Rhesus status should be known for all cases where pregnancy (or pregnancy loss) is involved.
- The oral contraceptive pill is normally continued perioperatively as there is a higher risk of VTE due to an unplanned pregnancy than from a day-case procedure, but HRT is usually stopped.
- A negative pregnancy test is required in those where there is a possibility of pregnancy (except in the case of a termination). Ensure the woman has no increased risk factors for bleeding.
- It is important to recognise the emotional impact of certain procedures, particularly termination of pregnancy and dilatation and curettage following miscarriage. Occasionally sedation may be required for the very anxious patient.
- Ensure there are no issues with the required position for surgery.
- Emergency surgery (such as bleeding after miscarriage), requires:
    - Full assessment of cardiovascular status
    - Fluid resuscitation if evidence of hypovolaemia exists
    - Preparation for managing blood loss (group and save or cross-match, as required)
    - Possible rapid transfer to the operating theatre for control of blood loss
- A risk of sepsis exists in patients where there has been prolonged vaginal bleeding or missed miscarriage. Resuscitation may be needed and antibiotics given as per hospital protocol.

INTRAOPERATIVE

- General or regional anaesthesia can be used (facemask alone is sometimes used in very short cases).
- Avoid using nitrous oxide due to the high risk of PONV; TIVA can be employed.
- Sedation and/or paracervical block may be requested, the choice depending on the surgeon's or patient's wishes and expectations.

- A spinal is possible but use short-acting agents to facilitate early discharge from the day-case unit.
- In the event of an emergency situation with heavy blood loss, ketamine can be considered for maintaining cardiovascular stability during induction.
- Administer analgesics and antiemetics intraoperatively. Fentanyl at induction will often provide enough opioid analgesia into the postoperative period without the need for long-acting opioids.
- Care should be taken with the lithotomy position, to prevent common peroneal injury from the stirrups.
- Cervical dilation risks vagal stimulation and can on occasions be profound enough to cause asystole; antimuscarinics must be available in advance. In the event of profound bradycardia, request that the surgeon stop immediately. This is more commonly seen in postmenopausal women and those who have had previous cervical surgery (as the cervix may be more stenotic).
- Ensure perioperative VTE prophylaxis is considered.

POSTOPERATIVE

- Aim for restoration of normality and discharge fitness as soon as possible.
- Prescribe antiemetics.
- The pain experienced after uterine procedures is comparable with significant period pain. NSAIDs have been shown to be effective. Combined with paracetamol this should provide adequate pain relief. Occasionally opioids are required.
- Uterine perforation is the most common serious immediate complication. If there is pain in excess of that which is normally expected, this diagnosis should be considered and reviewed by the obstetric team.

CROSS-REFERENCES

Spinal, epidural, CSE, Chapter 29
Day case surgery, Chapter 25

PELVIC FLOOR SURGERY

AMY HOBBS AND SOPHIE KIMBER CRAIG

Pelvic organ prolapse may involve uterus, vagina, bladder and/or bowel. It may occur in up to 50% of parous women. Other risk factors include connective tissue disorders, chronic cough, fibroids, patients postmenopause and following hysterectomy.

The surgical approach and technique depend on the organs involved and patient factors. Higher risk surgery, with lower rates of relapse, may be recommended in younger patients. If there is significant prolapse of the uterus, a hysterectomy may be required in addition to prolapse repair.

It is important to identify whether surgery will be performed via a vaginal, abdominal or laparoscopic approach and whether a mesh will be used.

PREOPERATIVE

Most patients are over 60 years old. Those with connective tissue disorders such as Ehlers–Danlos syndrome or Marfan syndrome may present at a younger age. Patients may have a range of comorbidities so a thorough preoperative assessment is required. Chronic cough is a risk factor and chronic lung disease is common in this population. In particular:

- In post-hysterectomy patients following malignancy, ascertain if there are any ongoing concerns related to the malignancy.
- In patients with high grades of prolapse or large fibroids, patients may have obstructive renal impairment.

Preoperative investigations should follow NICE recommendations. Patients having concomitant hysterectomy or other significant surgery should have a group and save sample sent.

Many patients are treated as day cases. Simple cystocele repairs may be performed under local anaesthetic.

INTRAOPERATIVE

If surgery is via the vaginal or abdominal approach, regional or general anaesthesia may be used, depending on patient comorbidities and preference. For laparoscopic procedures, general anaesthetic is almost always required, with intubation of the trachea and mechanical ventilation.

Routine monitoring as per AAGBI recommendations with additional invasive monitoring as required.

The Trendelenburg position is usually required with lithotomy for vaginal approaches. Take appropriate care with positioning and precaution against associated risks.

Antibiotics are usually recommended if a mesh is used; follow hospital protocols.

Paracetamol, NSAIDs and infiltration of local anaesthetic by the surgeon is recommended for analgesia.

At least one antiemetic should be given intraoperatively.

Mechanical VTE prophylaxis should be provided.

Blood transfusion is occasionally required in complex surgery or in those with pre-existing anaemia.

POSTOPERATIVE

Pain is usually mild to moderate and more significant following open abdominal procedures. Avoid constipation in the postoperative period, as straining will be detrimental to the postoperative recovery. Therefore, if opiates are required, regular laxatives should also be prescribed.

PONV is common and antiemetics should be prescribed.

Mechanical and/or pharmacological prophylaxis against VTE should be prescribed.

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CROSS-REFERENCES

Complications of position, Chapter 30
The elderly patient, Chapter 25

RADICAL GYNAECOLOGICAL CANCER SURGERY

AMY HOBBS AND SOPHIE KIMBER CRAIG

Uterine (endometrial) and ovarian cancer are the fourth and sixth most common cancers in women and cervical cancer is also in the top 20. Vaginal and vulval cancers are far less common. The nature of these cancers is variable but in the advanced stages they all involve local spread which may result in bladder or bowel involvement and possible spread to pelvic nodes. Metastatic spread can be peritoneal (directly in the case of ovarian cancer and tubal with endometrial disease) or haematogenous to lungs, liver, bone and brain. Women with ovarian cancer often present late in the disease as the tumour has vague, nonspecific symptoms and the space in the peritoneum affords it room for large growth prior to detection. It is likely that this is the reason that ovarian cancer is the leading cause of death from gynaecological cancers. Ascites is also common in stage III-IV ovarian cancer patients. There can also be bowel obstruction in all areas of the bowel and sometimes in multiple places (a contraindication to surgery).

PROCEDURES

The extent of surgery can be variable and depends on the type of cancer and spread. Hysterectomy can be performed for early stage cervical cancer but a radical hysterectomy will be needed for more advanced disease. This involves hysterectomy with excision of the utero-vesical and -sacral ligaments along with removal of the upper part of the vagina and dissection of the pelvic and parametrium lymph nodes. Uterine cancer will involve a bilateral salpingoophorectomy in addition.

Ovarian cancer requires laparoscopic or open clearance from the peritoneum and surrounding structures; it is often highly complex and extensive surgery. Ultra-radical surgery has been used for ovarian cancer (which includes additional resections of liver, spleen, bowel and gall bladder with stripping of the diaphragm and peritoneum) but its benefits over standard ('radical') surgery has not been proven and it has an increased risk of complications. Many procedures are now undertaken laparoscopically so consideration to the effects this type of surgery has on the anaesthetic process is necessary.

PREOPERATIVE PREPARATION

Imaging (e.g. ultrasound, MR and CT scanning) to assess staging of disease and local structure involvement will have been undertaken. A possible decision to involve other specialities (e.g. urology, general surgery) will need arrangement.

Many patients will have undergone a staging procedure which may include hysteroscopy or laparoscopy. Recent assessment of metastatic spread will have been undertaken.

Patients are often of childbearing age so management of fertility may need consideration.

Many gynaecological cancers are treated with adjuvant chemotherapy and radiotherapy. This can have effects on the surrounding tissues and may increase the risk of bleeding perioperatively.

PREOPERATIVE ASSESSMENT

Women presenting for radical cancer surgery can be of any age and therefore can be very different in their premorbid state. A full anaesthetic assessment should be undertaken with a focus on the disease affecting the patient and treatment she has already received.

Metastatic spread or complications may affect anaesthetic management. Ovarian cancer can cause nephritic syndrome and endometrial cancer can lead to hypercalcaemia, for example.

Determine which approach is planned – laparoscopic or open. Check any pre-existing conditions that might affect this choice.

CPET testing may be needed to assess suitability for surgery but it may not be possible depending on the woman's symptoms.

ICU care after surgery may be needed in ASA III or IV patients undergoing extensive surgery. Obesity is a risk factor for gynaecological cancers.

Investigations should include FBC, U&Es, LFTs, bone profile, clotting and group and save (± crossmatch). Do an ECG and CXR if clinically indicated.

Many gynaecological operations are performed as part of an enhanced recovery programme. Long

starvation periods should be avoided and carbohydrate loading drinks are given in the preoperative period. Bowel preparation should only be given where absolutely necessary.

Where possible avoid premedication with anxiolytics but this must be balanced against recognition of the difficult nature of this type of surgery for the patients.

INTRAOPERATIVE CARE

- GA is recommended due to the extensive nature of the surgery; only some vulval surgery may be amenable to spinal anaesthesia.
- Epidural anaesthesia may be used and may possibly confer some long-term benefit, although the technique may be less agreeable to the patient.
- Techniques for GA will depend on the patient and the anaesthetist's preference, but an endotracheal tube is required with muscle relaxation and IPPV.
- Use the ramped position in the obese patient.
- The risk of PONV is high so avoiding nitrous oxide is wise.
- Minimum monitoring as per AAGBI guidelines with additional invasive lines as indicated.
- Maintain normothermia with forced air warming, under body mattresses, warmed intravenous fluids and temperature monitoring.
- Cardiac output monitoring may be indicated.
- VTE prophylaxis is essential.
- Antibiotics as per hospital protocols prior to skin incision. In very long cases, a second dose might be required intraoperatively.
- Care with positioning particularly when the patient is obese. Many operations are now done laparoscopically and require the Trendelenburg position. Ensure that the patient cannot slip from the table. Protect pressure areas as peripheral nerve injury will be more likely in the long case.
- Analgesic requirements will depend on surgical approach, being much less if the case is done laparoscopically. Intrathecal opioids can be administered prior to induction; diamorphine is ideal for this as

it tends to have longer lasting effects than fentanyl or morphine. Transverse abdominus plane blocks or low-thoracic/lumbar epidural analgesia can be used. Wound infiltration by the surgeon is effective. Paracetamol and NSAIDs started intraoperatively should be continued into the postoperative period. It is usual for opioid analgesia to be required unless an epidural is in use; this can be given by PCA.

POSTOPERATIVE

- Encourage eating and drinking as soon as possible after surgery if no contraindications exist.
- Continue IV fluids as needed.
- PONV is common so administer regular antiemetics.
- VTE prophylaxis must continue postoperatively as there is a very high risk of DVT and subsequent PE. Pharmacological methods are usually used.
- Patients who are recovered as part of an enhanced recovery programme can usually expect to be discharged within about 3–4 days after surgery.

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CROSS-REFERENCES

Laparoscopic surgery, Chapter 11
Complications of position, Chapter 30
Prolonged surgery, Chapter 28

Obstetric surgery

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AMY HOBBS AND SOPHIE KIMBER CRAIG

ELECTIVE CAESAREAN DELIVERY

Elective caesarean is the most common obstetric operation (approximately 1 in 4 parturients). It is often performed as an urgent or emergency procedure. Elective caesarean delivery (CD) is most frequently performed under regional anaesthesia in the UK.

INDICATIONS

- Breech
- Multiple pregnancy (depending on the number of babies and their lie)
- Previous caesarean section (where the woman has declined or is not suitable for vaginal birth after caesarean)
- Malplacentation (minor or major placenta praevia) and morbidly adherent placenta (placenta accreta or percreta)
- Preventing transmission of HIV to babies from mothers with high viral loads or not receiving antiretroviral agents
- Maternal choice (including previous traumatic birth experience or tocophobia)

PREOPERATIVE ASSESSMENT

All women should be seen and assessed before surgery. In addition to routine anaesthetic assessment, the following information is relevant:

- Indications for CD and previous obstetric history
- Medical conditions complicating this pregnancy
- Pre-existing medical conditions requiring specialist input
- Neurological conditions with permanently altered sensation or motor function
- Drug history including allergies
- Airway assessment
- Position of placenta on ultrasound scan
- Full blood count (FBC)

High-risk (e.g. BMI >40, cardiac or respiratory disease, risk of massive haemorrhage) patients should be seen antenatally by an anaesthetist to plan and prepare for delivery. Discuss:

- Longer operating time
- Consultant anaesthetist and obstetrician involvement

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- Intraoperative cell salvage
- Specialist advice (e.g. haematologist/ cardiologist, neurologist)
- The potential for perioperative hysterectomy

CHOICE OF TECHNIQUE

The choice is either regional anaesthesia (RA) or general anaesthesia (GA). RA has a better safety profile (Table 12.1). Benefits, risks and technique should be discussed. The patient should be made aware of what to expect with regards to intraoperative sensation (e.g. tugging, stretching). Written information can be provided. The plan for postoperative analgesia should be discussed.

The principal decision will largely depend on either contraindications or patient preference. As with any RA it is important to know about recent anticoagulation therapy as this may influence the technique. Spinals, epidurals or CSE can be used for elective and emergency CD (Table 12.2).

PREOPERATIVE MANAGEMENT

FBC is required to confirm the absence of thrombocytopaenia or a significantly elevated white cell count and to assess anaemia. Group and Save is usual although not essential for low risk cases. Antacid prophylaxis (H_2-receptor antagonist or proton pump inhibitor) is required to reduce the risk of aspiration in the event of conversion to general anaesthesia. Normal starvation rules apply.

THEATRE PREPARATION

Drugs to perform an emergency rapid sequence induction should be prepared, along with vasopressors, sympathomimetics and antimuscarinics. A standard check of all theatre and anaesthetic equipment should be performed. The operating table must be able to tilt into the left lateral, reverse Trendelenburg and Trendelenburg positions. Ensure the weight limit for the table will not be exceeded.

INTRAOPERATIVE CARE

- Routine team brief and WHO Safer Surgery Checklist.
- Full AAGBI monitoring initiated prior to the start of RA.
- Intravenous access with at least one wide-bore cannula (two if there is a high risk of bleeding).
- Commence intravenous fluids; ideally these should be warmed. Spinal anaesthesia can cause profound hypotension. The question of whether to fluid preload or 'co-load' for RA has been researched extensively. There is some evidence that colloids might be more effective than crystalloids but timing does not appear to be a significant factor and fluids alone do not prevent hypotension from spinal anaesthesia. In the elective situation, large amounts of fluids are not usually needed.

Table 12.1 Benefits of regional versus general anaesthesia in elective caesarean delivery

Regional anaesthesia	General anaesthesia
Reduced blood loss	Provides anaesthesia when regional techniques are contraindicated, such as when the woman has:
Improved pain relief postoperatively	
Avoids risks of failed intubation	
The mother is awake when her baby is delivered	• Abnormalities of clotting or recent thromboprophylaxis
	• Significant cranial or spinal abnormalities (e.g. spina bifida, Chiari malformations)
Facilitates the presence of birth partners at delivery	
Supports skin-to-skin contact in the operating theatre	Alleviates profound anxiety of being awake during surgery
	Facilitates the management of complex cases from the outset of the procedure
	Eliminates any intraoperative sensation of tugging/stretching

Table 12.2 Comparison of different regional anaesthetic techniques for caesarean section

Choice of regional technique	Spinal	Epidural	CSE
Benefits of technique	Rapid onset Better quality of block than epidural Reduced incidence of breakthrough pain compared with epidural Reduced incidence of post-dural puncture headache Low rates of failure	Block can be titrated slowly to achieve adequate height Block can be extended for prolonged procedures Block can be augmented if woman experiences breakthrough pain Additional opioids can be administered	Useful for maintaining cardiac stability as block can be brought on slowly, avoiding sudden changes in BP Achieves some rapid pain relief/anaesthesia but can be extended to provide anaesthesia for a longer timeframe Additional opioids can be administered
Potential disadvantages	One-off injection that cannot be extended The rapidity of onset can result in more pronounced hypotension Dense block can lead to subjective feeling of difficulty in breathing	Increased risk of PDPH Risk of administration of intravenous drugs into epidural space	Time taken for insertion Test dose cannot be performed when used for surgery Potential of subarachnoid spread of drug administered into epidural space

REGIONAL ANAESTHESIA TECHNIQUE

Either sitting or lateral, there is little evidence that one is more favourable than the other. The local anaesthetic (LA) is usually combined with intrathecal opioids (diamorphine is usual but morphine or fentanyl are also suitable). Block quality is improved and postoperative analgesia provided when diamorphine is used. The dose of LA required in spinal anaesthesia is less than that required for surgery in the general adult population because engorgement of the epidural veins reduces the size of the intrathecal space. A typical dose might be 2.5 mL of 0.5% heavy bupivacaine combined with 300 µg of diamorphine. Adjust the dose as follows:

- *Extreme prematurity or very small for gestational age babies* – Increase dose.
- *Predicted very large babies or the presence of polyhydramnios* – Decrease dose.
- *The presence of an epidural that has been running for a long time or has required multiple top-up boluses* – Be prepared for exaggerated cephalad spread.

- *Very tall or short women* – May need dose increase or decrease or reverse Trendelenburg positioning.
- *Cardiovascular conditions requiring gradual onset of anaesthetic block* – Epidural or CSE are better.

PREVENTION OF HYPOTENSION

Hypotension following spinal anaesthesia is common. It may compromise placental blood flow. Symptoms of nausea, vomiting or light-headedness may be reported. The aim is to maintain systolic blood pressure at pre-spinal levels:

- Avoid aortocaval compression (tilt table 15° laterally to the left)
- Infuse intravenous fluids
- Administer phenylephrine or ephedrine

Phenylephrine is superior to epinephrine as it causes less foetal acidosis, has less effect on the foetal circulation and works more quickly. It can be administered as an infusion and/or boluses.

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CONFIRMING BLOCK HEIGHT

Bilateral loss of sensation to cold from T4, and light touch from T6 is required. Motor blockade of the legs should also be assessed. Block extent and mode of assessment must be recorded.

OTHER MEDICATIONS

- Prophylactic antibiotics should be given before the skin incision: choice depends on local hospital policies.
- Antiemetics: ondansetron is the ideal choice. Administer before spinal insertion as it also reduces hypotension, vasopressor use and itch.
- Give oxytocin 5 units slowly intravenously after cord clamp (last cord clamp in a multiple delivery). This can cause hypotension and tachycardia if given rapidly. Occasionally the obstetrician might request an infusion after delivery – refer to local protocols (use a concentrated preparation in pre-eclampsia).
- If more than 500 mL blood loss is expected, tranexamic acid 1 g can be given.

GENERAL MEASURES

- Insert a urinary catheter prior to surgery. Remove after the block has fully regressed.
- Protect pressure areas and prevent the patient's legs from falling from the operating table (risk increased by table tilt).
- Avoid inadvertent perioperative hypothermia by active warming. The use of forced air blankets on the upper torso is difficult so a heated mattresses or under-body air blower is recommended.
- Pregnant women are thrombophilic and therefore require thromboprophylaxis for surgery. Compression stockings or intermittent pneumatic calf compression boots are used.
- Accommodate any personal preferences such as music playing in theatre or viewing delivery by dropping the screen where possible. Be

supportive, where clinically feasible, of a mother's desire to have skin-to-skin contact with her baby.

BREAKTHROUGH PAIN MANAGEMENT

Pain during CD is one of the most common causes of litigation. It must be treated. If the woman complains of pain:

- Pause the surgery if possible.
- Establish the nature of the discomfort: is it pain, awareness of pressure or tugging?
- If she confirms pain, she must be offered a GA.
- If she declines GA, treat her pain.

Options include:

- Inhaled nitrous oxide.
- Rapid onset systemic opioids (e.g. alfentanil IV 100–250 μg boluses). Inform the neonatologist if this is done pre-delivery.
- Ketamine in boluses of 10–20 mg may be effective.
- LA infiltration of the skin at the site of incision.

All discussions with the patient and every intervention must be recorded. Document whether each intervention was effective and what further steps were taken to stop the pain if it was not effective. If possible, document the cause of pain (e.g. cleaning of the paracolic gutters). Provide an explanation at the time and debrief the patient postoperatively.

POSTOPERATIVE ANALGESIA

A multimodal regime should be used:

- Paracetamol
- NSAIDs
- Opioids: codeine (not in breastfeeding women) or dihydrocodeine with oral/intravenous morphine as required
- Patient-controlled analgesia: if no long-acting intrathecal opioids were administered
- Bilateral transversus abdominal plane blocks: may reduce postoperative opioid requirements

POSTOPERATIVE MANAGEMENT

- Prescribe thromboprophylaxis and continue into the postoperative period.
- Encourage early mobilisation.
- Women can normally eat and drink following surgery with no restrictions.
- An enhanced recovery programme may be used where, following a straightforward postoperative course, discharge is possible the day after surgery.

SPECIAL CASES

PLACENTA PRAEVIA, ACCRETA AND PERCRETA

- Cross-match four units of blood preoperatively.
- Plan for potentially massive haemorrhage.
- Site at least two large bore cannulae.
- Consider arterial line.
- Use intraoperative cell salvage.
- Ensure a consultant-led team.
- Consider the need for interventional radiology input (e.g. iliac artery balloon insertion).
- Administer an oxytocin infusion post-delivery.
- Give tranexamic acid.

PREMATURE BABIES

- Antenatal steroids will have been given to aid foetal lung maturation.
- Involve the neonatal team in the Team Brief to discuss the plan for delivery (e.g. baby delivered into a plastic bag). Cord clamping may be delayed for 'milking the cord' – give syntocinon only after the cord is eventually clamped.

DIABETES

- Commence a sliding scale of insulin prior to surgery.
- If it is gestational diabetes, insulin can usually be stopped after delivery.
- Involve the diabetologists.

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CROSS-REFERENCES

Epidural, spinal, CSE, Chapter 29
Massive blood transfusion, Chapter 30
Obstetric anaesthesia – general principles, Chapter 12
Emergency caesarean section, Chapter 12
Serious complications of pregnancy, Chapter 12

EMERGENCY CAESAREAN SECTION AND OPERATIVE VAGINAL DELIVERY

The timeframe and need for delivery will depend on the indications for surgery and may be classified into four groups (Table 12.3). These form a continuum of risk.

Table 12.3 Urgency of caesarean delivery (CD)

Category	Risk to mother and/or baby	Indication	Target time for decision to delivery interval (DDI)
1 Emergency	IMMEDIATE THREAT TO LIFE ⬆	Immediate threat to life of woman or foetus (e.g. severe foetal bradycardia, cord prolapse, uterine rupture, foetal blood sample pH ≤7.2)	≤30 minutes
2 Urgent	Maternal or foetal compromise ⬇	No immediate threat to life of woman or baby (e.g. APH, failure to progress)	≤75 minutes
3 Scheduled	TIME FOR PROCEDURE TO BE SCHEDULED	Requires early delivery (e.g. intrauterine growth retardation, failed induction of labour)	In the interests of mother and baby
4 Elective	No maternal or foetal compromise	At a time to suit the woman and maternity services (breech, previous CD)	Usually after 39 weeks of gestation if possible

It is vital to have clear dialogue with obstetric and midwifery teams about the indication for CD as this will guide the decision which anaesthetic technique to use. The indication may change (e.g. a foetal bradycardia may recover affording time for a spinal anaesthetic rather than needing a GA) and teams should be prepared for this.

OPTIONS FOR ANAESTHESIA FOR EMERGENCY DELIVERY

Regional anaesthesia (RA) is used for the majority of emergency and urgent CDs. Labour epidurals can be converted for anaesthesia at the time of decision to deliver. There are however times when a GA is the anaesthetic of choice (barring no contraindications or significant risks to its use).

EPIDURAL TOP-UP

If there is a well-functioning epidural (block height and spread adequate and not required additional clinician boluses to maintain analgesia), this can be used to provide anaesthesia. There is an increased risk of high block if a spinal is inserted following an epidural so be prepared to manage this. The top-up dose can be administered in the delivery room providing the anaesthetist remains present at all times and vasopressors

are immediately available. The agent used will depend on local protocols, but a block from sacral regions to T4 is necessary and usually requires around 2 mL/dermatome to achieve this. Giving the dose in 5–10 mL aliquots will help prevent hypotension or high block. A slight head down position and further 5 mL bolus may be required to anaesthetise the higher thoracic dermatomes. Opioids (fentanyl 100 mcg or diamorphine 2.5–5 mg) will improve the quality of analgesia. Further doses of local anaesthetic can be given during surgery to augment or improve the block.

Hypotension is less profound following epidural top-up compared to intrathecal anaesthesia but may still occur. The epidural catheter should be removed at the end of the operation, provided there is no contraindication.

INDICATIONS FOR GENERAL ANAESTHESIA IN CAESAREAN DELIVERY

When RA is contraindicated, where mothers decline RA or when conditions exist that are life-threatening for mother and/or baby:

- Prolonged foetal bradycardia on cardiotocograph (CTG) with no recovery in the foetal heart rate (FHR)

- Umbilical cord prolapse (with abnormal CTG)
- Major placental abruption (with maternal/foetal compromise)
- Uterine rupture
- Uncontrolled massive haemorrhage

PREOPERATIVE ASSESSMENT

A full assessment should normally be undertaken. In emergency situations, an AMPLE (allergies, medications, past medical history, last eaten, events leading to) history can be taken to aid rapid delivery. A recent FBC should be available and blood should be grouped and saved. If there is preoperative haemorrhage or maternal clinical instability (or a perceived high risk of postpartum haemorrhage), then blood samples for clotting with fibrinogen, cross-match, U&Es and calcium should be sent.

PREOPERATIVE MANAGEMENT

If there is a high risk of needing a CD, regular ranitidine should be given during labour and oral intake restricted to clear fluids. In cases of emergency CD, intramuscular or intravenous ranitidine can be given. Sodium citrate is administered immediately before induction.

In the case of emergency CD, rapid transfer from the delivery room or ward to theatre is required. Intrauterine foetal resuscitation (e.g. stopping oxytocin infusions, administering oxygen, tocolysis) should be considered.

THEATRE PREPARATION

The maternity theatre should always be open and ready to undertake an emergency CD. Difficult airway equipment should be available.

With category 2 and 3 CD, it is advisable to have a Team Brief to plan care. In category 1 CD, there is not usually time for this but open communication between all team members in theatre is recommended.

INTRAOPERATIVE CARE

A WHO Safer Surgery checklist is necessary for all cases including emergencies.

As with elective cases, full monitoring, IV access and IV fluids are essential. Prophylactic antibiotics should be given. Other general measures (as for elective caesarean section) should also be completed. The CTG should be attached until the abdomen is prepared for surgery in emergency CDs.

INDUCTION OF ANAESTHESIA

There is an increased risk of failed intubation in obstetric practice. Prior to induction the anaesthetist should decide whether to wake the patient or proceed with alternative airway management, in the event of a failed intubation.

A rapid sequence induction (RSI) is required. Left lateral tilt will be in place so optimise position for intubation in other ways (e.g. ramped position in the obese parturient). A trained anaesthetic assistant must be present. Ensure full preoxygenation. Cricoid pressure should be used if it is normally employed in RSI.

During preoxygenation, the surgical team can prepare the abdomen ready for surgery.

A predetermined dose of induction agent is given followed by a fast-acting muscle relaxant. Once the airway has been intubated, tube position confirmed and secured, surgery can then proceed.

CHOICE OF ANAESTHETIC AGENTS FOR GA

INDUCTION

Thiopental is commonly used for induction in the UK. Propofol is a suitable alternative although it is not currently licensed for use in pregnancy. Ketamine has advantages in cardiovascular instability but it does have well-reported side effects and a less clear end point for induction. Opioids are not normally given at induction due to the risk of placental transfer to foetus. Suxamethonium allows rapid intubation but it does have side effects including malignant hyperpyrexia and suxamethonium apnoea in susceptible patients. Rocuronium can be used in a dose of 1–1.2 mg/kg but its affects are long-lasting and it should only be used where suggamadex for reversal (16 mg/kg) is immediately available.

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MAINTENANCE

Inhalational agents are usually used to maintain anaesthesia (with a target MAC of 1.0). They cause relaxation of the uterus which may contribute to uterine atony and blood loss. Adding nitrous oxide will augment anaesthesia. Care should always be taken to ensure adequate depth of anaesthesia; emergency obstetric surgery is over-represented in the data for accidental awareness under general anaesthesia. An emergency CD requires the same care as an elective procedure. Opioids should be commenced intraoperatively after delivery if no epidural or intrathecal opioids have been given. If suxamethonium was used for intubation, a non-depolarising agent will be required.

MANAGING FAILED INTUBATION

If a failed intubation occurs there are two main options: wake up or use an alternative method for maintaining the airway. This decision depends on several factors – see the DAS and OAA guidelines on managing the difficult or failed obstetric airway. These should be followed and the critical nature of the situation declared to the theatre team.

POSTOPERATIVE MANAGEMENT

Ensure adequate reversal after muscle relaxation. Extubate once fully awake and in the left lateral position. If there is concern about stomach contents, an NG tube can be inserted and aspirated prior to waking. Transfer to a fully equipped and staffed recovery area. Cases of pre-eclampsia/eclampsia require careful management if they require emergency CD.

OPERATIVE/INSTRUMENTAL VAGINAL DELIVERY

Sometimes assistance with delivery at full cervical dilatation is required (e.g. suction or forceps). This may be performed in the delivery room either under existing epidural analgesic blockade or using a pudendal nerve block. If it is predicted to be potentially difficult and at high risk of conversion to CD, then it should be done in theatre. A spinal or epidural top-up is suitable for this procedure. A good sacral block that extends right up to the T4 dermatome is required.

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GENERAL PRINCIPLES OF OBSTETRIC ANAESTHESIA

As pregnancy progresses, multisystemic physiological changes develop rapidly. Obstetric anaesthetists must understand these to enable them to provide optimum care. Pregnant women may also

come under the care of nonobstetric anaesthetists at any stage of gestation due to incidental surgery (e.g. appendicitis), trauma (30% of domestic abuse starts in pregnancy) or complications in the immediate postnatal period (e.g. bleeding, breast abscess).

These physiological changes are not new, but the patient group in whom they are occurring is becoming increasingly complex. Obstetric units are now managing more patients who may have

- Significant cardiovascular or cerebrovascular disease
- Significant respiratory disease, which may worsen throughout pregnancy
- Morbid obesity (BMI >40) or super morbid obesity (BMI >50)
- Significant haematological disease which may previously have resulted in failure to reach viable gestation
- Corrected or palliated congenital heart disease

It is also important to remember that migrant women and women from minority ethnic groups are consistently over-represented in maternal morbidity audits.

Multidisciplinary team planning and management is now well established in the management of high risk parturients, involving anaesthetists, obstetricians, midwives and various medical and surgical specialist teams.

PHYSIOLOGY OF PREGNANCY

Physiological changes (Table 12.4) are largely due to the effects of progesterone. Modified early warning scores for obstetrics accommodate these changes and should be used.

PHARMACOLOGICAL CHANGES IN PREGNANCY

Pregnant patients are more sensitive to the effects of general anaesthetics than non-pregnant patients. Awareness is much more common in obstetrics than other surgical specialties – 10% of all cases of accidental awareness reported to the Royal College of Anaesthetists' NAP5 project.

Concerns about sedation of the foetus must not allow inadequate dosing for the mother, who is the primary patient at the time of any surgery. Table 12.5 summarises the important pharmacological changes in pregnancy.

UTEROTONIC AGENTS

Drugs to promote uterine contraction are frequently required during the peripartum period. Choice of drug must consider patient comorbidities and the side effect profiles of these agents (Table 12.6).

BREASTFEEDING MOTHERS IN THE POSTPARTUM PERIOD

Lipid soluble drugs are secreted into breast milk and are therefore passed to neonates in the postpartum period. Caution must be taken when prescribing drugs to any woman who is breastfeeding. In particular, MHRA advice states that codeine should not be used by breastfeeding mothers because it can pass to the baby through breast milk and potentially cause harm. Dihydrocodeine was not covered in this guidance.

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Table 12.4 Physiological changes in pregnancy

System	Parameter	Change	Relevance
Cardiovascular	Heart rate	Increases throughout pregnancy to 20%–25% above baseline by term	Masks sepsis and haemorrhage
	Systemic vascular resistance	Decreases by 35%–40% by 20 weeks, then plateaus or increases to term	Prone to postural hypotension
	Cardiac output	Increases by up to 45% by 24 weeks	Increased myocardial demand
	Aortocaval compression	Aorta and IVC compressed significantly in supine position by 20 weeks	Left lateral tilt or manual displacement of uterus required when supine
Respiratory	Minute volume	Increases by 45% by term due to 15% increase in respiratory rate by 20 weeks and 40% increase in tidal volume by term	$PaCO_2$ falls to 4.1 kPa, compensated for by fall in bicarbonate
	Functional residual capacity	Reduced by 20% at term	Desaturate quickly Preoxygenation less effective than in non-pregnant patients
	Oxygen consumption	Increased by 60% at term	Desaturate quickly
Airway	Lumen size	Increased capillary engorgement and oedema	Increased rate of failed intubation (2.6 per 1000 obstetric GAs) Increased risk of bleeding
Haematological	Plasma volume	Increases by 45% at term	Increased myocardial workload
	Red cell mass	Increases by 20%–30% at term	Physiological anaemia (plasma volume increases proportionally more than red cell mass)
	Coagulation factors	Increase in fibrinogen and all clotting factors except FXI and FXIII	Hypercoagulable state
		Increase in antithrombin III and fibrin degredation products	Increased fibrinolysis
Renal	Glomerular filtration rate	Increased by 50%–60% by term	Reabsoption capacity overwhelmed causing mild glycosuria and proteinuria
Gastrointestinal	Reflux	Cephalad displacement of stomach throughout pregnancy	Increased rates of heartburn and reflux
Endocrine	Insulin	Increased insulin production Increased insulin resistance	Overall insulin resistance Gestational diabetes

Table 12.5 Pharmacological changes in pregnancy

Variable	Change	Relevance
Volume of distribution	Increased	Resistance to water soluble drugs, e.g. non depolarising muscle relaxants and digoxin
Protein binding	Reduced	Increased free fraction of protein bound drugs, e.g. thiopentone and local anaesthetics
Plasma cholinesterase	Concentration falls by 25%	Counteracted by increased volume of distribution so duration of action of suxamethonium is unchanged
Neural sensitivity	Increased	MAC for volatile agents reduced by up to 30%
Placental transfer	Drug transfer to foetus	Important for lipid soluble drugs, e.g. opiates
Epidural and subarachnoid space	Volume reduced	Increased spread of neuraxial blocks

Table 12.6 Uterotonic agents

Drug	Dose	Route	Contraindications	Side effects
Oxytocin	5 units or as an infusion	IV slowly	Inactivated if given through same line as blood or plasma	Hypotension
Ergometrine	0.5 mg	IM	Pre-eclampsia or hypertension	Hypertension, diarrhoea, vomiting
Carboprost (Haemabate)	250 micrograms repeated every 15 minutes up to 8 doses	IM	Asthma Intracardiac shunt	Bronchospasm, diarrhoea, fever
Misoprostol	600–1000 μg	Usually PR (also PO or SL)		Fever

Note: Syntometrine is premixed oxytocin 5 units plus ergometrine 0.5 mg and should be given IM.

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MEDICAL PROBLEMS IN OBSTETRIC ANAESTHESIA

ACQUIRED CARDIAC DISEASE

Cardiac disease is the leading cause of maternal death in the UK. General principles of management are shown in Table 12.7. Acquired cardiac conditions contributing to mortality include:

- Ischaemic heart disease (obesity and advanced age increasingly seen in the obstetric population)
- Aortic dissection
- Cardiomyopathy
- Symptomatic valvular heart disease
- Sudden adult death syndrome (SADS)

CONGENITAL CARDIAC DISEASE

Patients with congenital heart disease are surviving longer and are subsequently being seen in the obstetric setting. Patients with good functional status and/or corrected anatomical abnormalities do not need specialised treatment. The WHO criteria can be used to risk stratify patients and plan for their place of delivery (Table 12.8).

Anaesthetic goals are outlined in Table 12.9. Key points in the management of patients with congenital heart disease during pregnancy, labour and delivery are

- Multidisciplinary planning and assessment throughout pregnancy, with transfer of care to a tertiary centre with cardiac surgery capability in all complex cases.
- Invasive blood pressure monitoring throughout labour and delivery.
- Epidural analgesia for labour and cautiously titrated CSE for operative interventions. Air should not be used for loss of resistance to avoid the risk of paradoxical air embolus.

NEUROLOGICAL DISEASE

Anaesthetists may become involved in the care of pregnant or recently pregnant women with acute presentations of commonly occurring neurological diseases, such as stroke, subarachnoid haemorrhage, epilepsy and status epilepticus. Pregnancy should not alter the usual medical care of these emergencies, including thrombolysis administration.

Multiple sclerosis commonly presents in women of childbearing age. There is a theoretical risk of neurotoxicity to demyelinated nerves with regional anaesthesia (RA). This potential risk is higher with the higher concentrations used in spinal anaesthetics compared to epidural anaesthesia. However, no convincing evidence exists that this actually occurs and relapse rates are no higher in postpartum women who received epidurals compared to those who did not.

Table 12.7 Peripartum management of women with acquired cardiac disease

Antenatal	Assess symptoms and functional status (NYHA class)
	Review recent ECG and echo
	Multidisciplinary planning for labour and delivery
	Assess need for transfer to tertiary centre with cardiac surgery capability
Labour and delivery	Continuous maternal monitoring in labour, with 5 lead ECG and invasive blood pressure monitoring for high risk patients
	Epidural analgesia reduces pain-induced sympathetic drive and is recommended (avoid using adrenaline in test dose)
	Second stage may need to be limited and assisted delivery is more likely
	CSE for caesarean section provides less haemodynamic fluctuation than spinal anaesthetic
	Precaution against hypertensive response to laryngoscopy is essential if GA is required
Postnatal	Caution with uterotonic agents due to side effects
	Haemodynamic monitoring for first 24 hours as risk of decompensation with autotransfusion postpartum

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Table 12.8 Pregnancy related risks of specific heart lesions

WHO class	Pregnancy related mortality	Example lesions
Class I	No detectable increased risk of maternal mortality and no/mild increase in morbidity	Pulmonary stenosis Patent ductus arteriosus Ventricular septal defect
Class II	Small increased risk of maternal mortality or moderate increase in morbidity	Atrial septal defect Repaired Tetralogy of Fallot Most arrhythmias
Class II-III (depending on status)		Hypertrophic cardiomyopathy Heart transplantation Marfan syndrome without aortic dilatation
Class III	Significantly increased risk of maternal mortality or severe morbidity	Mechanical valve Systemic right ventricle Any cyanotic heart disease
Class IV	Extremely high risk of maternal mortality or severe morbidity. Pregnancy is contraindicated	Pulmonary arterial hypertension Marfan syndrome with dilated aorta >40 mm Left ventricular ejection fraction <30%

Table 12.9 Anaesthetic goals for managing cyanotic congenital heart disease

Goal	Treatment
Maintain preload	Avoid prolonged fasting Give IV fluids
Maintain afterload	Avoid spinal anaesthetic (loss of sympathetic tone) Use phenylephrine for hypotension (or noradrenaline in non-responders) Slow (2 units/minute) administration of oxytocin
Avoid tachycardia	Effective analgesia Early cardioversion for any tachyarrhythmia
Keep pulmonary vascular resistance low	Administer oxygen throughout labour Avoid hypercarbia by preventing sedation

Myasthenia gravis affects women of child-bearing age. Instrumental delivery is often recommended as muscles fatigue quickly. RA is preferable to general anaesthesia but patients cannot compensate for a high block. Magnesium is relatively contraindicated as it can precipitate a myasthenic crisis.

RESPIRATORY DISEASE

Women continue to die from asthma exacerbations in or soon after pregnancy. Although progesterone is a bronchodilator, the reduced functional residual capacity as pregnancy progresses, combined with a relative pulmonary resistance to cortisol, mean that asthma may worsen during pregnancy. Severity is often underestimated – remember that $PaCO_2$ is physiologically lower in normal pregnancy.

Women with significant lung disease should be screened for pulmonary hypertension, as this is associated with higher mortality and appropriate counselling and treatment should be offered.

HAEMATOLOGICAL DISEASE

Women with pre-existing or newly diagnosed haematological disease should be seen in a joint obstetric/haematology clinic. Broadly, three groups exist:

1. Increased risk of VTE, e.g. Factor V Leiden, antiphospholipid syndrome

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2. Increased risk of bleeding, e.g. von Willebrand's disease, thrombocytopaenias
3. Reduced oxygen carrying capacity, e.g. sickle cell disease, thalassaemia, spherocytosis

Use of RA is not contraindicated but attention must be paid to the timing of anticoagulant, if used, and a recent assessment of platelet count is required in thrombocytopaenia.

BACK PROBLEMS

Many women presenting to antenatal anaesthetic clinics are referred due to concerns about RA and backache. Prospective randomised studies have proved that women who receive labour epidurals are no more likely to suffer long-term backache than those who do not.

Patients who have undergone most types of spinal surgery may be offered RA, avoiding scar sites. RA is contraindicated in women with implanted rods following scoliosis surgery.

Women with spina bifida must have an MR to exclude tethered spinal cord. If this has been excluded, RA can be sited at an unaffected level. Accidental dural puncture is more common as the ligaments are abnormal. There is altered dural permeability and a reduced epidural volume, so lower volumes are needed to provide an adequate block.

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Congenital heart disease in adult life, Chapter 2
Amniotic fluid embolism, Chapter 30

PAIN RELIEF IN LABOUR

During the first and early second stages of labour, visceral pain (mediated by the T10 to L1 spinal segments) is experienced. This is usually felt in the abdomen, sacrum and back. Somatic pain, modulated via T12-L1 and S2-4, is felt in the latter part of the first stage of labour and into the second stage and is more localised to the vagina, rectum and perineum.

MANAGING EXPECTATIONS

It is important to understand the complex nature of maternal satisfaction with childbirth and pain relief. It is likely that attitudes and behaviours of caregivers have a more powerful influence than the effects of labour pain, analgesia and interventions. Important factors in maternal satisfaction include:

- Personal expectations
- Support from caregivers (amount and quality)
- Mother's involvement in decision making

Most women present in labour with some expectation or plan for the management of their pain.

METHODS FOR PAIN RELIEF

Non-pharmacological methods have varied evidence of efficacy (Table 12.10).

ENTONOX®

Entonox® is a 50:50 mixture of nitrous oxide and oxygen given via a demand mouthpiece. It has rapid

Table 12.10 Non-pharmacological pain relief in labour

Evidence supports use	May offer some benefit	No evidence of benefit
Immersion in water	Acupuncture	TENS machine
Breathing and relaxation techniques	Hypnosis	Injected water papules
Massage		Aromatherapy
		Audio-analgesia/music

onset and offset so women are encouraged to breathe it during the course of their contractions and to cease use in between them. It relieves pain but may cause some drowsiness, nausea and vomiting. It has no effect on maternal or neonatal outcome and can be used in both hospital and community settings. It can also be used during pool births.

OPIOID ANALGESIA (PETHIDINE AND DIAMORPHINE)

Intramuscular pethidine and diamorphine are commonly used in UK practice. They can both be administered by midwives. The evidence for their use in significantly reducing labour pain is limited. They do not perform well against alternative methods of pain relief in RCTs and cause sedation, nausea and vomiting (pethidine > diamorphine). There is also the risk of transfer to the foetus with consequent respiratory depression. An antiemetic should be administered.

REMIFENTANIL PATIENT-CONTROLLED ANALGESIA (PCA)

Small bolus doses (20–40 µg) are self-administered with a lockout of 2–5 minutes with contractions. Remifentanil compares well against pethidine but is less effective than an epidural. One in 10 women convert to epidural following its use (less than convert with pethidine). It is particularly advantageous when regional analgesia is contraindicated. There is currently no evidence that it adversely affects labour or neonatal outcomes.

A significant risk of respiratory depression is associated with its use; 1:1 midwifery care during labour is essential, never leaving the patient alone. Naloxone and a self-inflating bag with oxygen should be immediately available. A separate cannula should be used for its administration. Counselling in its use

is essential. Family members/birth partners must be advised not to activate the PCA.

EPIDURAL ANALGESIA

Epidurals are the most effective method of pain relief in labour but are associated with complications. In addition to the usual risks associated with epidural insertion and use, they may have the following additional impact on labour:

- Increased length of second stage (but not the first stage)
- Increased risk of instrumental delivery (but no change in the caesarean delivery rate)
- Increased levels of monitoring for both mother and baby which may affect mobility in labour

Perform full assessment, take consent and check that there are no contraindications to insertion. There is some concern about the validity of obtaining consent whilst the woman is in pain (although it is common practice) so make every effort to ensure good understanding and where possible, provide antenatal information about epidurals. Offer written information about the insertion and risks. She also requires:

- IV access (preloading with fluids is not required)
- CTG monitoring
- Positioning – either lateral or sitting
- Vital sign monitoring

Watch carefully for accidental dural puncture (ADP). If this occurs, then the epidural catheter can either be placed intrathecally or a repeat epidural procedure attempted. Always inform the woman if ADP occurs and advise her what to expect.

It is common practice to use some of the local anaesthetic (LA) and opioid mixture as a test dose.

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Table 12.11 Problems encountered with epidural analgesia

Issue	Recommendation
Missed segments	• Sometimes responds to positioning the patient on the unblocked side
	• Administer a top up
Unilateral block	• Sometimes responds to positioning the patient on the unblocked side
	• Administer a top up
	• If this is not effective withdraw catheter so that only 3 cm remains in the epidural space – then repeat the bolus
Inadequate sacral/ perineal block	• This can be difficult to get rid of particularly if the foetus is positioned occipital-posteriorly
	• Try giving a bolus dose in the sitting position
	• Add further fentanyl in divided doses of 50 mcg in 10 mL saline up to a maximum of 200 mcg in 2 hours
	• If delivery is imminent, a spinal with a small volume bolus of low-dose mixture with the mother in the sitting position is an option
	• Resiting may not be effective in this instance
Breakthrough pain in the first stage	• Check the distribution of the block
	• Always consider uterine scar rupture if there is breakthrough pain in the presence of adequate distribution
	• If block is <T10:
	• Check catheter connection and site
	• Give bolus of 10 mL
	• Bolus can be repeated after 30 minutes
Breakthrough pain in second stage	• Give a top-up dose of 10 mL just before pushing starts
	(Low dose local anaesthesia will not remove the urge to bear down for more than a few minutes so should not be withheld at this stage)

Following the test dose, administer bolus doses (either intermittently or as patient-controlled epidural analgesia) rather than an infusion. Low-dose (≤0.125% bupivacaine) local anaesthesia combined with opioids (e.g. fentanyl 2 μg/mL) are usually adequate. When these low concentrations are used mobilisation is possible ('walking epidurals') but patient and staff must be aware of the risk of injury if inadequately supervised.

CSEs can be used as an alternative to epidural alone. They allow for rapid analgesia if distressed when approaching full dilatation. The epidural component will need to be tested once the spinal component starts to recede.

Maternal observations (block height and motor effect) should be undertaken every 5 minutes after each bolus for 15 minutes and hourly thereafter. If the block is higher than T6 or there is motor block of the legs, the epidural should be stopped and the anaesthetist consulted. Epidurals are not always 100% effective (Table 12.11).

FOLLOW-UP

All women receiving intervention for pain relief from an anaesthetist during their labour should be followed up. In the event of ADP, an epidural blood patch may be needed. Whatever management is chosen, ensure that they are given information about how to seek help and what to look out for on discharge. Their GP should also be informed.

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Obstetric anaesthesia – general principles, Chapter 12
Postoperative pain management, Chapter 30

SERIOUS COMPLICATIONS OF PREGNANCY

The UK maternal mortality rate is currently the lowest it has ever been. Globally, indirect causes of death (e.g. cardiac disease, influenza) are now more common than deaths directly attributable to pregnancy (e.g. thromboembolic disease, pre-eclampsia). For every death, 9 women develop severe maternal morbidity. High care units are increasingly seen integrated into obstetric settings.

THROMBOEMBOLIC EVENTS

Due to increases in clotting factors and the compressive effects of the uterus on the pelvis, the relative risk of venous thromboembolic events (VTE) is increased by 4–6 times during pregnancy. The risk is even higher in the first 6 weeks postpartum. VTE was the leading direct cause of maternal death in the last two confidential enquiries. Although multiple risk factors are identified (Table 12.12), in 17% of deaths due to VTE in the last triennial report, no additional risk factor was found.

Traditional tools for predicting DVT such as D dimer and the Wells score are not appropriate in pregnancy. Compression duplex ultrasound is recommended. CTPA remains the gold standard for diagnosis of pulmonary embolism (PE) and should be carried out within an hour of suspicion of PE. Concerns about foetal radiation should not restrict access to appropriate imaging. Treatment dose low

Table 12.12 Risk factors for peripartum VTE

Pre-existing	Obstetric	Transient
Previous VTE	Multiple pregnancy	Immobility
Thrombophilia, e.g. Factor V Leiden, protein C/S deficiency	Pre-eclampsia	Current systemic infection
Medical comorbidities, e.g. cancer, active SLE, heart failure	Mid-cavity or rotational operative delivery	Long-distance travel (>4 hours)
Age >35	Caesarean section	Hyperemesis/dehydration
BMI ≥30 kg/m²	Prolonged labour (>24 hours)	
Smoking	Stillbirth	
Parity ≥3	Preterm birth	
Gross varicose veins	Postpartum haemorrhage	

molecular weight heparin (LMWH) should be commenced in suspected cases as soon as possible.

Patients at intermediate and high risk of VTE should be on LMWH throughout their pregnancy. Pay attention to the timing of doses before any regional block is sited. In accordance with national guidelines, blocks should not be sited for 12 hours after the last prophylactic dose of LMWH and the next dose should be delayed for 4 hours after a spinal or after removal of an epidural catheter. Epidural catheters should not be removed until 12 hours after the last dose of LMWH. If treatment dose LMWH is used, a block must be delayed for 24 hours after the last dose.

HAEMORRHAGE

Haemorrhage is the leading cause of maternal death worldwide. In the UK, it is responsible for approximately 0.5 deaths per 100,000 maternities but accounts for a high rate of maternal morbidity in the peripartum period. The definition of major obstetric haemorrhage varies from the loss of 1,000 mL to 2,500 mL acutely, or need for blood transfusion of ≥5 units red cells or for treatment for coagulopathy. It is essential to consider blood loss as a proportion of the circulating volume of 100 mL/kg for a pregnant patient. Therefore 2,000 mL blood loss would be 25% of circulating blood volume for an 80 kg patient but 40% of circulating blood volume for a patient weighing 50 kg.

Anaemia should be identified as early as possible and treated appropriately throughout pregnancy, usually with iron supplementation.

ANTEPARTUM HAEMORRHAGE

The most common causes of antepartum haemorrhage (APH) are placenta praevia and placental abruption. Vasa praevia and uterine rupture are rarer causes. Placental abruption is a clinical diagnosis, indicated by continuous abdominal pain and foetal distress.

Placenta praevia is graded 1 (mild) to 4 (complete) depending on the degree to which the placenta covers the internal os of the cervix. Elective caesarean section is recommended when the placental edge is <2 cm from the internal os. Significant bleeding should be expected at caesarean section and cell

salvage should be used. The care bundle for management of high grade placenta praevia is

- Consultant obstetrician and anaesthetist directly supervising delivery
- Blood and blood products available
- Multidisciplinary involvement in preoperative planning
- Discussion and consent to include possible interventions
- Availability of a level 2 critical care bed

The choice of regional (RA) or general (GA) anaesthetic is dependent on the urgency of the situation and any contraindications. Evidence suggests that there is more blood loss and a higher transfusion rate in patients receiving GA compared to those receiving RA.

POSTPARTUM HAEMORRHAGE

The causes of postpartum haemorrhage (PPH) are commonly referred to as the '4 Ts':

1. Poor uterine **T**one (most common cause)
2. **T**rauma to genital tract
3. Retained **T**issue, including morbidly adherent placenta (accreta, percreta, increta)
4. Disorder of **T**hrombin or other coagulopathy

Haemorrhage may be concealed in the peritoneal cavity, uterus or retroperitoneal space. Signs of shock should be sought, i.e. tachycardia, tachypnoea, poor peripheral perfusion (may present to the anaesthetist as difficulty obtaining venous access), confusion, oliguria and hypotension. Bleeding must be excluded in all tachycardic patients.

MANAGEMENT

Multidisciplinary teamwork is essential. Resuscitation should follow an ABC approach.

- Administer oxygen.
- Left lateral tilt if antepartum.
- Stop epidural if in use for labour analgesia.
- Consider intraosseous access early in compromised patients if large bore intravenous access cannot be established.
- Give warmed crystalloid fluid until blood is available.

- Take blood for full blood count, coagulation screen including fibrinogen, U&E and cross-match.
- Activate major (obstetric) haemorrhage pathway.
- Transfuse warmed blood (O negative, group specific or cross-matched blood, depending on availability and need).
- Fibrinogen should be maintained >2 g/L; therefore, cryoprecipitate may be required sooner than in other adult massive haemorrhage.

Specific management will depend on the cause but consider the use of bimanual uterine compression, uterotonics, tranexamic acid and transfer to theatre for surgical management of bleeding. This may include balloon tamponade, brace suturing, interventional radiology, iliac artery ligation or hysterectomy.

The presence of cardiovascular instability is a relative contraindication to RA and may worsen hypotension. Developing coagulopathy also increases the risk of complications following RA. In many situations, rapid sequence induction is, on balance, the safest option. Some would advocate the use of ketamine (1–2 mg/kg) for induction but this ultimately depends on anaesthetist preference and experience. Remember that inhalational anaesthetics are uterine relaxants – keep the concentration as low as possible while ensuring adequate depth of anaesthesia. Invasive monitoring may be appropriate.

AMNIOTIC FLUID EMBOLISM

Amniotic fluid embolism (AFE) is estimated to occur in 1 in 50,000 deliveries in the UK. The mortality rate ranges from 11%–61% and this range may reflect the fact that AFE is difficult to diagnose and is a diagnosis of exclusion or made at postmortem.

AFE presents as acute maternal collapse during labour or within 30 minutes of delivery. Other features vary, making it difficult to distinguish from many more common diagnoses, e.g. placental abruption, eclampsia, sepsis, PPH, MI, transfusion reaction, local anaesthetic toxicity and anaphylaxis. The most common features are maternal collapse associated with:

1. Haemorrhage (65%)
2. Hypotension (63%)
3. Shortness of breath (62%)
4. Coagulopathy (62%)

Management needs multidisciplinary involvement in all cases. Adopt an ABC approach, give oxygen and be prepared to intubate and ventilate early. Cardiopulmonary resuscitation, if needed, should be prompt and follow ALS guidelines, maintaining uterine displacement. Perimortem caesarean section should be carried out within 5 minutes of cardiac arrest, in situ not in theatre. This is to reduce myocardial oxygen demands of the mother and not to improve the outcome for the foetus. Management for haemorrhage is as above and the major haemorrhage protocol should be triggered at the time of decision for caesarean section. Survivors will require ICU admission.

SEPSIS

Genital tract sepsis alone was the fourth most common cause of direct death in the 2015 triennial report on maternal death. It also accounts for significant morbidity in pregnancy. Patients may require ICU admission, adding to the difficulties of planning delivery of the baby in a timely manner. Sepsis from other sources may also occur in obstetrics. Scottish data shows a rate of severe maternal sepsis of 0.1 per 1,000 live births.

Sepsis should be managed as in any other patient following the Surviving Sepsis Campaign bundle (Table 12.13). Antibiotic choice should follow local protocols.

While sepsis screens in other populations might include blood, urine and sputum samples, in pregnant or recently pregnant patients, additional potential sources must be sought, i.e. high vaginal swabs and breast milk samples. Also remember that positive microbiology results may come back sooner for the neonate (if delivered) than the mother, so liaison with the neonatal unit is essential.

PRE-ECLAMPSIA AND ECLAMPSIA

Pre-eclampsia is defined as hypertension presenting after 20 weeks gestation with significant proteinuria (spot urinary protein:creatinine ratio >30 mg/mmol or a 24-h urine collection with >300 mg protein). It is mild-moderate if systolic blood pressure is 140–159 mmHg or diastolic blood pressure is 90–109 mmHg. Severe pre-eclampsia is defined as:

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Table 12.13 Management bundle for sepsis

Within 1 hours of presentation/diagnosis		Within 6 hours of presentation/diagnosis	
TAKE	Blood and measure lactate	GIVE	Vasopressors if not fluid responsive
	Blood cultures prior to antibiotics		to maintain MAP >65 mmHg
	Urine and measure fluid balance		
GIVE	Broad spectrum antibiotics		Reassess fluid status and lactate
	30 mL/kg crystalloid for hypotension or lactate		
	>4 mmol/L		
	High flow oxygen		

- Systolic blood pressure >170 mmHg or diastolic blood pressure >110 mmHg on two or more occasions, with proteinuria >3+ OR
- Systolic blood pressure >160 mmHg or diastolic blood pressure >100 mmHg on two or more occasions with any of: headache, visual disturbance, vomiting, subcostal pain, papilloedema, clonus (≥3 beats), liver tenderness, thrombocytopaenia (<100 × 10⁹/L), abnormal liver enzymes, HELLP syndrome (haemolysis, elevated liver enzymes and low platelets)

Pre-eclampsia is a multisystem disease which results from impaired trophoblast cell invasion during early development of the placenta. This results in failure of spiral artery dilatation and eventually placental hypoperfusion and hypoxia. The placenta secretes cytokines and inflammatory mediators into the maternal circulation in response to hypoxia and the result is widespread endothelial damage and organ dysfunction. Proteinuria is evidence of renal endothelial involvement. HELLP is a variant of pre-eclampsia, diagnosed with evidence of haemolysis, ALT levels above 75 IU/L and a platelet count <100 × 10⁹/L.

Eclampsia is at the extreme end of the disease spectrum and is defined as the occurrence of one or more convulsions during pregnancy or the first 10 days postpartum, with two or more of the following features within 24 hours of the convulsions:

- Hypertension
- Proteinuria
- Thrombocytopaenia
- Elevated liver enzymes

Pre-eclampsia and eclampsia are now rare causes of maternal mortality (mortality rate of 0.25 per 100,000 deliveries in the 2011–13 triennial report). However, pre-eclampsia affects 5%–8% of pregnancies and so is a common cause of maternal and foetal morbidity.

Management requires multidisciplinary teamwork and coordination to plan for delivery at an appropriate time. Hospitals have their own protocols for management of severe pre-eclampsia and eclampsia. First line management for hypertension is labetalol. If this fails to control hypertension, or is contraindicated (e.g. in asthma), second line agents are nifedipine or hydralazine. Fluid overload and pulmonary oedema can occur rapidly with severe pre-eclampsia. Total fluid input should be restricted to 80 mL/h and output should be closely monitored via urinary catheterisation.

Seizure prophylaxis with magnesium should be considered for all patients with severe pre-eclampsia. Magnesium is also the first choice agent for seizure treatment in eclampsia.

Senior anaesthetists should be closely involved in the care of pre-eclamptic patients, providing advice concerning blood pressure control and fluid balance. Individual units may have guidance on invasive blood pressure monitoring. It is advisable to consider this in unstable or obese patients, once two antihypertensive agents are being used or frequent blood samples are required.

Epidural analgesia for labour is often recommended in pre-eclampsia, as it can prevent pain contributing to hypertension. Pre-eclampsia can be associated with thrombocytopaenia, so check platelet count. Blood pressure rarely falls in true pre-eclampsia with any RA, but can be managed with phenylephrine as in the non-hypertensive obstetric patient if it does occur.

Where RA is not possible or there is coagulopathy, GA may be required in pre-eclamptic patients

for operative delivery, manual removal of placenta or for management of PPH. It is imperative that the hypertensive response to laryngoscopy is ameliorated, as this has been a cause of direct maternal mortality on the past. No agent has been proved to be superior to others. The anaesthetist should choose an agent with which they are familiar, e.g. alfentanil, fentanyl, lidocaine, esmolol, labetalol or magnesium. Emergence hypertension may require a further bolus of an appropriate agent. The widespread oedema seen in pre-eclampsia will also affect the airway so anticipate and plan for a difficult airway and expect to need a smaller than usual endotracheal tube. Magnesium potentiates the action of the nondepolarising muscle relaxants and fasciculations may be reduced following suxamethonium. In most cases, NSAIDs should be avoided as acute kidney injury is often already present.

PERIPARTUM CARDIOMYOPATHY

Peripartum cardiomyopathy (PPCM) is an uncommon cause of dilated cardiomyopathy, occurring in 1 in 2,500 to 1 in 4,000 deliveries. It occurs between 36 weeks gestation and 5 months postpartum. Symptoms are typical of heart failure, with breathlessness, orthopnoea, tachycardia, gross oedema and fatigue. These can be difficult to distinguish from pregnancy-related symptoms so echocardiography is required for diagnosis. There is a high rate of recurrence in future pregnancies.

Dilated cardiomyopathy results in mainly systolic dysfunction. The mainstay of treatment during pregnancy is diuretics and beta blockers. Postpartum, angiotensin converting enzyme inhibitors should be added. Prophylactic LMWH may be used to reduce VTE risk and in these cases, attention must be paid to the timing of any RA.

Any patient with antenatal PPCM should be seen by an anaesthetist and an agreed plan made for labour and delivery. A recent assessment of electrolytes and an echocardiogram will be required. Arrhythmias should be treated appropriately, particularly rate control for atrial fibrillation. Invasive blood pressure monitoring is recommended in the perioperative period. RA is useful as the vasodilatation reduces afterload and can improve cardiac output. However, tight control of blood pressure is recommended as

myocardial hypoperfusion can occur with hypotension. Epidural or low dose spinal as part of a combined spinal-epidural (CSE) offers the best balance and allows for titration to effect.

The anaesthetic goals, whether under RA or GA, are

- Avoid tachycardia.
- Minimise the effects of negatively inotropic drugs (e.g. anaesthetics).
- Prevent increases in afterload.
- Maintain adequate preload.

ANAESTHETIC COMPLICATIONS

The rate for anaesthetic-related deaths in pregnancy in the UK is less than 0.1 cases per 100,000 deliveries. The key messages from the MBRRACE report were

- Follow-up all patients with PDPH by the anaesthetic team; inform the patient's GP (one woman died from an undiagnosed cerebral vein thrombosis and another from an undiagnosed subdural haematoma).
- Anaesthetists should take part in multidisciplinary drills for airway crises such as bronchospasm, mechanical obstruction and failed intubation.
- Fixation errors or task focussing remain a common cause of patient harm during critical incidents.

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Amniotic fluid embolism, Chapter 30
Cardiomyopathy, Chapter 2
Obstetric anaesthesia – general principles, Chapter 12

13

Urology

MATTHEW JAMES JACKSON

ANAESTHESIA FOR UROLOGICAL SURGERY – GENERAL PRINCIPLES

A wide spectrum of patients presents for urological surgery; procedures range from minor day-case operations to large open surgeries. The majority of patients are elderly with multiple comorbidities. Most surgeries are undertaken to treat or investigate cancer. The incidence of urological cancer is increasing. As techniques advance, an increasingly elderly population is presenting for operative management. Reassuringly survival rates for most conditions are also improving. Many patients presenting for urological surgery will require repeat procedures. Urologists have historically used endoscopic techniques through natural orifices. They are pioneering new minimally invasive techniques and are at the forefront of robot-assisted surgery.

With an increasing case load, the need for anaesthetists skilled in providing anaesthesia for urological surgery is increasing. The challenges of urological anaesthesia are typically a high turnover list with high-risk patients undergoing low-risk surgical procedures; or medium-risk patients undergoing long and complex surgeries.

PREOPERATIVE ASSESSMENT

Patients are typically:

- Male
- Over 50 years of age
- High incidence of chronic disease

Preoperative assessment will be dictated by the nature of the surgery. Patients undergoing transurethral procedures with a rigid scope are often repeat attenders with multiple comorbidities. Assessment should include history and examination to identify comorbidities and consideration of further investigations to quantify the severity of disease. Where patients have already had a similar procedure, assessment should focus upon any deterioration of health in the intervening time. In high-risk cases, the merits of nonsurgical management or undertaking

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the procedure with a flexible scope and no anaesthesia should be discussed with the surgeon.

Patients undergoing radical surgeries require a through preoperative assessment. CPET is increasingly used to risk stratify patients and plan an appropriate level of perioperative care. Comorbidities should be optimised, but should not unduly delay tumour excision.

Genitourinary pathologies may bleed insidiously or obstruct the tract; therefore, preoperative U&E and FBC are mandated in most patients. Both radical surgeries and transurethral resections can result in large blood loss; patients should have blood samples taken for group and save and/or cross-match depending on local guidelines.

Premedication is rarely required. In the majority of cases, a clear explanation of the procedure will alleviate anxiety. When this does not suffice, a short-acting benzodiazepine may be used.

PERIOPERATIVE MANAGEMENT

Antibiotics are often requested. This should be according to local hospital protocols. Often gentamicin is chosen, which should be used with caution in patients with pre-existing renal failure.

Many procedures are performed in the lithotomy or Lloyd–Davis position. A head-down tilt may be requested. In elderly patients care should be taken when moving the legs as limited movement due to joint disease or risk of dislocating a prosthetic joint are common.

Laparoscopic urological surgery often requires a steep head down positioning and insufflation with carbon dioxide, the physiological sequelae of which should not be ignored. There is splinting of the diaphragm resulting in atelectasis, reduction in FRC and ventilation-perfusion mismatch. Raised intracranial pressure, raised intraocular pressure, reduced venous return and increased systemic vascular resistance are also possible. Oedema of the head and neck is common and can result in cerebral oedema, laryngeal oedema and optic disc oedema. Passive regurgitation, air embolism and bradycardia are additional complications.

Major urological surgery can last for up to about 6 hours. Due to delicate skin and reduced muscle mass in the elderly, these patients are at high risk of pressure sores. Extreme care should be taken to protect and check pressure points.

Where irrigation is used in a transurethral procedure, the irrigation fluid should be warmed to body temperature before infusion. For larger surgeries, warmed intravenous fluids and at least one body warming device should be used throughout the operation.

These patients are moderate to high risk of DVT. Due to the risk of bleeding and common use of neuraxial techniques, low molecular weight heparin is usually postponed until after the surgery is complete. Graduated compression stockings and intermittent calf compression should be used.

ANAESTHETIC TECHNIQUE

Minor procedures are normally undertaken spontaneously breathing on a laryngeal mask or under spinal anaesthesia. There is no general evidence for one technique over the other; however, relative merits should be considered for individual patients.

Major procedures require general anaesthesia, mechanical ventilation through an endotracheal tube and invasive monitoring. Wide bore vascular access should be placed. Tranexamic acid has been used in many procedures to reduce blood loss. The NICE has approved the use of cell salvage for radical prostatectomies and cystectomies.

There are early signals in the research literature that certain anaesthetic techniques may reduce the risk of recurrence in cancer surgery including total intravenous anaesthesia, epidural anaesthesia and avoidance of homologous blood transfusion.

MONITORING

Routine AAGBI minimum standards are essential. Additionally, in the following circumstances additional monitoring is required:

- Inspired and expired oxygen, carbon dioxide, nitrous oxide and volatile anaesthetic agent (if used) and airway pressure, when general anaesthesia is used.
- Temperature, when the time from induction to emergence is anticipated to be over 30 minutes duration.

- Peripheral nerve stimulator, if neuromuscular blocking drugs are used.
- Depth of anaesthesia monitoring when using total intravenous anaesthesia (TIVA) in the presence of a muscle relaxant.

For patients with a precarious cardiovascular system, or those undergoing major surgery an arterial catheter is essential for frequent sampling and haemodynamic monitoring. Indications for the placement of a central venous catheter are few. Cardiac output monitoring may be used to guide fluid management in larger surgeries.

COMPLICATIONS

Complications can be as a result of pre-existing comorbidities or intraoperative events. Perioperative myocardial infarction is not uncommon in this patient cohort. Postoperative confusion is frequently seen. Care should be taken with drug administration in patients with compromised renal function.

Where a hypotonic, nonionic irrigation fluid is used there is a risk of developing TURP syndrome. Most urological surgeries have the potential for massive blood loss. This could either be insidious ooze from the operative site or perforation of a major blood vessel. Management should follow the local major haemorrhage protocols.

POSTOPERATIVE MANAGEMENT

Many patients undergoing simple procedures require a period of postoperative bladder irrigation, which prevents same day discharge. Patients undergoing large procedures may benefit from a period on HDU. Enhanced recovery packages are frequently used by centres with a high caseload.

ANALGESIA

Transurethral procedures are relatively painless, the urinary catheter being responsible for the majority of the discomfort. For most, simple analgesics suffice. Use caution when prescribing NSAIDS in this patient group.

Major surgery requires a robust multimodal approach to postoperative pain relief. For open surgery, an epidural delivering local anaesthetic and opioid is the standard technique. Rectus sheath catheters, wound catheters, paravertebral catheters and local infiltration with an opiate PCA are alternative techniques. Laparoscopic and robot-assisted approaches are less painful; many of these operations do not need epidural analgesia.

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Nephrectomy, Chapter 13
Cystectomy, Chapter 13
Transurethral resection of the prostate, Chapter 13
Percutaneous nephrolithotomy, Chapter 13
Radical prostatectomy, Chapter 13

Penile surgery, Chapter 13
Anaesthesia for the elderly, Chapter 25
Massive haemorrhage, Chapter 30

CYSTECTOMY

Bladder cancer is the most common malignancy of the urinary tract; the incidence is four times higher in men than in women. Tumours are divided into those which invade the muscle (muscle-invasive bladder cancer) and those that do not (non-muscle-invasive bladder cancer). Trans-urethral resection of bladder tumour (TURBT) is the first-line treatment for patients with non-muscle-invasive bladder cancer. This procedure is similar to trans-urethral resection of the prostate (TURP), as is the anaesthetic management. During endoscopic resection, if the tumour lies over the obturator nerve electrocautery can cause adduction of the lower limb–muscle relaxation may be required. The high rate of recurrence and progression after TURBT necessitates the use of adjuvant treatments, and repeat cystoscopies.

Radical cystectomy is predominantly performed for muscle-invasive bladder cancer; it is occasionally performed if a tumour in a surrounding structure is invading the bladder. Preoperative radiation may be used prior to the surgery to shrink the tumour and is associated with increased intraoperative bleeding risk. Radical cystectomy includes bilateral pelvic lymphadenectomy in addition to removal of the prostate and seminal vesicles in men and the uterus, ovaries and part of the anterior vaginal vault in women. The flow of urine needs to be diverted. This can be achieved by creating either a conduit or pouch from an isolated portion of the gastrointestinal tract into which the ureters are implanted.

Cystectomy is traditionally performed via an extensive midline incision. Laparoscopic and robot-assisted approaches are becoming increasingly common due to decreased blood loss, reduced postoperative pain, early return of bowel function, and shorter hospital stay.

PREOPERATIVE ASSESSMENT

Patients for cystectomy are generally over 50 years of age, smokers and have had previous anaesthetics for cystoscopy.

- Systematic review and optimization of chronic disease states is important.
- An association between long-term cigarette smoking and bladder cancer; screen for other smoking-related conditions.
- Check full blood count and ensure that blood is available for transfusion. Bladder tumours bleed insidiously and a low starting haemoglobin is not uncommon. Large intraoperative blood loss occurs occasionally and the risk is higher in patients who have received preoperative radiotherapy.
- Tumours may obstruct the ureters causing hydronephrosis. Request urea and electrolytes to evaluate renal function.
- Bowel preparation with powerful laxatives may be required. Ensure that the patient is well hydrated and has maintained adequate oral intake of clear liquids the day before surgery. An enhanced recovery approach is used at many centres.

PERIOPERATIVE MANAGEMENT

- These are long operations often in frail patients.
- Maintain body temperature using warm operating theatre, warming blanket, warm air duvet, fluid warmer and humidification of inspired gases.
- Positioning is important. In open surgery, male patients are positioned supine with the legs apart with gentle hyperextension. Female patients are placed in lithotomy position to allow for access to the perineum. All pressure points should be padded.
- Minimally invasive approaches use steep Trendelenburg.

ANAESTHETIC TECHNIQUE

- General anaesthesia alone or combined with epidural anaesthesia is usual.
- Controlled hypotension may reduce intraoperative blood loss and transfusion requirements.
- Spinal or epidural anaesthesia may facilitate induced hypotension but can produce hyperperistalsis due to unopposed

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parasympathetic activity and hinder the construction of the urinary reservoir.

- Measure CVP due to the potential for excessive blood loss and inability to measure urine output during the first part of the operation.
- The patient should be well hydrated to maintain a vigorous urine output once the ureters are implanted.
- As with any long procedure, the use of thromboembolic prophylaxis should be used.

MONITORING

- Routine minimal monitoring
- Invasive arterial pressure
- Central venous pressure (CVP)
- Oesophageal Doppler

POSTOPERATIVE MANAGEMENT

Patients may be cared for on a general ward or on HDU depending upon individual circumstances. The management of postoperative pain is best achieved via an epidural infusion of low-dose local anaesthetic and opioids.

The prolonged contact of urine with bowel mucosa may produce significant metabolic disorders including electrolyte abnormalities, altered sensorium, osteomalacia, recurrent urinary tract infections and formation of calculi. The electrolyte abnormalities seen depend on the segment of bowel used. Treatment consists of administering alkalinising agents or chloride transport blockers. As hyperkalaemia usually is present, treatment must involve both correction of the acidosis with bicarbonate and replacement of potassium.

This is a major operation. The operative mortality is 1%–3%. Continuous epidural analgesia may contribute to a lower postoperative mortality. The overall complications rate after radical cystectomy and urinary diversion may be as high as 25%–35%.

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CROSS-REFERENCES

CYSTOSCOPY

Cystoscopy allows the visualisation and biopsy of the urethra and bladder mucosa via either a flexible or a rigid fibre-optic scope. Flexible cystoscopy is normally an outpatient procedure. Lubricant gel containing local anaesthetic is applied to the urethral meatus and the scope passed into the bladder. The bladder is not distended, and therapeutic interventions are limited. Rigid cystoscopy requires distention of the bladder, achieved with a constant stream of irrigation fluid. It is uncomfortable and is therefore undertaken in theatre under anaesthesia.

Rigid cystoscopy is used for bladder biopsies, resection of bladder tumours, extraction of stones and placement of ureteral catheters (stents). Transurethral laser lithotripsy under local anaesthesia appears to be safe and effective for large bladder calculi. TURP and ureteroscopy are performed with modified cystoscopes.

Cystoscopy is a short procedure. Patients often return regularly for several years to monitor the lesion under investigation. The management of carcinoma of the bladder by radiotherapy and intravesical chemotherapy ensures a steady supply of

patients for this procedure. These patients are often very knowledgeable about the procedure, and their wishes, particularly regarding premedication and regional blocks, should be heeded. A small number of patients have disease that cannot be controlled and proceed to cystectomy.

PREOPERATIVE ASSESSMENT

- Assess cardiovascular and respiratory systems.
- Assess renal function: urea and electrolytes.
- The lesion under investigation may be an insidious source of blood loss; request an FBC.
- If repeat cystoscopy, ask if there has been any change in health status since the last visit and check previous anaesthetic records.
- Assess suitability for day-case anaesthesia.

In high-risk patients, a discussion with the surgeon regarding the relative risks and benefits of using a flexible cystoscope is advised.

PERIOPERATIVE MANAGEMENT

Both general anaesthesia (using a supraglottic airway and spontaneously ventilation) and spinal anaesthesia are used. There is little evidence that either approach is superior. Spinal anaesthesia is preferred in severe respiratory disease. The patient will be required to lie flat or head down during the operation. A block to T10 should be achieved. General anaesthesia routinely uses propofol and a short-acting opiate followed by inhalational agent or TIVA. Prophylactic antibiotics are often required.

The procedure is performed in the lithotomy or Lloyd–Davis position. When lifting the legs take care not to dislocate previous joint replacements.

POSTOPERATIVE MANAGEMENT

Diagnostic and check cystoscopies where no resection has taken place do not result in significant postoperative pain. Paracetamol may be adequate. If a biopsy has been taken, intraoperative supplementation with an intravenous analgesic will usually be sufficient. Simple analgesics with an antiemetic are normally adequate.

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CROSS-REFERENCES

Diabetes mellitus, Chapter 6
The elderly patient, Chapter 25
Day-case surgery, Chapter 25
Preoperative assessment of pulmonary risk, Chapter 25
Complications of position, Chapter 30

NEPHRECTOMY

Renal cell carcinoma (RCC) is responsible for over 90% of tumours. These present between the fourth and seventh decade. The tumour commonly spreads to bone, lung and brain and can extend along the inferior vena cava (IVC) in 10% of cases. Paraneoplastic syndromes (hypertension, polycythaemia, hypercalcaemia, nonmetastatic hepatic dysfunction) are found in 20% of patients with renal cell carcinoma. In addition to cancer, nephrectomy may also be performed for hydronephrosis, trauma, shrunken kidney, hypertension chronic infection and in living donors.

Nephrectomy involves the removal of a kidney with or without part of the ureter. In cases of radical nephrectomy the renal fascia, adrenal gland and regional lymph nodes are removed. Nephrectomies may be open, laparoscopic or robot-assisted. Thermal ablative therapies or partial resection are used to treat smaller tumours and preserve renal function in high-risk patients.

The open operation is carried out via a dorsal, anterior subcostal, flank, midline or thoracoabdominal incision. The laparoscopic approach is gaining popularity. While operating time is longer than in open approaches, it is associated with less pain and quicker recovery times. It may be performed transperitoneal or retro-peritoneal; the patient is positioned laterally. In cases of caval extension, input from vascular and cardiac surgeons may be required.

The majority of patients with renal cell carcinoma are anaemic and there is potential for considerable blood loss with all approaches.

PREOPERATIVE ASSESSMENT

- *Exclude associated conditions* – Anaemia, paraneoplastic syndromes, respiratory disease (cigarette smoking is a major risk factor), hypertension, hypercalcaemia.
- *Perform a systematic review* – CPET may be helpful in high risk patients.
- Evaluate the degree of renal impairment.
- Establish the extent of the lesions and the type of ablative therapy.
- Patients with suspected or proven urinary tract infection should receive 48 h of antibiotic therapy.
- Ensure the availability of blood.

When a large tumour is to be resected, preoperative blood transfusion may be required in anaemic patients.

PERIOPERATIVE MANAGEMENT

As there are several surgical approaches, it is important to discuss with the surgeon which position is to be used. Generally, the patient is placed laterally on the operating table with the side of operation uppermost. The table is flexed such that the head and feet are both lowered to facilitate surgical access. Careful positioning of the patient is important as it can be lengthy and the lateral position has a high risk of nerve and soft tissue damage.

In case of radical nephrectomy for excision of tumour thrombus, a thoracoabdominal approach may be necessary. Cardiopulmonary bypass to prevent embolization may be required.

ANAESTHETIC TECHNIQUE

General anaesthesia is required with endotracheal tube and controlled ventilation. The lateral position results in a ventilation – perfusion mismatch. In addition, there is a risk of surgical disruption of the pleura during the dissection.

Arterial line and wide-bore venous cannulae should be placed on the side of the tumour and well secured. Blood should be readily available. Tranexamic acid can be used to reduce bleeding; cell salvage is recommended, with a leuco-depleting filter used to administer salvaged red cells. Fluids should be warmed and the patient placed on or under a warming device. Thromboprophylaxis is necessary – graduated compression stocking and intermittent calf compression.

In renal cell carcinoma with thrombus, anaesthetic management is complex and may require massive blood transfusion (up to 50 units of packed red blood cells, plasma and platelets).

MONITORING

The potential for massive blood loss is an indication for invasive monitoring. Central venous line is necessary in selected patients. In addition to routine monitoring, blood loss, temperature and urine output are useful.

COMPLICATIONS

Pneumothorax may occur during thoracoabdominal or flank incisions. At the end of the procedure, several large-volume breaths may be given to attempt to detect any pleural leak. If there is a pneumothorax, the pleural injuries should be repaired and a chest tube or drain inserted.

The physiological effects of a pneumoperitoneum or pneumoretroperitoneum are seen in laparoscopic approaches.

In patients with partial nephrectomy, complications include haemorrhage, urinary fistula, uretheral obstruction and renal insufficiency.

POSTOPERATIVE MANAGEMENT

There is a risk of atelectasis in the dependent lung and postoperative physiotherapy is essential. Most patients benefit from a period of level one care for the first 12–24 hours postoperatively. Enhanced recovery principles can be used in high volume centres.

Long-term outcome depends largely on the aetiology of the renal damage and the type of procedure. After radical nephrectomy, postoperative

complications occur in approximately 20% of patients and the operative mortality rate is approximately 2%.

ANALGESIA

Open nephrectomy is a painful operation and requires optimal multimodal pain relief. Paracetamol should be used in all patients with NSAIDs where renal function allows. Epidural analgesia via a low thoracic or high lumbar catheter is used to deliver local anaesthetic and low-dose opiates. If an epidural is not possible, intercostal nerve blocks from T9 to T12, paravertebral blocks or wound-bed catheters in addition to patient controlled analgesia (PCA) may be used.

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CROSS-REFERENCES

Blood transfusion, Chapter 30
Complications of position, Chapter 30
Kidney transplant, Chapter 23

PENILE SURGERY

Penile surgery includes a wide range of surgeries that are performed for medical, religious and cosmetic reasons. Circumcision is the most common surgery; other surgeries are briefly considered.

Circumcision is the surgical removal of the foreskin; it is commonly performed in children and young adults. It is used when nonsurgical measures have failed to manage phimosis, recurrent balanitis, paraphimosis, balanitis xerotica obliterans and penile cancer. It may also be performed for religious reasons and has been shown to reduce the risk of HIV transmission.

Penile cancer is rare, occurring in men over 60 years old; it is associated with chronic inflammatory conditions of the penis. The most common procedure is local resection using a carbon dioxide laser. Larger tumours require wide local incision, subtotal and total excision of the penis. Various grafts may be used to cover the deficit or staged reconstruction of the penis may be considered.

Cosmetic surgery is performed to improve the appearance of the penis. Penile lengthening is most commonly achieved by dividing the suspensory ligament and then grafting skin to cover its new length. Penile girth can be enhanced by injecting liposuction fat or by forming a flap.

PREOPERATIVE ASSESSMENT

With the exception of patients presenting for penile cancer surgery, this patient group tends to be young and fit. The preoperative visit should be used to elicit evidence of multisystem disease.

PERIOPERATIVE MANAGEMENT

For the majority of patients, penile surgery will be undertaken as a day case.

Patients are positioned supine. The surgery is short.

A spontaneously breathing technique using a laryngeal mask is appropriate for most patients. General anaesthesia may be supplemented by a penile block or local anaesthetic infiltration. Adrenaline should be avoided due to the risk of causing ischaemia. In children, a caudal injection is performed asleep to provide postoperative analgesia.

POSTOPERATIVE MANAGEMENT

The majority of patients can return to the day-case unit and be discharged with simple analgesics several hours after surgery.

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CROSS-REFERENCES

General principles of urological surgery, Chapter 13
Blocks: penile block, Chapter 29
Day case surgery, Chapter 25

PERCUTANEOUS NEPHROLITHOTOMY

Percutaneous nephrolithotomy is a procedure whereby stones in the renal tract are removed with a rigid or flexible endoscope via ultrasound-guided puncture and fluoroscopy-controlled placement of the endoscope. A guidewire is inserted through a hollow needle and advanced into the collecting system. Then tract dilation is performed over the guidewire. At the completion of access tract dilation, a working sheath is left in place to accommodate the endoscope and drain the irrigation fluid. The procedure is particularly indicated in patients with staghorn calculi, and lower pole calculi larger than 10 mm. Patients with stones resistant to extracorporeal shock wave lithotripsy should also be treated by this process. Small calculi are removed through the endoscope under direct vision using a forceps or a stone basket. Stones larger than 1 cm require fragmentation by a lithotripsy device. The most efficient are the ultrasonic and the pneumatic rigid lithotripters. Morbidly obese patients in whom shock wave lithotripsy is impractical or technically impossible may also need to be treated in this way.

Ureteroscopy via the transurethral route allows an alternative approach to investigate ureteric disease and undertake treatments. The scope is passed transurethrally and navigated into the ureter. It may be used to inspect and biopsy the ureter as well as remove smaller distal stones and place ureteric stents. Patients should have renal function evaluated prior to the procedure.

PREOPERATIVE ASSESSMENT

- Exclusion of commonly associated medical conditions (Crohn's disease, processed diet, metabolic syndromes).
- Patients may have compromised renal function due to obstructive nephropathy: review renal function.
- Bacteriologic evaluation of the urine. Urinary calculi may harbour bacteria.
- Antibiotic prophylaxis according to local guidelines.
- Correction of an existing coagulopathy.
- Antiplatelet medication should be discontinued 7 days before procedure.

PERIOPERATIVE MANAGEMENT

The patient is usually placed in the prone position with the stone-containing side elevated:

- Protect the eyes, shoulders, knees, elbows.
- Place arm with intravenous access above the head (beware of brachial plexus strain).
- The other arm with the BP cuff may be placed by the side.
- Place a pillow under the chest and pelvis to free the abdomen for ventilation.
- Place a pad under the flank to prevent a mobile kidney from rotating anteriorly in the prone position.
- Turn the head to the side to be punctured in order to prevent neck strain.

In high-risk patients, in order to minimize the haemodynamic and respiratory changes, a full lateral or a supine anterolateral position may be used. It prevents the discomfort and ventilation difficulties of the prone position, particularly in obese patients.

ANAESTHETIC TECHNIQUE

General anaesthesia is necessary when a lengthy procedure is planned. For shorter procedures, a spontaneously breathing technique with a laryngeal mask is suitable. Muscular paralysis is usually required because the patient will be placed prone or lateral and coughing must be avoided during renal

puncture. An armoured (reinforced) tracheal tube should be used if placed prone.

Spinal anaesthesia has been used safely in selected patients. Intrathecal low-dose bupivacaine and fentanyl offers reliable neuraxial block. Combined spinal-epidural anaesthesia with a sensory block above T6 is an attractive alternative to general anaesthesia. It has the advantage of shorter hospital stay, better patient satisfaction and superior postoperative pain relief.

Local anaesthesia from delivering the local anaesthetic through the access track combined with sedation is safe and effective in selected patients.

COMPLICATIONS

Bleeding is the most significant complication. The kidney is a very vascular organ and tears in the parenchyma may occur if the rigid scope is not handled with care. Bleeding requiring transfusion is rare. Most reports quote 3%. About 0.5% of cases may require balloon tamponade of the tract or arterial embolization.

Extravasation of irrigation fluid may result from a tear in the pelvicalyceal system. It is important, therefore, that normal saline is used as the endoscopic irrigation fluid. Water and glycine can cause fluid intoxication because they are absorbed from the peritoneum.

Pleural complications can result from an intercostal puncture to reach an upper calyceal calculus. The pleura may be entered and either a minor pleural reaction is seen or following endoscopy there could be a massive collection of irrigation fluid and air within the thoracic cavity.

Infection is the most serious complication and may be seen in 0.3%–2.5% of cases. Infected stones may be disintegrated at percutaneous nephrolithotomy, releasing bacteria into the urine and potentially into the bloodstream. Bacteraemia is unavoidable, but the time of the endoscopy should be limited to 1 h for a large infected stone and 1.5 h in case of noninfected stone. If Gram-negative septicaemia is suspected, the patient should be treated aggressively immediately.

POSTOPERATIVE MANAGEMENT

Intravenous fluids should be given to increase urine output and flush out any gravel via the nephrostomy

left in situ. Analgesia with opioids and NSAIDs if renal function is normal. Peritubal infiltration of 0.25% bupivacaine solution is efficient in alleviating postoperative pain.

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CROSS-REFERENCES

Blood transfusion, Chapter 30
Complications of position, Chapter 30
TUR syndrome, Chapter 30

RADICAL PROSTATECTOMY

Prostate cancer affects older males. The tumour can spread to surrounding structures and metastasize to bone and lung. The cancer is often reliant upon an androgenic drive for growth and malignant transformation. Treatment options include watchful waiting, radiotherapy, hormonal therapy and surgery. There remain controversies in the optimal management of this disease; multidisciplinary teams must consider

the tumour characteristics on the background of the patient's wishes and other comorbidities when considering the treatment pathway.

Radical prostatectomy is performed for malignant disease via an open, laparoscopic or robot-assisted technique. Open surgery is usually via the retropubic approach; the perineal approach is rarely used. Laparoscopy and robot-assisted are transperitoneal. The prostate is removed together with pelvic lymph nodes, seminal vesicles, ejaculatory ducts and part of the bladder neck.

PREOPERATIVE ASSESSMENT

During the preoperative visit:

- Identify and assess chronic disease.
- Assess suitability for enhanced recovery.
- Assess suitability for spinal or epidural analgesia. This is particularly useful in conjunction with general anaesthesia because it provides excellent postoperative analgesia, reduces perioperative blood loss and decreases the quantity of inhalational agent required.
- Check haemoglobin and ensure blood transfusion availability, as radical prostatectomy is often associated with significant operative blood loss.

PERIOPERATIVE MANAGEMENT

Patient positioning requires care since surgery is often lengthy. The patient is usually placed supine in a hyperextended position which places the pubis above the head. A steep Trendelenburg position is required for the laparoscopic approach.

ANAESTHETIC TECHNIQUE

The combination of epidural and general anaesthesia with a muscle relaxant and volatile agent is a suitable technique for open surgery. For minimally invasive surgery, an endotracheal tube is mandated; however, an epidural is not required. The insufflated carbon dioxide spreads into the retroperitoneal space and increases the intra-abdominal and intra-thoracic pressures. In combination with the steep

Trendelenburg position, this can lead to reduced cardiac and respiratory performance in addition to head and neck swelling including cerebral and laryngeal oedema.

Retropubic prostatectomy can be performed under neuraxial analgesia alone if a T6 sensory level is achieved.

The intraoperative blood loss varies with the length of surgery and grade and stage of malignancy. In some cases, transfusion up to 6 units could be necessary. It has been suggested that fibrinolysins, which exacerbate bleeding, are released by prostatic handling. In case of laparoscopic approach, the amount of bleeding and need for transfusion are much reduced. Less blood loss and a lower frequency of pulmonary emboli are associated with regional anaesthesia. Because of the potential for massive blood loss, two large-bore intravenous cannulae, tranexamic acid and cell salvage are advisable.

Elasticated stockings and intermittent calf compression are used for deep vein thrombosis prophylaxis.

Body temperature should be maintained with a warming mattress, warm air overblanket, warmed intravenous fluids and a ventilator circuit humidifier.

Antibiotic prophylaxis according to local guidelines.

In addition to routine monitoring, invasive BP, core temperature, neuromuscular function and fluid balance should be monitored.

POSTOPERATIVE MANAGEMENT

When compared with the open surgical approach, laparoscopy is associated with shorter operating times, lower urinary leakage rates, lower stricture rates, lower blood loss and less pain. If an epidural infusion is already in situ, then a multimodal approach using an infusion of local anaesthetic and an opioid will provide excellent analgesia for open surgery. TAP block and penile block reduce opiate requirements when an epidural has not been placed.

Postoperative blood loss and urine output should be monitored. Thrombophlebitis with pulmonary embolism is a major cause of postoperative mortality and low molecular weight. Heparin should be used postoperatively.

CPET can be used to select the level of postoperative care; most patients will be able to return to

general ward care within 24 hours of the operation. Principles of enhanced recovery have been successfully employed in this patient group.

With good case selection, the 5-year survival is 95%. Operative mortality is low.

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CROSS-REFERENCES

The elderly patient, Chapter 25
Blood transfusion, Chapter 30
Thrombotic embolism, Chapter 30
Preoperative assessment of pulmonary risk, Chapter 25
Complications of position, Chapter 30

TRANSURETHRAL RESECTION OF PROSTATE (TURP)

Benign prostatic hyperplasia (BPH) occurs in over 40% of men aged over 60 years. Resection of the prostate is reserved as second-line treatment for BPH for symptoms resistant to medical management. The majority of prostatectomies are performed endoscopically using the transurethral route, with open procedures reserved for cancers and exceptionally large prostates.

To perform the operation, the patient is placed in the lithotomy position and a modified cystoscope (a resectoscope) is used to shave away the prostate at the bladder neck. As the body of the prostate is removed, veins are exposed, but the capsule is maintained. The exposed veins can bleed, causing significant blood loss; they can also absorb large amounts of irrigation fluid resulting in TURP syndrome.

Resectoscopes traditionally use mono-polar electrocautery. This requires a nonionic solution to be used as irrigation to prevent current dissipation: 1.5% glycine solution is used. This solution, in the presence of open veins, is responsible for TURP syndrome. Bipolar electrocautery and lasers are replacing the mono-polar technique. They result in better haemostasis and reduce the absorption of irrigation fluid.

PREOPERATIVE ASSESSMENT

- Patients have a high incidence of cardiopulmonary problems. The ability of the patient to manage an increased circulating volume as a result of absorbing irrigation fluid should be considered.
- In patients with cardiovascular comorbidities, anticoagulant therapy is common and the risk for bleeding is increased. The anticoagulation medications may preclude neuraxial anaesthesia.
- The operation may be performed as a repeat procedure; changes in health during the intervening time should be assessed.
- Patients may present with haematuria or may have longstanding obstruction. U&E and FBC should be check preoperatively.
- Prostatic bleeding can be difficult to control through the cystoscope. Routine group and save is recommended. Cross-matched blood should be available for the patients with large glands.

PERIOPERATIVE MANAGEMENT

The procedure is performed in the lithotomy or Lloyd–Davis position. Irrigating fluid is warmed to maintain patient core temperature.

All aspects of laser safety should be adhered to if a laser technique is used. This includes protective eyewear for staff and patients, blinds for windows and signs on doors. Antibiotic administration according to the hospital policy (usually gentamicin or a cephalosporin).

ANAESTHETIC TECHNIQUE

Spinal anaesthesia is commonly used, although a number of patients prefer to be asleep. There several considerations when performing a spinal technique:

- A block to T10 is required.
- Patients often have chest disease and may benefit from not having a general anaesthetic.
- In awake patients, the evaluation of mental status is the best monitor of the onset of the TURP syndrome and of bladder perforation.
- Spinal anaesthesia reduces central venous pressure, potentially resulting in greater absorption of irrigating fluid than with general anaesthesia.
- Degenerative changes in the spine of elderly patients may make neuraxial anaesthesia technically difficult. Vertebral metastasis in patients with carcinoma represents a contraindication to regional anaesthesia.

If a general anaesthesia is used, a spontaneously breathing technique using a laryngeal mask is usually appropriate.

Vigilance for development of the TURP syndrome is required.

MONITORING

- Routine monitoring in both general and spinal anaesthesia
- Mental status if the patient is awake
- Arterial line in these with precarious cardiovascular function

POSTOPERATIVE MANAGEMENT

TURP syndrome can develop intraoperatively or up to 24 hours postoperatively. Postoperative full blood count and renal function test are useful to screen for anaemia (which may be due to haemodilution or excessive bleeding) and hyponatremia. The initial postoperative period is not overly painful with the catheter being the major irritant; regular simple analgesia will suffice.

OUTCOME

The reported hospital mortality is 0.2%–6% and may be as low as 0.5%–1% in specialist centres. There is evidence of increased intermediate and long-term mortality and morbidity with TURP compared with open prostatectomy, and with other minimally invasive surgery in this age group. Increased morbidity may be found after resections exceeding 90 min, gland size greater than 45 g and age older than 80 years.

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CROSS-REFERENCES

The elderly patient, Chapter 25
Blood transfusion, Chapter 30
Fluid and electrolyte balance, Chapter 30
TURP syndrome, Chapter 30
Complications of position, Chapter 30

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Neurosurgery

ELEANOR CHAPMAN

ANAESTHESIA FOR INTRACRANIAL NEUROVASCULAR SURGERY

Patients may require neurosurgery for treatment of cerebral aneurysms, arteriovenous malformations and other vascular abnormalities, or following intracranial haemorrhage.

CEREBRAL ANEURYSMS

Most patients present acutely following aneurysm rupture with the signs and symptoms of subarachnoid haemorrhage (SAH). Unruptured aneurysms are increasingly being detected incidentally on cranial radiological investigations but can also present with symptoms related to mass effect.

NEUROSURGICAL TREATMENT

Endovascular techniques (coiling) have been shown to be preferable to an open approach (clipping) for patients with ruptured aneurysms. Open neurosurgical clipping has thus become increasingly uncommon unless the aneurysm

- Has a wide neck or difficult anatomy
- Is too distal to reach endovascularly

Although mortality and disability have been shown to be reduced at 1 year, long-term coiled aneurysms are 8 times more likely to rebleed. Consideration should be taken in the under 40s to opt for open neurosurgical clipping. The optimum timing for securing a ruptured aneurysm is still unclear, with little evidence that performing surgery within 24 hours confers any benefit over 24–72 hours.

PREOPERATIVE ASSESSMENT

- Patients with poor grade SAH may already be intubated and ventilated on ICU.
- If conscious, a neurological exam needs to document the GCS, cranial nerve involvement and any sensory or motor deficit.
- Patients should have their headache controlled with appropriate analgesia.

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- Continue nimodipine and anticonvulsants where necessary.
- Optimise cardiac function; a preop ECG is mandatory.
- Extremes of blood pressure should be avoided; keep MAP <110 mmHg and SBP <160 mmHg while ensuring a CPP of 60 mmHg.

INTRAOPERATIVE MANAGEMENT

Anaesthetic management is similar to that of any neurosurgical procedure involving raised ICP but particular attention should be paid to:

- Careful induction avoiding surges in blood pressure where an increase in transmural pressure in the affected artery could precipitate a further rupture. Conversely, hypotension may worsen ischaemia and cause infarction.
- In addition to standard monitoring, invasive blood pressure monitoring is essential. Frequently central venous access is inserted if the patient is likely to need hypertensive therapy postoperatively. Temperature monitoring is advisable as is a urinary catheter as a lot of contrast and flushes will be used in coilings.
- Propofol TIVA, sevoflurane or desflurane accompanied by remifentanil to keep the MAC <1.0 are appropriate choices for maintenance.
- Maintain normocarbia, normoglycaemia and normothermia.
- Cardiac dysfunction and arrhythmias are common and should be managed with correction of electrolyte imbalances in the first instance.
- Position supine for all coiling procedures and anterior circulatory aneurysm clipping but for posterior aneurysms the patient will need to be in the park bench or prone position.
- During clipping the surgeon might use a temporary clip while dissecting around the aneurysm to reduce the risk of further rupture. Whilst these clips are in place the surgeon may ask for some cerebral protection in the form of mannitol 20% as a free radical scavenger, metabolic suppression with a bolus of thiopental or hypothermia.

INTRAOPERATIVE ANEURYSM RUPTURE

Intraoperative aneurysm rupture occurs most commonly as the neck is dissected. At this stage, a temporary clip can be used to stop haemorrhage from the main vessel. However, if the aneurysm ruptures as the dura is being opened, and the circle of Willis is not dissected, the situation will be uncontrolled. Under these circumstances, acute hypotension is essential to allow surgical access and control of haemorrhage. Blood pressure should be reduced only to a level that allows the surgeon to gain control under direct vision.

If a rupture is suspected while undergoing coiling, the goals are to both lower the blood pressure and to increase the coagulation by reversing any heparin. Techniques may be required to lower ICP. Once the bleeding is controlled, the blood pressure should be raised again to check for leaks and then proceed again with coiling.

CEREBRAL OCCLUSION AND VASOSPASM

During coiling, ischaemia may be noticed intra-operatively. This may be due to thromboembolism, arterial dissection, catheters, coil misplacement or vasospasm. Management should be to increase blood pressure to improve the contralateral flow. The radiologists may ask for IV antiplatelet therapy, heparin or thrombolytic therapy or may give nimodipine direct to the vasospasm themselves.

POSTOPERATIVE MANAGEMENT

- Grade I, II and most III SAHs with uneventful intraoperative course should be extubated using a technique to avoid coughing and surges in blood pressure (e.g. remifentanil with 5–10 mg boluses of labetalol where appropriate).
- It is prudent to send all patients to ICU as many will develop further complications such as delayed cerebral ischaemia or non-neurological complication.
- Grade IV and V patients should be transferred back to ICU with continued ICP monitoring and a sedation hold or trial of extubation taken in a timely manner.

ELECTIVE ANEURYSMS

Elective aneurysms are less unstable preoperatively but the procedures are essentially the same. The complications (vasospasm, rebleeding) are much less likely and following coiling, patients can usually be extubated and discharged to level one care.

ARTERIOVENOUS MALFORMATIONS

Arteriovenous malformations (AVMs) are congenital abnormalities of the vascular network in which abnormal connections between arteries and veins, without intervening capillary, result in a direct arterial-to-venous shunt and development of twisted dilated vessels. Approximately 5%–10% of AVMs present acutely following an SAH, but the majority present with seizures, headache or progressive neurological signs.

Many AVMs are now treated by staged radiological glue embolization and/or gamma knife. For 85%–95% this may be curative but, in others, surgical excision is required.

- Anaesthesia is very similar to that of aneurysm surgery.
- If open surgery is undertaken, there is great potential for bleeding and cross-matched blood should be available.
- Postoperatively sudden restoration of a chronically hypotensive area of brain can overwhelm the autoregulatory mechanisms resulting in microhaemorrhage and diffuse swelling: normal perfusion pressure breakthrough syndrome.
- Surges in blood pressure during extubation can be particularly problematic so an antihypertensive agent (labetalol 5–10 mg boluses) should be ready to use.
- In the postoperative period, blood pressure should be kept low-normal with labetalol or esmolol infusions where necessary.

INTRACRANIAL HAEMORRHAGE

Intracranial haemorhage (ICH) is a devastating cause of stroke. It consists of 10%–15% of all strokes and 85% of all intracerebral haemorrhages. The 30-day mortality is 40%–50% and of the survivors only 20%–25% are able to function independently at 6 months. The aetiologies for ICH are detailed in Box 14.1. Most patients present with a rapid onset neurological deficit associated with vomiting, head-ache, seizures and decreased level of consciousness, including coma.

Patients may need surgical intervention for:

- Evacuation of clot (particularly in peripheral clots and cerebellar haematomas)
- Insertion of EVD (for hydrocephalus due to ventricular extension)
- Decompressive craniectomy
- Insertion of catheter for thrombolysis (currently under trials)

Specific issues related to the management of ICH:

- Anaesthetic considerations are similar to other neurovascular procedures. Many patients will already be intubated and ventilated on ICU.
- Patients often have cardiac and other systemic complications related to the acute haemorrhage and also chronic hypertension; as such, invasive blood pressure monitoring is mandatory.
- While it is important to avoid extremely high blood pressure to avoid haematoma expansion, strict lowering of systolic blood pressure to 110–140 mmHg has failed to show benefit.
- Postoperative ICU care is often required and an ICP monitor should be inserted at the end of surgery if the patient will remain sedated.

BOX 14.1: Risk factors of primary intracranial haemorrhage

- Increasing age
- Male sex
- African/Asian descent
- Chronic hypertension
- Amyloid angiopathy
- Anticoagulation treatment
- Excess alcohol
- Recreational drugs, e.g. cocaine, ecstasy

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CROSS-REFERENCE

Subarachnoid haemorrhage, Chapter 3

ANAESTHESIA FOR MAGNETIC RESONANCE (MR) IMAGING

Images are produced by placing patients within a strong magnetic field and applying pulses of radio-frequency (RF) energy. This results in intermittent release of RF energy from hydrogen nuclei, which is detected by a series of close-fitting receiving antennae (coils). The RF signals are collected and interpreted by computer to produce extremely accurate images. The strength of the magnetic field used is measured in tesla (T). One tesla is equal to 10,000 gauss (G); the Earth's magnetic field is approximately 0.5–1.5 G. The most common MR scanners in clinical use range from 0.5 to 3 T, although the majority is 1.5 T. The patient is placed in the centre of a magnetic field within the bore of a magnet and, as a result, is enclosed within a narrow tube to which access is extremely limited. Newer designs include open and wider bore magnets that allow improved access and are less claustrophobic for awake patients.

MR scans are produced in sequences of up to 10 minutes and any movement during that time produces profound distortion of the final images. The aim of anaesthesia for MR is therefore to provide immobility while maintaining safety and patient comfort throughout.

SAFETY ISSUES IN MR UNITS

- The strong magnetic field poses the most important hazard related to anaesthesia and care of patients requiring MR.
- Ferromagnetic objects within the 50 G line will move and may be rapidly accelerated into the magnetic field becoming dangerous projectiles causing injury to anyone in their path, damage to equipment and interference with the MR image.
- Implanted ferromagnetic objects may move in the magnet or heat up, causing local tissue damage. This includes foreign bodies in the eye that may be dislodged during scanning, with the associated risk of vitreous haemorrhage.
- Non-ferromagnetic metals may heat up causing burns. They will also cause image artefact if they are adjacent to the area being scanned.
- Implanted pacemakers, defibrillators and other devices may be inactivated, reprogrammed, dislodged or revert to an asynchronous mode. Although implanted programmed devices are a general contraindication to MR, some may be scanned under strictly controlled conditions in specialist centres.
- Pregnant patients and staff should not enter the scanner during the first trimester.
- No patients or staff should be allowed past the 5 G contour line without going through a check for implantable devices or contraindications.
- Noise levels above 85 decibels may be generated by the scanner and can cause potential hearing loss in those having long scans. Staff and all patients should wear ear protection.

The most commonly used intravenous MR contrast agent is gadolinium dimeglumine (Gd-DTPA), which can cause nausea, vomiting and pain on injection. It has an extremely low incidence of anaphylactoid reactions. However, Gd-DTPA has been implicated in nephrogenic systemic fibrosis in patients with impaired renal function. An assessment of renal function should be performed if a patient's scan requires contrast.

PRACTICAL CONSIDERATIONS

- The MR unit is often isolated so must be self-sufficient in terms of anaesthesia and resuscitation equipment.
- The patient is placed inside a narrow bore tube, is relatively inaccessible and may be difficult to observe. Many MR units were not designed with anaesthesia in mind and space is often limited.
- Anaesthetic and recovery rooms should be placed adjacent to the scanner.
- Ferromagnetic items such as scissors, oxygen cylinders and laryngoscopes must never be taken into the scanning room.

MONITORING AND EQUIPMENT

Monitoring should conform to the same standards as anaesthesia or sedation in the operating theatre and allow the anaesthetist to view monitor and patient from outside the scanning room. Equipment is

- *MR safe* – No additional risks anywhere in the MR environment.
- *MR conditional* – Pose no known hazard in the MR environment with specified conditions of use. Field conditions that define the specified MR environment include field strength, spatial gradient, rate of change of magnetic field, RF fields and specific absorption rate.
- *MR unsafe* – Pose hazards in all MR environments but necessary to have in adjacent anaesthetic and recovery areas.

Additional considerations include:

- ECG cables must be shielded and special electrodes used. Furthermore, the magnetic field causes specific problems with ECG

interpretation, including MR-induced changes in the ST segment and T waves similar to those seen with hyperkalaemia or pericarditis.
- Pulse oximeters must use fibre-optic cables or be telemetric to avoid burns.
- There may be a delay in obtaining a capnograph signal and monitoring of airway pressures and gases because the sampling tubing will be longer than normal.
- Measurement of temperature is difficult but the technology is now available to measure peripheral temperature.
- An anaesthetic machine with piped gases should always be available inside the scanning room and this should be MR conditional. Non-MR conditional anaesthetic machines must either be bolted onto the floor or kept outside the 50 G line. All gas cylinders must be MR safe.

PATIENT ASSESSMENT

Patients who may require general anaesthesia during MR scanning are shown in Box 14.2. Screening is essential to exclude those who cannot enter the magnetic field, and this is conducted using a standard checklist. The exact make of an implantable device is required in order to assess its safety in the MR scanner. All patients with traditional pacemakers and internal defibrillators may be excluded, as these devices may be inactivated by the magnetic field. Any metallic implants must be screened because aneurysm clips, cochlear implants and prosthetic heart valves may become dislodged, heat up or cause the induction of

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BOX 14.2: Indications for general anaesthesia during MRI

- Children
- Ventilated and other ICU patients
- Patients with severe movement disorders
- Patients whose position is limited by pain
- Adults with learning disorders
- Claustrophobic patients
- Certain patients undergoing stereotactic neurosurgical procedures
- Patients receiving intraoperative MR

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electric currents. Patients who are metal workers or who have known intraocular foreign bodies must be screened with a plain X-ray prior to scanning and all female patients should have a pregnancy test.

Tattoos may heat up in the magnetic field.

The increasing use of MR scans and pacemakers has prompted manufacturers to develop MR conditional pacemakers. These have less ferromagnetic components and behave more predictably with a dedicated 'MRI mode' that needs to be switched on before entering the scanner and switched off immediately afterwards. They are not, however, MR safe and specific conditions of the pacemaker, the leads and the scanner need to be met before you can proceed with a scan.

ANAESTHETIC MANAGEMENT

An MR scan is not painful, and the requirements are therefore hypnosis, amnesia and immobility. Recovery will be rapid and most patients can be treated as day cases. The following rules facilitate anaesthesia in the MRI suite:

- The patient is anaesthetized on a tipping trolley in the anaesthetic room.
- Use short-acting agents and a laryngeal mask (LM). With a standard LM, the pilot balloon must be taped away from the site to be scanned, as the small spring inside may cause artefact. The airway should be clear as partial airway obstruction may cause increased respiratory movement and image artefact.
- Maintenance is usually easier with an inhalational agent as this avoids the need for MR compatible infusion pumps or the use of long extensions and a pump placed outside the 50 G line.
- Patients with a poor gag reflex or oesophageal reflux and pregnant women may need intubation and ventilation. A preformed endotracheal tube will allow close-fitting head coils to be applied, but the pilot balloon must again be taped away from the site to be scanned.
- Padding should be placed between the patient's skin and monitoring cables to prevent burns. Loops in cables must be avoided.

- Patients are transferred to a docking table or are taken into the scanning room on a non-ferromagnetic trolley.
- Contrast may be needed for scans to examine tumours or for MR angiography.
- In the event of a cardiac arrest or other critical incident, the patient must be removed from the scanner for resuscitation.

SEDATION

Many patients can have MR successfully performed under sedation.

ADULTS

- Claustrophobic adults may often be adequately managed with oral benzodiazepines.
- Pulse oximetry should be used in all cases.
- Short MR sequences may improve compliance.
- Intravenous sedation must always be given by an anaesthetist and with extreme caution. Monitoring of $ETCO_2$ is advisable.
- Bolus doses of midazolam or low-dose propofol/remifentanil infusion are frequently used.

CHILDREN

- Young children cannot lie still without being asleep and conscious sedation may not ensure compliance because of the noise in the scanner.
- Small infants will sleep deeply after a feed.
- Children over 7 years are often compliant without sedation.
- Many anaesthetists recommend general anaesthesia, rather than sedation, for children under the age of 7 years.
- Sedation must always be performed by adequately trained personnel and with extreme care. In some busy paediatric MR units, nurse-led sedation techniques have been developed.
- Sedation techniques include chloral hydrate, benzodiazepines and low-dose propofol infusion.
- Supplemental oxygen should always be given and adequate monitoring established.

INTRAOPERATIVE MRI

Intraoperative MRI (iMRI) during neurosurgical procedures offers near real-time imaging surgical guidance. Intraoperative scanning allows the surgeon to scan the patient at an appropriate time during surgery and then conclude the surgical procedure or perform further resection. This approach is associated with improved clinical outcomes and, if repeated operations can be avoided, economic savings.

The successful use of iMRI has been reported in tumour surgery (ventricular tumours, gliomas, particularly low-grade and difficult pituitary tumours), epilepsy surgery (including placement of depth electrodes for monitoring) and deep brain stimulation surgery.

The concerns for safety, physiological monitoring and equipment are the same as in the conventional MRI environment but there is now the additional focus on complex anaesthesia techniques, prolonged surgical procedures, repeated intraoperative scans, intraoperative thermoregulation and the need for meticulous attention to patient positioning on the operating table and during the transfer into the scanner. With some procedures lasting more than 6 hours, cases of hyperthermia have been reported, possibly due to the RF heating effect of the scanner.

The presence of a large multidisciplinary team in the iMRI suite highlights the need for a compulsory safety induction and training, and defined patterns of workflow. During the surgery, an MRI responsible person, usually a senior radiographer, controls the flow of people and equipment through the environment.

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ANAESTHESIA FOR NON-CRANIOTOMY NEUROSURGERY

STEREOTACTIC SURGERY

Stereotatic neurosurgery is used to facilitate precise localisation of intracranial lesions. CT, MRI or digital angiography is used to image the brain and provide a three-dimensional reference to accurately define the lesion. The initial step is to apply the extracranial stereotactic frame which attaches to the head under GA or a scalp block and sedation; this then acts as the reference point for localisation. This method is still utilised for the insertion of deep brain stimulators (DBS) for movement disorders and Parkinson's, epilepsy and when treating deep brain lesions closely associated with important functional centres. More recently frameless technology has developed using small adhesive reference markers (fiducials) that are attached to the patient's scalp while awake, giving better surgical and anaesthetic access but slightly less precision. Many surgical procedures including tumour excision that utilised frames in the past are

now excised reliably with image guided technology such as Brain Lab®.

PREOPERATIVE MANAGEMENT

If undertaking awake testing, drugs that inhibit tremor or rigidity in Parkinson and movement disorders may need to be withdrawn as they will mask the symptoms being assessed during surgery.

PERIOPERATIVE MANAGEMENT

- Procedures can be prolonged; pay attention to positioning; insert a urinary catheter.
- When attaching the frame a short-acting opiate should be given as this is particularly stimulating.
- Once the frame is in place, access to the airway is challenging. Alternative airway management devices such as LM and a fibre-optic scope should be accessible as well as the key to dismantle the frame.
- An awake technique is preferable in those in which somatotrophic localisation is required, e.g. thalamotomy or pallidotomy, DBS insertion and some epilepsy surgery.
- Several sedation techniques have been described including the use of midazolam, remifentanil and propofol. All should be used with caution to avoid airway compromise; capnography is advisable.
- For general anaesthesia, follow the same principals for all neurosurgical cases.
- TIVA may be preferable as this will mean continuous anaesthesia while transferring patients to scanners.

POSTOPERATIVE MANAGEMENT

- Stereotatic surgery has lower morbidity and mortality compared with more invasive procedures.
- Following 2–4 hours in recovery patients can return to a neurosurgical ward.
- Complications include broken or misplaced leads, infection, seizures, intracranial haemorrhage and air embolism.

ANAESTHESIA FOR NEUROMODULATION

Stimulators that are inserted for neuromodulation include occipital nerve stimulators (ONSs), sacral nerve stimulators and spinal cord stimulators.

OCCIPITAL NERVE STIMULATORS

A greater occipital nerve stimulator is a treatment recommended by NICE for chronic migraine when medical management has failed. The procedure is usually done in 2 stages:

1. Under local anaesthesia and fluoroscopic guidance, the electrodes are tunnelled under the skin and placed over the occipital nerves. Placement is confirmed by stimulation and patient feedback. A lead is then tunnelled to an exit site where it is connected to an external stimulator.
2. If stage 1 is successful, the neurostimulator is surgically inserted in the infraclavicular region or abdominal wall under general anaesthetic. The patient operates the stimulator by remote control.

Anaesthetic management

The anaesthetic management is straightforward. Patients should be counselled that the trial should be done under local anaesthesia to allow for awake testing. When implanting the stimulator, a general anaesthetic using a laryngeal mask is acceptable although if implanting in the abdomen muscle relaxants may be required.

SPINAL CORD STIMULATORS

Spinal cord stimulators are recommended by NICE as a treatment for chronic neuropathic pain in patients that have suffered pain for over 6 months despite medical management and have had a successful trial. During the initial trial the leads are passed percutaneously using a Tuohy needle into the epidural space and then attached to a temporary external stimulator. If the patient can tolerate the stimulation and pain scores are improved, then

they are suitable for implantation. The electrodes can then be put in place either percutaneously or surgically and the neurostimulator implanted either in the buttock area or abdomen.

Anaesthetic management

- Chronic pain medication should be continued perioperatively.
- Patient's need to be positioned prone and as such need to be intubated and ventilated.
- Postoperatively patients are likely to require long-acting opioid analgesia due to the increased analgesic requirements often seen in chronic pain patients.

SACRAL NERVE STIMULATORS

Sacral nerve stimulation was initially developed for patients with urinary retention but is now employed to treat faecal incontinence, constipation and chronic pelvic pain. Sacral nerve stimulators can be inserted under local or general anaesthesia. During the initial trial, an incision is made over the lower back and the electrodes placed in contact with the sacral nerve roots. These are then connected to an external stimulator for a period of about 2–3 weeks. If successful, the leads are then tunneled beneath the skin to the buttock or lower abdomen, where the pulse generator is sited.

Anaesthetic management

- If using a general anaesthetic technique, for placement of the electrodes muscle relaxants must be avoided as correct electrode placement is identified using perineal and foot movement to stimulation.
- Patients are positioned prone and appropriate care must be paid to pressure points.
- This procedure carries a high degree of postoperative discomfort. Opioids, in addition to simple analgesic therapies, will be required.

ANAESTHESIA FOR PROCEDURES TO RELIEVE HYDROCEPHALUS

Hydrocephalus has a variety of causes, which largely fall into two groups:

- Obstruction of CSF outflow (non-communicating hydrocephalus)
  - Space-occupying lesion
  - SAH
  - Spina bifida
  - Arnold–Chiari malformation
  - Head injury
- Failure of absorption of CSF by the arachnoid villi (communicating hydrocephalus)
  - SAH
  - Meningitis
  - Head injury

In acute and emergency scenarios, surgeons will opt to insert an external ventricular drain (EVD). These are usually inserted into the frontal horn of the lateral ventricle. The drain reduces the ICP and provides a means to measure ICP but is only appropriate for short-term management given the risk of infection. If patients are likely to suffer from hydrocephalus long term or if they have a more insidious presentation, a shunt is inserted. Shunts permit the drainage of CSF to distal sites including peritoneum, atrium and pleura.

ANAESTHETIC MANAGEMENT

Preoperative

- Emergency patients will often be intubated and ventilated in ICU or arrive as an emergency transfer from a non-neuroscience centre.
- Patients should be assessed for signs of raised ICP, including headache, vomiting and altered level of consciousness.
- Vomiting can lead to dehydration and electrolyte disturbance.
- Shunt procedures are more common in children who need to be assessed for prematurity and congenital abnormalities.
- Blocked shunts can present as acute cases when patients may have a full stomach or decreased conscious level.

Perioperative

- Routine anaesthetic monitoring should be instituted in the emergency situation and a rapid sequence induction may be required.

PATHOLOGY

Tumours are the most common pathology in the posterior fossa, particularly in children where they account for 60% of all tumours. The pathologies that require intervention are detailed in Box 14.3.

PREOPERATIVE MANAGEMENT

- Cranial nerve dysfunction may involve loss of the gag reflex and patients may be suffering from aspiration pneumonitis. If there is bulbar involvement postoperative ventilation may be required and/or a tracheostomy.
- Evaluation of cardiovascular status and the ability to tolerate prone or sitting positions must be carried out since hypertensive patients will be prone to hypotension and cerebral ischaemia. An echocardiogram should be arranged for those undergoing surgery in the sitting position. A patent foramen ovale (PFO) is a relative contraindication to the sitting position.
- Fluid and electrolyte status must be determined since patients can be dehydrated and have abnormal plasma electrolytes because of vomiting or concurrent steroid therapy.

POSITIONING

Posterior fossa surgery can be done in supine, prone, lateral, park bench or sitting positions.

PRONE

The prone position allows good surgical access to midline structures, although bleeding may obscure the surgical field. Careful padding of pressure points and avoidance of increased venous pressure is essential.

PARK BENCH

In the park bench position the patient is semiprone with the head flexed facing the floor. It is used for lateral lesions, especially those in the cerebellopontine (CP) angle. Careful padding is required to reduce

> **BOX 14.3: Pathology of posterior fossa lesions**
>
> - Tumours
>   - Axial: astrocytomas (most common in children), medulloblastoma (most common in adults), metastatic, brainstem glioma, ependymoma, dermoid tumours and haemangioblastoma
>   - Cerebellopontine angle: schwannoma, meningioma, acoustic neuroma, glomus jugulare tumour
> - Vascular lesions
>   - Angiomas, arteriovenous malformations and aneurysms (all uncommon)
>   - Nerve decompression for hemifacial spasm and trigeminal pain
>   - Haematomas: spontaneous and traumatic

the risk of pressure damage to peripheral nerves and excessive flexion/rotation of the neck must also be avoided.

SITTING

The sitting position carries the highest risk and should only be used by experienced clinicians in carefully selected cases. Advantages include good surgical access to midline tumours and decreased blood loss. However, there are substantial risks, including cardiovascular instability, decreased cerebral perfusion, air embolism, airway obstruction and pneumoencephalus. The sitting position is achieved by removing the head end from a standard operating table, placing the middle portion in the vertical position and arranging the patient's legs in a flexed position to ensure the buttocks remain firmly wedged against the vertical part of the table. The head is held in a three- point pin fixator mounted on a frame across the table. Excessive head flexion must be avoided to prevent jugular compression, swelling of the tongue, and facial and cervical cord ischaemia. A gap should be maintained between the chin and suprasternal notch and care taken to avoid pressure damage to peripheral nerves.

AIR EMBOLISM

Venous air embolism (VAE) can occur whenever the operative site is above the level of the heart, particularly if large areas of tissue are exposed. Air may enter via dural vessels, dural sinuses, or vessels within a lesion. The greater the head-up tilt, the greater the negative hydrostatic pressure between open veins and the heart, and the greater the rate at which air can be entrained. VAE is particularly common in the sitting position when an 8%–25% incidence is reported.

PATHOPHYSIOLOGY

Morbidity and mortality are directly related to the rate and volume of entrained air. Although the fatal dose of air embolus is unknown, it is likely to be of the order of 100–300 mL. Air is drawn through the right atrium and ventricle into the pulmonary arterioles. Although large volumes (>3 mL kg^{-1}) act as an airlock and cause circulatory failure, microvascular bubbles result in activation and release of endothelial mediators, leading to an increase in pulmonary vascular resistance, a fall in left atrial and ventricular filling and a consequent reduction in cardiac output. Ventricular ectopic beats are common and gas exchange is impaired as physiological dead space increases, causing ventilation–perfusion (V/Q) mismatch, an acute reduction in $ETCO_2$, increase in $PaCO_2$ and a reduction in PaO_2.

DETECTION

Precordial Doppler, end-tidal nitrogen, pulmonary artery catheters and transoesophageal echocardiography have all been used to detect VAE. Capnography is generally regarded as the most useful monitor for VAE, with a fall in $ETCO_2$ being an indication for immediate intervention.

PREVENTION

Volume loading reduces the fall in CVP as the patient is tilted head-up. CVP must be monitored in all cases and the tip of the catheter should be correctly placed. The use of positive end-expiratory pressure is controversial because, although it increases right atrial pressure and might minimize air entrainment, it may adversely affect surgical conditions and increases the risk of paradoxical air embolus if VAE does occur. Compression of the lower limbs and/or abdomen, by the use of leg bandages, a G-suit or medical anti-shock trousers, raises venous pressure. Nitrous oxide should not be used as it will cause expansion of any air bubbles that enter the circulation.

MANAGEMENT

The aims of management are to stop further air entry, remove air already present and treat cardio-respiratory collapse. Immediate measures include:

- Notifying the surgeons and instructing them to flood the operative area with saline and cover the wound with wet swabs.
- Giving 100% oxygen.
- Raising venous pressure by levelling the table and compressing neck veins.
- Aspirating via the CVP line; as well as guiding therapeutic increases in venous pressure and reductions in the hydrostatic gradient, CVP catheters can also be used to aspirate air that has entered the circulation; for optimum recovery of air, the tip of the catheter should be close to where the superior vena cava enters the right atrium.

PARADOXICAL AIR EMBOLISM

Postmortem studies show that approximately 25% of the population has a patent foramen ovale, a potential route for air to pass from the right to left atrium. Whereas the presence of small amounts of air in the venous circulation and pulmonary vascular bed may not adversely affect the patient, the presence of minimal volumes (100–150 µL kg^{-1}) in the arterial circulation can be fatal since a small air embolism reaching the cerebral or coronary circulation will result in irreversible damage. In addition, if air enters the pulmonary circulation, the resultant obstruction to flow will cause the pressure to rise on the right side of the heart and fall on the left, thereby increasing the pressure gradient and potentially reversing flow through the shunt. Other anatomical routes, such as

arteriovenous shunts, may also allow air to pass from the right to the left side of the circulation.

OTHER INTRAOPERATIVE CONSIDERATIONS

- Although the choice of anaesthetic agents is not critical, nitrous oxide should not be used.
- Routine monitoring should be used with the addition of invasive blood pressure monitoring to allow for accurate control of blood pressure.
- Central venous catheters are frequently used particularly when using the sitting position as this allows measurement of venous pressure and may assist in the aspiration of a VAE.
- Damage to midbrain vital centres and cranial nerves through direct intervention, retraction or occlusion of blood supply may result in sudden changes to systemic physiological variables.
- Dramatic and abrupt cardiovascular changes may also occur and the surgeon should be advised of any significant instability. Drugs such as atropine and beta-blockers should be avoided if possible since they will mask midbrain responses to surgical manipulation.
- Electrophysiological techniques, such as somatosensory-evoked potentials, are increasingly used to monitor the integrity of crucial pathways during complex posterior fossa surgery.
- The facial nerve (VII) is stretched across the capsule of acoustic neuromas and monitoring of the VIIth nerve function is often performed to minimize the risk of intraoperative damage. During VIIth nerve monitoring, neuromuscular blocking agents should be avoided after the initial dose used for intubation.
- Normotension should be achieved prior to surgical closure to confirm the adequacy of haemostasis.

POSTOPERATIVE MANAGEMENT

- Following posterior fossa surgery, patients should be managed in a critical care environment.

- Patients who were neurologically intact preoperatively and had uneventful surgery should be extubated, avoiding coughing and straining and monitored in a high dependency area.
- Poor preoperative neurological status, adverse intraoperative events, prolonged surgery with significant tissue retraction and a lesion >30 mm in diameter with mass effect are all indicators of possible slow recovery from anaesthesia and the potential need for elective postoperative ventilation.
- Postoperative swelling in the posterior fossa is a potentially life-threatening complication. The small anatomical space, tendency of the cerebellum to swell following prolonged retraction and the risk of bleeding all add to the threat. A reduced respiratory drive may result from, and in its turn increase, swelling. This may be delayed, sometimes developing hours after an initially good recovery. Deterioration in neurological status after posterior fossa surgery is therefore an indication for an immediate CT scan.
- Hydrocephalus may occur as a result of occlusion of CSF outflow and insertion of an EVD may be necessary.
- Macroglossia is a rare but potentially life-threatening complication. It is likely to be related to occlusion of lingual drainage during prolonged surgery with excessive neck flexion, and may also be associated with the use of an oropharyngeal airway intraoperatively.
- The gag reflex may be obtunded as a result of swelling or damage to the glossopharyngeal and vagus nerves. A nasogastric tube and nil-by-mouth orders are indicated after surgery for large lesions and should be continued until the gag has been formally assessed postoperatively.
- Postoperative nausea and vomiting is common, especially following CP angle surgery. Multimodal antiemetic therapy is often required.

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ANAESTHESIA FOR SPINE SURGERY

The scope of spinal surgery is vast. Patients usually present with one of five pathologies at any site from cervical to lumbosacral:

- Trauma (unstable vertebral fractures)
- Infection (epidural abscess)
- Malignancy (either primary or metastatic)
- Congenital (scoliosis)
- Degenerative

In an ageing population with a growing lower back pain problem, the majority of spinal cases are simple laminectomies and microdiscectomies but many cases involve high-risk multiple level surgery with major blood loss. All present significant challenges to the anaesthetist.

SURGICAL APPROACH

The majority of spinal procedures are performed in the prone position with a few notable exceptions (anterior cervical surgery, thoracic discectomies). There are multiple complications to proning patients detailed in Box 14.4. However, if the right precautions and equipment are used the complications will be minimised.

Pillows, gel pads and foam bolsters can be constructed to support the patient ensuring:

- The abdomen is free.
- The head is at or above the level of the heart in a neutral position using a head rest or a Mayfield head fixator.
- The eyes are taped closed, without padding and free from external pressure, regularly checking them where possible.
- The arms are in a natural position no more than 90° abduction with slight internal rotation paying particular attention to the ulnar nerve at the elbow.

Specific devices are available to facilitate proning: Montreal mattress, Jackson operating table, Wilson Frame and the Andrews operating table. A commonly used alternative to the classic prone position is the knee-elbow position whereby the patient has a foam bolster under their chest, their elbows and

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BOX 14.4: Complications of the prone position

- Accidental extubation
- Ophthalmic complications (corneal abrasions, postoperative visual loss)
- Peripheral nerve injury (ulnar nerve at elbow, brachial plexus)
- Pressure injuries (skin necrosis, breast/genital injury)
- Abdominal compression (venous congestion in epidural veins, organ ischaemia, impaired ventilation and reduced cardiac output)

arms lie beneath them on the operating table and their bottom rests on a support. This has the advantage of keeping the abdomen free and can reduce the lumbar lordosis and improve surgical access for lumbar surgery but can be technically difficult to do if the personnel positioning are not experienced with the position.

INTRAOPERATIVE CONSIDERATIONS

MONITORING

For simple, single-level spinal procedures routine monitoring, including ECG, noninvasive blood pressure monitoring, pulse oximetry, capnography and temperature are adequate. Arterial blood pressure monitoring is required for complex or prolonged procedures when substantial blood loss is anticipated, serial blood gas monitoring is required, there are concerns about spinal cord perfusion or in the presence of significant comorbidities. A central venous catheter (CVC) can assist with fluid balance management or delivery of inotropes/vasopressors. The internal jugular or subclavian routes may be used for thoracic or lumbar procedures, whereas a femoral CVC is more frequently used for cervical approaches. A urinary catheter is mandatory for long procedures and when significant blood loss is anticipated.

EVOKED POTENTIAL MONITORING

Evoked potentials are used during spinal surgery to identify potentially reversible changes in spinal cord function and allow intervention before permanent neurological damage occurs. Somatosensory-evoked potentials (SSEPs) monitor the integrity of the sensory pathway, specifically the dorsal column. SSEPs are recorded from the cerebral cortex using scalp electrodes following electrical stimulation of a peripheral nerve. Motor-evoked potentials (MEPs) allow the integrity of the motor pathways to be assessed. MEP monitoring involves transcranial stimulation (electrical or magnetic) of the motor cortex with the evoked responses being recorded most commonly as compound motor action potentials in peripheral muscles, but occasionally via epidural/intrathecal electrodes or an electrode placed directly

on the exposed spinal cord at surgery. SSEPs and MEPs are sensitive to anaesthetic agents. SSEPs are preserved with low/modest dose volatile agent and during intravenous anaesthesia. MEPs are more sensitive and intravenous anaesthesia techniques, with a high-dose remifentanil and no muscle relaxant, are required.

AIRWAY MANAGEMENT

Difficult laryngoscopy is common in patients with disease of the upper three cervical vertebrae and airway access with an alternative to direct laryngoscopy may be required. Patients with limited extension at the craniocervical junction tend to also have poor mouth opening because of a direct effect as well as an association with temporomandibular joint disease.

There is no evidence that any method of airway management has a better outcome than another in patients with an 'unstable' cervical spine. External cervical spine fixation devices make direct laryngoscopy more difficult and an alternative technique (e.g. awake fibre-optic intubation) rather than the application of force should be used.

BLOOD LOSS

Massive blood loss can occur during spinal surgery particularly in scoliosis surgery and extensive stabilisations. In the prone position, venous return via the IVC can be obstructed and blood then travels back to the heart via epidural veins leading to the risk of large blood loss from these veins. Although venous bleeding is usually insidious, it can be responsible for major blood loss. Catastrophic bleeding can occur as a result of injury to major vessels, including vertebral or carotid injury during cervical surgery, iliac artery injury during abdominal approaches and penetration of the aorta by misplaced pedicle screws or rongeurs during lumbar microdiscectomy. Adequate large-bore venous access, rapid transfusors, cell salvage and readily available blood and blood products should be available for all major spinal cases.

TEMPERATURE

Exposure of patients during prolonged induction of anaesthesia (e.g. during awake fibre-optic

intubation), patient positioning and X-raying can lead to pronounced hypothermia prior to the start of surgery. Patients should be kept warm with forced warm air blankets and heated fluids since hypothermia can contribute to morbidity in terms of coagulopathy and increased infection rates.

ANALGESIA

Spinal procedures are frequently painful. In addition to pre-existing neuropathic pain, extensive muscle retraction and disruption can lead to muscle injury and ischaemia, resulting in severe postoperative pain. With straightforward discectomies paracetamol, a long-acting opiate and local anaesthetic to the wound may be sufficient. If the patient is on extensive chronic pain medication, all pain medications should be continued perioperatively, gabapentin or pregabalin should be considered pre-operatively if not already taken and additional analgesic such as ketamine or clonidine may be required intraoperatively. Major spinal surgery pain can be severe. Additional acute pain techniques including ketamine, clonidine and local anaesthetic infusions are utilized intraoperatively and postoperatively on critical care units in some centres.

INTRAOPERATIVE NEUROLOGICAL DETERIORATION

Spinal cord injury (SCI) can occur during anaesthesia in patients with normal spines and is usually related to poor positioning or severe hypotension. Spinal abnormalities, including spinal stenosis, instability or pre-existing myelopathy, increase the risk of intraoperative SCI.

Reports of cervical SCI during anaesthesia are often raised by non-anaesthetists and may confuse association with causation. The proposed mechanism is acute cord compression during airway management, but studies of cervical movement during intubation in unstable spinal preparations do not support this concept. The injuries described in the reports (usually central and anterior cord syndromes) would be better explained by hypoperfusion and it is likely that most of these injuries are due to a combination of hypoperfusion and malposition for extended periods of time.

POSTOPERATIVE CONSIDERATIONS

- An appropriate postoperative destination must be chosen to facilitate pain control, haemodynamic monitoring or postoperative ventilation.
- *Airway obstruction* – There is a small incidence of airway obstruction after anterior cervical surgery. This can be due to a postoperative haematoma or, more likely, marked tissue swelling of the pharynx or upper airway. Patients may complain of 'not being able to breathe' and want to sit up. They rarely have stridor and do not desaturate until the obstruction is nearly complete. Opening the wound is a priority even if a haematoma is not suspected, since this reduces lymphatic and venous obstruction and improves airway patency. Following evacuation of the haematoma or relief of tissue pressure, a tracheal tube should be left *in situ* for at least 24 hours until swelling subsides.
- *Postoperative neurological deterioration* – Meticulous neurological observation is required to elicit any signs of spinal haematoma formation.
- *Venous thromboembolism (VTE)* – Spinal surgery patients are at high risk of postoperative VTE because of prolonged surgery, paresis, tumour resection and postoperative immobility. Graduated compression stockings and intermittent calf compression should be used in all patents and low-molecular-weight heparin instituted after 12–24 hours.
- *Aperients* – Large opioid requirements and immobility make constipation a frequent postoperative problem.
- *Early mobilization* – In conjunction with physiotherapy, early mobilization reduces postoperative respiratory tract infections and VTE.

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ANAESTHESIA FOR SUPRATENTORIAL SURGERY

Anaesthesia for supratentorial surgery is indicated for a wide range of pathologies. The anaesthetist's knowledge and skill to manipulate the intracranial physiology is vital to optimise surgical conditions and improve the outcome of the patient.

ANATOMY AND PATHOLOGY

The supratentorial region of the brain consists predominantly of the cerebral hemispheres and their meninges. Indications for surgery are detailed in Box 14.5 and the aetiologies are in Box 14.6. Tumours make up the vast majority of elective surgery. It is

> **BOX 14.5: Indications for supratentorial surgery**
>
> - Burrhole biopsy for histological diagnosis of a lesion
> - Craniotomy for excision or debulking of tumour
> - Aspiration of cerebral abscess for antibiotic resistance
> - Vascular procedures
> - Evacuation of haematomas

> **BOX 14.6: Aetiology of brain tumours in adults**
>
> - Glioma
> - Meningioma
> - Metstasis
> - Schwannomas
> - Pituitary adenomas
> - Medulloblastomas
> - Craniopharyngiomas

> **BOX 14.7: Presenting symptoms of brain tumours**
>
> - Headache
> - Nausea and vomiting
> - Seizures
> - Visual disturbances
> - Focal neurology
> - Speech difficulties
> - Confusion and behavioural changes
> - Hearing problems

the most common site for brain tumours in adults but only one-third in children. The usual presentation is described in Box 14.7.

PREOPERATIVE MANAGEMENT

- In addition to the diagnostic CT head, further CT or MRI imaging may be required to aid an image-guided technique or a digital subtraction angiography to determine how vascular the lesion is.
- The neurological status of the patient should be carefully documented so any postoperative deterioration can be identified.
- All routine medication excluding anticoagulants should be continued perioperatively, particularly corticosteroids and anticonvulsants.
- Clotting studies and blood cross-match should be arranged particularly for the meningiomas and vascular tumours.
- Blood glucose and urea and electrolytes may flag up hyperglycaemia secondary to steroid use and sodium abnormalities related to the tumour.

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- Sedative premedications should be given with caution and if given the patient should be monitored.

INDUCTION

- As per the basic principals of neuroanaesthesia, the induction should be smooth avoiding coughing, straining, swings in blood pressure and ICP. This is usually undertaken with propofol and a short acting opiate like fentanyl or remifentanil.
- Orotracheal intubation should be facilitated with a nondepolarising muscle relaxant and then fixed securely with adhesive tape, avoiding ties to prevent obstruction of cerebral venous drainage.
- Standard monitoring and temperature should be included with all patients and many cases require direct blood pressure monitoring. Central venous access should be considered in particularly long cases where multiple infusions are being used.
- Urinary catheters should be inserted for all long operations and those likely to involve the use of osmotic therapy.
- If the surgeons are using image-guided surgery, they may ask you to delay applying waterproof dressing to the eyes, so as not to interfere with navigation.

POSITIONING

The position is dictated by the surgical approach, although the supine position is satisfactory for many cases.

- Head-up tilt (10°–15°).
- Avoid excessive head rotation or flexion since this impairs cerebral venous drainage.
- Secure the head with a horseshoe headrest or three-point pin fixator (bolus dose of opioid to prevent hypertension during pinning).
- Before draping check that there are no loose connections/kinks in the breathing circuit and that there is unimpeded access to intravenous cannulae.

MAINTENANCE

There are some theoretical advantages but no proven outcome benefits to the use of TIVA in neurosurgery. However, unless the ICP is critically raised, many anaesthetists use a balanced technique with controlled ventilation, short-acting opioids and a volatile agent such as sevoflurane or desflurane.

- Adjust ventilation to maintain $PaCO_2$ between 4.5 and 5.0 kPa.
- Air–oxygen mix with FiO_2 0.3–0.5.
- Avoid nitrous oxide.
- Volatile agents should be used at doses below 1.0 MAC to avoid increases in cerebral blood flow.
- Remifentanil infusion allows easy control of cardiovascular variables during periods of surgical stimulation and rapid emergence.
- Normothermia should be maintained using a warming mattress, warm air blanket and warmed fluids.
- A balanced salt solution should be used as maintenance fluid, but bear in mind that large volumes of normal saline can produce hyperchloraemic metabolic acidosis. Blood loss should be replaced with packed red cells and glucose-containing solutions avoided.
- Steroids such as dexamethasone can be given perioperatively to reduce cerebral oedema and prevent postoperative nausea and vomiting.
- All patients should receive prophylactic antibiotics according to local guidelines.
- Deep vein thrombosis (DVT) prophylaxis should include the use of graduated compression stockings and pneumatic calf compression.

INTRAOPERATIVE MANAGEMENT OF A TIGHT BRAIN

Bulging dura on removal of the craniotomy flap indicates a 'tight' brain and the following manoeuvres can be used to prevent cerebral ischaemia and improve operating conditions:

- Check head position and use reverse Trendelenburg.

- Ensure CO_2 is 4.5–5.0 KPa. If necessary temporary hypocapnia 4.0–4.5 KPa can be considered if all other treatments have failed.
- Control blood pressure, particularly in a non-autoregulating brain.
- Dexamethasone 4–10 mg if not already given.
- Switch to TIVA if using volatile.
- Osmotic therapy: mannitol 0.25–1 g/Kg, hypertonic saline 3%–7.2% 30–150 mL/h or furosemide 10–20 mg boluses.

EMERGENCE

- Prior to closure, return the blood pressure to normal while the surgeon ensures haemostasis.
- As with induction, the emergence and extubation should be smooth and avoid straining, coughing, swings in ICP and blood pressure. There are various techniques used to achieve this including extubating deep and inserting a laryngeal mask but more frequently anaesthetists will extubate while the patients are on a remifentanil infusion as an antitussive agent.
- Give analgesia (morphine) and antiemetics (ondansetron, cyclizine).
- Treat emergence hypertension with an antihypertensive (labetolol).
- Consider postoperative ventilation only if the patient was severely obtunded preoperatively or there have been intraoperative problems.
- ICP should be monitored if the patient will be sedated and ventilated in the postoperative period.

POSTOPERATIVE MANAGEMENT

- Most postoperative complications occur in the first 6 hours; Box 14.8.
- After supratentorial surgery patients experience moderate to severe pain and analgesia should include regular paracetamol and opioids either orally or via PCA.
- For patients with known pain problems, a scalp block should be considered.
- Postoperative nausea and vomiting are common and antiemetics should be prescribed prophylactically.

> **BOX 14.8: Postoperative complications**
>
> - Bleeding at the operative site
> - Subdural haematoma
> - Pneumocephalus
> - Seizures
> - Cerebrospinal fluid leak
> - Infarct
> - Infection

- Mechanical methods of DVT prophylaxis should be continued until the patient is mobilizing. Low-molecular-weight heparin is used in consultation with the neurosurgeon but is probably safe after 24 hours.

AWAKE CRANIOTOMY

This is the technique of choice for surgical procedures in which lesions are adjacent to or within eloquent areas in the motor and sensory strip, and speech area. It can also be used during epilepsy surgery when intraoperative electrocorticography (ECoG) is being used to define the resection margins precisely and during deep brain surgery to facilitate accurate placement of stimulating electrodes. Awake craniotomy allows the patient's neurological status to be assessed continually during surgery so that maximal resection can be achieved while minimizing the risk of permanent damage. The technique is growing in popularity due to the increased survival, reduced length of stay and postoperative complications. It should now be considered for all supratentorial tumours not just those in eloquent areas.

PREOPERATIVE

PREOPERATIVE ASSESSMENT

The key to successful awake surgery is the relationship between patient, surgeon and anaesthetist.

- Identify those patients in whom contraindications (Box 14.9) to awake surgery exist.

- Absolute
  – Patient refusal
  – Inability to stay still
  – Inability to cooperate (confusion)
- Relative
  – Gross obesity
  – Difficult airway
  – Anxiety
  – Extreme response to pain
  – Communication problems
  – Obstructive sleep apnoea
  – Young age
  – Poor motivation

- The anaesthetist should explain all of the steps of the proposed technique in detail, highlighting that the aim is to provide an awake, lucid and pain-free experience during intraoperative testing.
- Patients may be seen by neuropsychologists if the lesion involves speech and language areas and they determine what neurological function is going to be tested and document baseline responses.
- Explain to patients that they should communicate with the anaesthetist if they feel pain, anxiety or nausea and reassure them that these problems can be dealt with quickly and effectively.

INTRAOPERATIVE

Many combinations of sedation, analgesia and anaesthetic techniques have been described, each with their advocates and proposed advantages. Essentially there are three parts to the operation:

- Craniotomy
- Tumour excision
- Closure

The key principal of an awake craniotomy is that the patient is either awake or lightly sedated during the tumour excision. The patient can be asleep for the craniotomy preparation stage and the closure;

this is known as the asleep, awake, asleep technique where usually a laryngeal mask airway is used to maintain the airway. The choice of technique will be determined by the surgeon, pathology, length of surgery and patient factors. The essential anaesthesia requirements are

- Optimal analgesia during painful stimuli
- Prevention of nausea, vomiting and seizures
- Patient immobility and comfort during awake testing and resection
- Whichever technique is chosen, effective local anaesthesia is essential usually in the form of a scalp block that can provide effective analgesia for 8 hours

OTHER IMPORTANT INTRAOPERATIVE CONSIDERATIONS

- Full anaesthetic monitoring, with most anaesthetists inserting invasive blood pressure monitoring and using capnography when the patients are awake also.
- Urinary catheterization should be considered for the longer operations. If not, then an adult nappy could be offered should they need to urinate intraop.
- BiS is particularly useful if using the asleep-awake technique as it can minimize the amount of propofol/volatile used and hence the patient wakes up more promptly for functional testing.
- Clear surgical drapes should be used to reduce feelings of claustrophobia during the awake phase and positioned to allow continuous and unimpeded access to the patient's airway by the anaesthetist.
- Antiemetics (ondansetron, cyclizine and dexamethasone) should be given at the start of surgery.
- Analgesia: paracetamol and a long-acting opiate intravenously.
- Seizures can occur in up to 20% of cases, usually during epilepsy surgery, and can be treated with cortical irrigation with cold saline or bolus doses of propofol. Magnesium up to 10 g given by slow intravenous infusion at the start of surgery may also have some protective effect.

- Loss of patient cooperation terminates any possibility of useful functional testing and thus imposes deepening of anaesthesia to ensure the safe completion of surgery.
- Complications are summarized in Box 14.10.

AIRWAY MANAGEMENT

Whatever anaesthetic technique is chosen, there is always the risk of airway/breathing problems and strategies must be in place to deal with hypoventilation and airway obstruction. Difficult airway equipment and adjuncts should be available including access to a fibre-optic scope.

Overall, awake craniotomy is a very safe procedure with minimal mortality and morbidity related to the anaesthetic technique with several institutions safely discharging patients home on the day.

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BOX 14.10: Complications of awake craniotomy

- Seizures
- Nausea and vomiting
- Dysphoric reaction
- Respiratory depression
- Airway obstruction
- Air embolism
- Loss of patient cooperation, leading to conversion to general anaesthesia
- Brain swelling
- Focal neurological deficit

Thoracic surgery

MATTHEW STAGG

15

BRONCHOPLEURAL FISTULA

MATTHEW STAGG

Bronchopleural fistula (BPF) is a direct communication between the tracheobronchial tree and the pleural cavity. Causes include dehiscence of bronchial stump, cancer, inflammatory lesions and trauma. In developed countries, dehiscence of the bronchial stump following pneumonectomy is the most common cause. The incidence of BPF following pneumonectomy is extremely low in specialized centres.

Minor forms of post-pneumonectomy BPF can be sealed bronchoscopically with fibrin glue. Large fistulas require resuture of the bronchial stump via a repeat thoracotomy.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

Symptoms relate either to accumulation of pneumothorax in spontaneously breathing patients, a difficulty providing IPPV due to significant leak or from fluid from the infected space flowing over to the 'normal' lung.

SMALL BPF

- Malaise and low-grade fever
- Cough ± haemoptysis, wheeze or dyspnoea

LARGE BPF

- Severe dyspnoea and debilitation
- Coughing up copious amounts of thin brown fluid

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INVESTIGATIONS

- Chest X-ray. Loss of pneumonectomy space fluid. Consolidation/collapse of remaining lung.
- Blood gas analysis to assess hypoxaemia, hypercarbia and acid-base status.

PREOPERATIVE PREPARATION

- General resuscitation including oxygen by face-mask.
- Sit patient up to prevent further spillover.
- Insert chest drain on pneumonectomized side.
- Transport patient to theatre in sitting position with drain open.

PREMEDICATION

- None required

MONITORING

- Routine basic monitoring
- Invasive arterial pressure
- Central venous pressure
- Arterial blood gases
- Core temperature
- Urine output

ANAESTHETIC TECHNIQUE

Classically it has been advocated that a post-pneumonectomy fistula should be isolated with an endobronchial tube before IPPV is employed. This can be achieved either with awake endobronchial intubation with local analgesia of the airway (with or without fibre-optic bronchoscopy) or inhalational induction and intubation under deep inhalational anaesthesia. These techniques should be discussed at examinations, but both are fraught with difficulty. Most experienced anaesthetists now use the following technique:

- Sit patient upright with drain open.
- Preoxygenate.
- Use intravenous induction and suxamethonium or rocuronium.
- Perform rigid bronchoscopy.
- Insert double-lumen tube into the remaining bronchus with fibre-optic bronchoscope.

- Administer further muscle relaxant.
- IPPV via endobronchial portion of tube.
- Place patient in lateral position for thoracotomy.

POSTOPERATIVE MANAGEMENT

- Treat as for pneumonectomy.
- Sputum retention, infection, acute lung injury (ALI) and respiratory failure are common and carry a high mortality. Treat with physiotherapy, antibiotics, ventilation and early tracheostomy.
- Infection in pneumonectomy space.

OUTCOME

- Mortality around 10%–20%

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CROSS-REFERENCES

Bronchiectasis, Chapter 1
Bronchial carcinoma, Chapter 1
Pneumonectomy, Chapter 15
Postoperative analgesia for thoracic surgery patients, Chapter 15
One-lung anaesthesia, Chapter 28
Preoperative assessment of pulmonary risk, Chapter 25

ANAESTHESIA FOR THORACIC SURGERY – GENERAL PRINCIPLES

MATTHEW STAGG

Anaesthesia for thoracic surgery has evolved since the 1930s when Gale and Waters (USA) and Magill

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(UK) introduced single-lumen endobronchial tubes for selective endobronchial intubation.

The majority of lung resections in the UK are carried out to treat lung cancer: 20% of patients with lung cancer have resectable tumours. Thoracic surgery is very invasive and many patients are elderly smokers with smoking associated comorbidities, especially COPD and cardiac disease. The operative mortality following pneumonectomy and lobectomy is 6.2% and 2.4% respectively (Table 15.1). Increasing numbers of procedures (including lobectomy) can be undertaken thoracoscopically using video assisted thoracic surgery (VATS).

Pulmonary resection is one of the most physiologically traumatic surgical procedures. The inflammatory and neurohumoral response is correspondingly large. Inflammation is a trigger to arterial plaque rupture and arterial thrombosis. Interleukin 6 (IL6) plays a central role. Plaque rupture in the coronary circulation may cause acute coronary syndrome (ACS). An ACS, either unstable angina or MI (STEMI or NSTEMI), is a major cause of death and morbidity. The incidence of ACS is higher in thoracic surgery than any other area except major vascular surgery. Valvular heart disease is less common but relevant, with the prevalence of significant aortic stenosis 3% in those aged over 75. Cardiac failure has a prevalence of 2% in the sixth decade rising to 10% in the eighth decade. Its presence carries a very high risk.

Rigid bronchoscopy and mediastinoscopy are investigative procedures although bronchoscopy can be used for therapeutic purposes. Pleurectomy is used to treat recurrent spontaneous pneumothoraces in young adults. All these are low risk. Pleurodesis is undertaken for pneumothorax secondary to COPD and for malignant pleural effusions. These patients are usually debilitated and risk is higher (Table 15.1).

HDU care is always indicated following lung resection or major mediastinal surgery. Most of the complications of thoracic surgery are pulmonary or cardiac. Some patients require postoperative ventilation as a result of the development of respiratory failure secondary to infection or acute lung injury (ALI). The use of intravenous fluid during and following lung resections should be cautious. Over-hydration is associated with ALI and other pulmonary complications, especially after pneumonectomy.

Operations carried out through a posterolateral thoracotomy are extremely painful. Poor analgesia causes much distress and impairs both respiratory function and sputum clearance; regional analgesia is always advisable either in the form of paravertebral blocks or thoracic epidurals. These have the benefit of avoiding systemic opioids that may cause respiratory depression, atelectasis or cough suppression.

PREOPERATIVE ASSESSMENT

PULMONARY FUNCTION

- Clinical – check functional status, sputum production and physical examination.

Table 15.1 Thoracic surgical results from 42 UK centres (2007–2008)

Lung resection for primary malignant tumours	Numbers	Deaths (%)
Pneumonectomy	497	31 (6.2)
Lobectomy	2800	68 (2.4)
Segmentectomy/Wedge resection	508	7 (1.4)
Pleural procedures (open)		
Pleurectomy/pleurodesis ± closure of air leak	379	9 (2.4)
VATS for pulmonary/pleural disease		
Lobectomy	146	2 (1.4)
Wedge resection	161	2 (1.2)
Closure of air leak ± pleurectomy/ pleurodesis	1531	16 (1.0)

Source: Adapted from The database of the Society of Cardiothoracic Surgeons of Great Britain and Ireland.

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- Imaging – perform chest X-ray and CT scan.
- Pulmonary function tests – spirometry (FEV_1), diffusion (DLCO), arterial blood gases (PCO_2).
- If needed (see below), perform a cardiopulmonary exercise test (CPET) (VO_{2max}).
- The calculation of whether a patient can withstand lung resection is based on the triad of spirometry (FEV_1), parenchymal function (DL_{CO}) and exercise capacity (VO_{2max}).
- Survival correlates with the amount of functional lung remaining postoperatively. This is known as the predicted postoperative (PPO) lung function.
- The PPO values for FEV_1 and DL_{CO} are calculated in a stepwise manner using the formula: PPO value (%) = preop value (%) × (1 – % lung resected/100).
- The proportion of lung volume possessed by each lobe is given in Figure 15.1. For example, if the preoperative FEV_1 is 60% predicted and a left lower lobectomy is proposed, the PPO FEV_1 = 60 × (1 – 23/100). Thus, the PPO FEV_1 is 60 × 0.77, i.e. 46%.
- If the PPO FEV_1 is over 40%, mortality is low. If it is less than 30%, mortality is very high.
- If PPO FEV_1 is between 30% and 40%, perform the same calculation for PPO DL_{CO}. Again, mortality is low if this is over 40% and very high if it is below 30%.
- If PPO DL_{CO} is between 30% and 40%, measure preoperative VO_{2max} using a CPET. If VO_{2max} is greater than 15 mL/kg/min, mortality is low

whereas if it is much below 15 mL/kg/min it is high.
- Patients with infected sputum must be treated with antibiotics and physiotherapy.
- Bronchodilator medication must be continued throughout.

CARDIAC DISEASE

Some patients will have a high risk for heart disease. Patients with poor functional reserve (NYHA or CCS classes III and IV) are at greatest risk and it is this group that requires cardiological input.

- Check functional status.
- Assess for murmurs and cardiac failure.
- Perform ECG.
- Perform echo if systolic murmur to quantify possible aortic stenosis (AS). Surgery can be performed safely in patients even with severe AS provided truly asymptomatic and LV systolic function normal.
- If in doubt about myocardial reserve, refer to cardiologist for opinion and possible stress test such as a CPET, dobutamine stress echo (DSE) or stress MRI.
- Continue beta-blockers and statins through the perioperative period.
- Preoperative cardiac intervention of any type is only indicated in those patients with cardiac disease that requires intervention regardless of the planned surgery.

Right lung

Upper 14%

Middle 10%

Lower 30%

Left lung

Upper 23%

Lower 23%
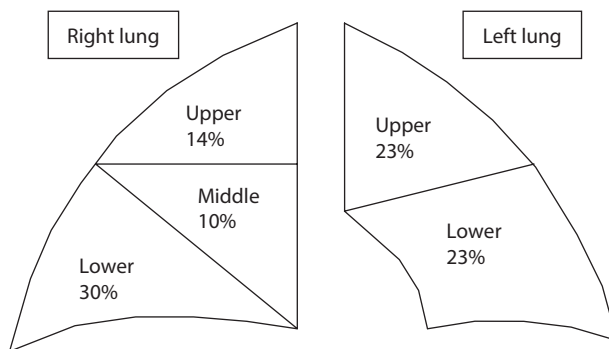
Cardiac disease

Figure 15.1 Percentage contribution of each lung lobe.

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Patients with coronary stents are at high risk of perioperative cardiac events and need multidisciplinary assessment. The degree of risk depends on the interval between stent insertion and surgery, and the site and type of stent. Those with recently inserted stents, <6 weeks for bare metal stents (BMS) and <1 year for drug eluting stents (DES), and those with left main stem and proximal LAD stents are at greatest risk. Patients receive aspirin plus clopidogrel for at least a year after DES and 6 weeks after BMS insertion. Stopping dual therapy in that time is associated with a high risk of stent thrombosis. This is usually fatal if the stent is in a major artery. Conversely, continuation of dual therapy increases the risk of perioperative haemorrhage. Local protocols should exist for managing these patients. A suggested approach is as follows:

- Continue the aspirin throughout but stop the clopidogrel 1 week preoperatively.
- Admit 3 days preoperatively and commence short-acting intravenous antiplatelet agent infusion.
- Stop the infusion 8 hours preoperatively and restart postoperatively when the risk of bleeding is low (usually after a few hours).
- Stop the infusion and restart clopidogrel when the patient can take oral medication.

Diabetes and chronic kidney disease are managed in standard fashion. Routine ECG and haematological and biochemical screening are always carried out. Thromboprophylaxis is indicated for all procedures except isolated bronchoscopy.

ENDOBRONCHIAL INTUBATION

The first double-lumen endobronchial tube, the Carlens, was introduced for differential bronchopulmonary spirometry in 1949. The Robertshaw tube, the best nondisposable type, was introduced in 1962. There are left and right versions and it exists in three sizes: small, medium and large. In many units, reusable tubes have been replaced by disposable varieties, of which the 'Bronchocath' is the most widely used. It comes in a range of sizes. Sizes 35, 37, 39 and 41 are applicable to adults. Choice of tube size depends on patient height (Table 15.2).

Table 15.2 Patient height and sizes of disposable endobronchial tubes

Male		Female	
Height (cm)	Size	Height (cm)	Size
>180	41	>170	39
160–180	39	150–170	37
<160	37	<150	35

Most anaesthetists use double lumen tubes for thoracic surgery. These are usually easy to insert and stable once placed. They also allow rapid lung deflation and good secretion clearance by suction. The blind placement of these tubes is not recommended. Fibre-optic bronchoscopy through the bronchial lumen should be used to place the tube under direct vision. Bronchoscopy through the tracheal lumen then confirms position relative to the carina. Alternatively, bronchoscopic checking of position following blind placement is acceptable. There are two schools of thought as to which side of the tube should be used. Some prefer to intubate the bronchus of the dependent lung. Others prefer to use a left-sided tube unless surgery (such as a left pneumonectomy) precludes this.

PROCEDURE FOR CUFF INFLATION

- Inflate the tracheal cuff first except in bronchiectasis with purulent secretions, severe pulmonary haemorrhage and bronchopleural fistula. In these situations, inflate the bronchial cuff first to prevent overspill.
- With the tracheal cuff inflated, check for bilateral breath sounds. Place the stethoscope laterally close to the axilla.
- With bilateral ventilation confirmed, ventilate solely down the endobronchial lumen, and open the tracheal lumen of the tube.
- Listen and feel for air emanating from the tracheal lumen during IPPV while inflating the bronchial cuff. Air egress will cease when the cuff is inflated if the tube is the right size and correctly placed.

An alternative to a double lumen tube is a bronchus blocker with an ordinary endotracheal tube or a combined tube and blocker (the Combivent).

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There are several disposable types of blocker. The blocker is passed through the endotracheal tube and placed in the main bronchus of the upper lung using bronchoscopy. When lung isolation is required, the blocker is inflated. Deflation of the lung occurs through the lumen of the blocker but this is often considerably slower than by using a double lumen tube. Positional stability is not as good as with a double lumen tube.

ONE-LUNG VENTILATION (OLV)

- The majority of major thoracic surgery is carried out with the patient in the lateral position.
- When the upper lung is deflated to aid surgery, pulmonary blood flow continues to that lung. A true shunt is created and hypoxaemia may occur.
- Use an inspired oxygen concentration of 50% initially during OLV if oxygen saturations

PaO_2 are acceptable. Increase it incrementally if arterial oxygen saturation (SpO_2) is too low. This does not affect the shunt in the upper lung, but improves oxygenation in alveoli with low ventilation-perfusion ratios in the lower lung.
- There is evidence that over-inflation (barotrauma and volutrauma) leads to acute lung injury. The use of low tidal volumes improves outcome in ventilated patients with acute respiratory distress syndrome.
- Limiting ventilation can lead to carbon dioxide retention, but permissive hypercarbia is preferable to lung trauma.
- Hypoxic pulmonary vasoconstriction (HPV) plays little part in reducing hypoxaemia during the time it takes to complete surgery.
- Many inhalational agents inhibit HPV but they do not appear to impair arterial oxygenation during OLV.
- General guidelines for OLV are set out in Table 15.3.

Table 15.3 Guidelines for the management of one-lung ventilation

- Use inspired oxygen concentration 50%–100% (FiO_2 0.5–1.0). Increase if SpO_2 <90%.
- Use normal I:E ratio, but increase expiratory phase if gas trapping is likely.
- Use pressure limited ventilation and small tidal volumes (e.g. 6 mL/kg).
- Allow permissive hypercarbia if necessary.

If desaturation occurs during one-lung ventilation despite FiO_2 of 1.0

Check the machine and circuit to ensure integrity.
- Check tube with (fibre-optic bronchoscope if necessary) to exclude malposition, cuff herniation or leak.
- Use a small positive end-expiratory pressure (PEEP) on lower lung.
- Consider CPAP on the upper lung (operative lung). May cause upper lung insufflation impairing operative view.
- Ask surgeon to occlude the pulmonary artery to the upper lung.
- Revert temporarily to double-lung ventilation.

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CROSS-REFERENCES

Preoperative assessment of cardiovascular risk in non-cardiac surgery, Chapter 25

Preoperative assessment of pulmonary risk, Chapter 25

One lung anaesthesia, Chapter 28

INHALED FOREIGN BODY

MATTHEW STAGG

Foreign bodies can be inhaled at any age but are more common in children under 3, the elderly, debilitated and inebriated patients. Foreign bodies in the tracheobronchial tree require removal by bronchoscopy. The rigid bronchoscope is the best instrument for this procedure, as it allows grasping forceps of an adequate size to be used. Peanuts are of a size easily inhaled by children and they are liable to fragment in the airway, releasing irritant oil that causes severe inflammation.

PREOPERATIVE ASSESSMENT

There may be a history of inhalation such as a bout of coughing whilst eating, chewing or nibbling an object. Alternatively, a chronic cough with wheeze, stridor or fever may be the presenting symptoms. A persistent chest infection in an otherwise healthy child warrants investigation for an inhaled foreign body.

INVESTIGATIONS

- Chest X-ray. May be normal or show obstructive emphysema, best seen on an expiratory film, atelectasis or consolidation. The object may be seen if radio-opaque.
- Full blood count in children.
- Standard investigations relating to age and medical condition.

PREMEDICATION

- Anaesthetist's preferred regime in children; none necessary in adults
- Omit premedication if upper airway obstruction

PERIOPERATIVE MANAGEMENT

MONITORING

- Routine basic monitoring

ANAESTHETIC TECHNIQUE

- Traditionally inhalation induction recommended because IPPV may cause further displacement of the foreign object. Intravenous induction in adults and older children is becoming acceptable.
- Inhalational induction in smaller children and all patients with upper airway obstruction is still recommended.
- In most cases it is safe to use relaxants. However, if upper airway obstruction is present due to a foreign body in the upper trachea or larynx, it may be safer to perform bronchoscopy under deep inhalational anaesthesia.
- If the procedure is likely to be prolonged, rocuronium may be used with sugammadex available for reversal.
- Maintain anaesthesia intravenously in adults, and ventilate gently with a venturi system, taking care not to blow fragments further down the airway.
- In children, use a volatile agent with a ventilating bronchoscope.

POSTBRONCHOSCOPY MANAGEMENT

AFTER SHORT ATRAUMATIC PROCEDURES

- Ventilate with oxygen by facemask and Guedel airway or laryngeal mask.
- Continue IV anaesthesia and check for muscle relaxant reversal (nerve stimulator).
- Allow patient to awaken.
- Sit up.
- Humidified air/oxygen.

AFTER LONG OR TRAUMATIC PROCEDURES

- There may be upper airway oedema especially in small children and postoperative ventilation may be required.
- Remove bronchoscope and reintubate with a small oral endotracheal tube.
- Allow patient to awake on side breathing 100% O_2.
- Extubate awake and sit up.
- Humidified air/oxygen.
- Steroids and nebulized epinephrine may be useful.
- Be prepared for emergency reintubation.
- Return all children to HDU.

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LOBECTOMY

MATTHEW STAGG

Lobectomy is the surgical excision of one lung lobe (or two if the right middle lobe is resected with another lobe). It is performed either through a posterolateral thoracotomy or using VATS. The indications are primarily tumours (usually malignant), bronchiectasis, TB and fungal infections.

PREOPERATIVE ASSESSMENT

- Patients with bronchiectasis are usually admitted several days preoperatively for antibiotics and physiotherapy with postural drainage.
- If a small cell cancer, it may be associated with myasthenic syndrome (Eaton–Lambert). It presents as proximal limb weakness which improves with exercise. There is no bulbar weakness. These patients are very sensitive to all muscle relaxants.
- Blood must be rapidly available.

PREMEDICATION

- Full explanation about high-dependency care, postoperative monitoring and analgesia including benefits and risks of neuraxial blockade.

MONITORING

- Routine basic monitoring.
- Invasive arterial pressure.
- Central venous pressure.
- Core temperature.
- Arterial blood gases.
- Urine output (if epidural analgesia employed or high-risk patient).

ANAESTHETIC TECHNIQUE

- General anaesthesia using a volatile agent or TIVA, supplemented with an opioid/relaxant. Remifentanil, atracurium and desflurane for a

smooth but sharp wakeup is suggested. Nitrous oxide not advised.

- Regional block (thoracic epidural, paravertebral or epipleural) routinely used unless contraindicated.
- Bronchoscopy is usually performed first through a standard single lumen tube.
- Intubate with double-lumen endobronchial tube, or continue with a standard endotracheal tube with bronchial blocker.
- Treat epidural related hypotension with vasoconstrictor, not fluid.
- One-lung anaesthesia used while the chest is open.
- In bronchiectasis, the remaining lobe of the upper lung is unprotected from the spread of secretions. Infected material can also seep past the endobronchial cuff into the lower lung. Repeated suction to both lungs limits contamination. Spread of secretions from lobe to lobe can be reduced by using a bronchus blocker to block specific bronchi but this is tricky.
- Integrity of bronchial suture lines tested prior to chest closure. Bronchial stump is covered with sterile water. Pressure up to 30 cm H_2O exerted by manual compression of the rebreathing bag. Any leak is seen as bubbles.
- Significant leaks from raw lung surfaces require stapling or sealing with tissue glue.
- Maintenance of normothermia crucial to postoperative respiratory function. Underbody warming, fluid warming and use of heat and moisture exchangers mandatory.

POSTOPERATIVE MANAGEMENT

- Apical and basal drains placed at surgery (apical anterior to basal) with suction on –2 kPa and under water seal.
- Ensure relaxant fully reversed (nerve stimulator).
- Extubate in the sitting position, breathing spontaneously.
- Administer humidified oxygen by face-mask.
- Persistent air leak can be a problem.

COMPLICATIONS

- Haemorrhage. Replace volume lost. May need surgical exploration.
- Sputum retention, infection, acute lung injury and respiratory failure. Treat with physiotherapy, antibiotics, ventilation and early tracheostomy.

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MEDIASTINAL SURGERY

MATTHEW STAGG

Mediastinal surgery can be split into two categories:

- Diagnostic – mediastinoscopy, mediastinotomy
- Therapeutic – excision of tumours and cysts

Mediastinoscopy and mediastinotomy procedures are used to assess mediastinal lymph node involvement to stage carcinoma, during which samples are often taken. These patients are in the categories outlined for pneumonectomy and lobectomy but the staging procedure is low risk.

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Patients with large primary mediastinal tumours are at high risk, mainly from airway obstruction. Such patients may present for minor diagnostic procedures but the airway problems outlined for major mediastinal surgery also apply. Some patients with thymoma have myasthenia gravis. Thymomas are rarely large enough to cause obstruction.

MEDIASTINOSCOPY/ MEDIASTINOTOMY

Mediastinoscopy is passage of a mediastinoscope into the pretracheal area via a small incision above the suprasternal notch. Biopsies can be taken and nodes palpated digitally. Mediastinotomy is opening of the anterior mediastinum via an incision through the bed of the second costal cartilage. The pleura may be breached.

PREOPERATIVE ASSESSMENT AND CONSIDERATIONS

- Assess for tracheal obstruction or deviation with clinical examination (inspiratory stridor), chest X-ray (PA and lateral) or CT scan.
- Myasthenia is a special case requiring expert input.

PREMEDICATION

- None needed.

MONITORING

- Routine basic monitoring; arterial line may be useful.

ANAESTHETIC TECHNIQUE

- Rigid/fiber-optic bronchoscopy usually performed first.
- Position patient supine with sandbag under shoulders and neck extended.
- General anaesthesia with a volatile agent or TIVA, supplemented with an opioid/relaxant. Remifentanil, atracurium and desflurane recommended. Nitrous oxide not advised.
- Airway obstruction may be a real possibility.

- If pleura is breached during mediastinotomy, this is not usually a problem as there is no leak from the lung. IPPV with PEEP keeps the lung expanded.

POSTOPERATIVE MANAGEMENT

- Ensure relaxant fully reversed.
- Extubate sitting up.
- Check chest X-ray for pneumothoraces.

MAJOR MEDIASTINAL SURGERY

Primary anterior and superior mediastinal tumours are most common in young adults. Tumours include thymoma, retrosternal thyroid and teratoma. Ten percent of patients with myasthenia gravis have thymomas and myasthenia presents its own unique problems. Anterior mediastinal tumours are particularly likely to cause problems during anaesthesia. The greatest of these is compression and obstruction of the airway and vascular structures – most commonly the superior vena cava (SVC). Operative mortality is low in specialized centres with good outcomes following curative resection. Recurrence is a problem with some tumours. These may respond to chemotherapy or radiotherapy. Some tumours (e.g. secondary teratoma) may require reoperation.

PROCEDURE

Surgery is usually performed through a median sternotomy in a supine position, but small tumours may be resectable transcervically. Blood loss can be considerable.

PREOPERATIVE ASSESSMENT AND CONSIDERATIONS

- Respiratory symptoms, cough, wheeze, stridor or dyspnoea suggest tracheal obstruction.
- Chest X-ray (PA and lateral) and CT scan to evaluate trachea.
- Lung function tests with flow volume loop.
- Occasionally echo or pulmonary angiography (involvement of pericardium or pulmonary artery).

- Myasthenic patients are often receiving steroids and other immunosuppression. Steroid cover may be required.
- Optimize preoperative anticholinesterase therapy (seek advice from a neurologist). Stop anticholinesterase therapy 4 hours prior to surgery.
- Full explanation about high-dependency care, possibility of postoperative ventilation, monitoring and analgesia including benefits and risks of neuraxial blockade.
- Blood must be rapidly available.

PREMEDICATION

- None needed.

MONITORING

- Routine basic monitoring.
- Invasive arterial pressure.
- Central venous pressure (femoral if SVC obstruction).
- Core temperature.
- Arterial blood gases.
- Urine output (if epidural analgesia employed or high risk patient).

ANAESTHETIC TECHNIQUE

- Some tumours compress the trachea when the patient is supine and the patient may not be able to lie supine (see below).
- Lower limb venous access if SVC obstruction.
- General anaesthesia with volatile agent or TIVA, supplemented with opioid and relaxant. Remifentanil, atracurium and desflurane recommended. Nitrous oxide not advised.
- Special considerations apply to muscle relaxants in myasthenia gravis. Patients are uniquely sensitive to nondepolarising relaxants but resistant to suxamethonium. Either use very small doses of atracurium or avoid relaxants altogether. Always use a nerve stimulator if administering relaxants.
- Sternotomy less painful than thoracotomy. Regional block optional but may be useful in myasthenia provided motor block is minimal.

- Blood loss a major problem and blood must be rapidly available.
- Use lowest FiO_2 compatible with adequate arterial oxygen saturation and severely restrict intravenous fluid if patient has received chemotherapy with bleomycin (risk of acute lung injury).

AIRWAY OBSTRUCTION

Options for dealing with tumours compressing the airway include:

- Induction with the patient in whichever position they find it easiest to breathe (often sitting up or lateral). Turn supine to intubate.
- Inhalational induction (which can be difficult).
- Intravenous induction followed by suxamethonium or rocuronium and rigid bronchoscopy to splint the airway.
- Use of cardiopulmonary bypass with awake percutaneous femoral artery and vein cannulation, especially in the presence of gross airway obstruction or pulmonary artery compression.

POSTOPERATIVE MANAGEMENT

- Ensure relaxant fully reversed (nerve stimulator).
- Extubate in the sitting position, breathing spontaneously.
- Administer humidified oxygen by face-mask.
- Caution with intravenous fluid (acute lung injury).
- Treat epidural-related hypotension with vasoconstrictor, not fluid.
- Consider mechanical ventilation if surgery prolonged, myasthenic patient, major nerves sectioned (e.g. phrenic) or airway patency still a problem.
- Reintroduce anticholinesterases (myasthenia only).

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CROSS-REFERENCES

Bronchial carcinoma, Chapter 1
Sarcoidosis, Chapter 1
Lobectomy, Chapter 15
Pneumonectomy, Chapter 15

PLEURECTOMY AND PLEURODESIS

MATTHEW STAGG

Pleurectomy is removal of the parietal pleura. It
is the treatment of choice for fit patients (usually
thin young adults) with a spontaneous pneumo-
thorax. The pleura is stripped except over the dia-
phragmatic and mediastinal surfaces of the lung
which then adheres to the chest wall. It is com-
monly carried out as a VATS technique but an
open approach can be used. Bilateral pleurectomy
is sometimes required and is performed through
a sternotomy. Pleurodesis is the introduction of a
substance into the pleural space (usually talc) to
create inflammatory adhesions. It is indicated in
debilitated patients with pneumothoraces second-
ary to COPD, young tall often male patients and
those with malignant effusions. This is carried out
as a VATS technique.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- All but small pneumothoraces should be
  drained prior to anaesthesia.

PREMEDICATION

- None needed.

MONITORING

- Routine basic monitoring.
- Invasive arterial pressure in debilitated
  patients.

ANAESTHETIC TECHNIQUE

OPEN AND VATS PLEURECTOMY/VATS PLEURODESIS

- General anaesthesia with a volatile agent or
  TIVA supplemented with an opioid/relaxant.
  Nitrous oxide increases size of pneumothorax
  and must not be used.
- Regional block (thoracic epidural, paravertebral
  or epipleural) routinely used for open
  pleurectomy unless contraindicated.
- Epidural not needed for VATS procedures.
- Intubate with double-lumen endobronchial
  tube. Minimize inflation pressures.
  Positive pressure ventilation may create
  tension pneumothorax. Potential exists for
  pneumothorax on nonoperative side.
- One-lung anaesthesia used while the chest is
  open.
- Use vasoconstrictor, not fluid, to treat epidural
  related hypotension.
- Test for air leaks as described for lobectomy.
- Maintenance of normothermia crucial to
  postoperative respiratory function. Underbody
  warming, fluid warming and use of heat and
  moisture exchangers mandatory.

POSTOPERATIVE MANAGEMENT

- Apical and basal drains placed at surgery
  (apical anterior to basal) with suction at 2 kPa
  and under water seal.
- Reverse relaxant.
- Extubate in the sitting position, breathing
  spontaneously.
- Administer humidified oxygen by face-mask.
- Adequate pain relief essential.
- Persistent air leak can be a problem.
- Incidence of recurrence of pneumothorax after
  pleurectomy very low.

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CROSS-REFERENCES

PNEUMONECTOMY

MATTHEW STAGG

Pneumonectomy is excision of a whole lung for lung cancer. It is performed via a posterolateral thoracotomy. It is higher risk than lobectomy with an operative mortality of over 6%.

PREOPERATIVE ASSESSMENT

- Standard for any major thoracic case.
- Blood must be rapidly available.
- Myasthenic syndrome may be present.

PREMEDICATION

- None needed.
- Full explanation about high-dependency care, postoperative monitoring and analgesia including benefits and risks of neuraxial blockade.

MONITORING

- Routine basic monitoring.
- Invasive arterial pressure.
- Central venous pressure.
- Core temperature.
- Arterial blood gases.
- Urine output (if epidural analgesia employed or high risk patient).

ANAESTHETIC TECHNIQUE

- General anaesthesia with volatile agent or TIVA supplemented with an opioid/relaxant. Remifentanil, atracurium and desflurane recommended. Nitrous oxide not advised.
- Regional block (thoracic epidural, paravertebral or epipleural) routinely used unless contraindicated.
- Rigid bronchoscopy usually performed first.
- Intubate with double-lumen endobronchial tube.
- One-lung anaesthesia used while the chest is open.
- Treat epidural hypotension with vasoconstrictor, not fluid.
- Catastrophic haemorrhage from pulmonary artery occurs occasionally.
- Integrity of the bronchial suture line tested prior to chest closure. Bronchial stump covered with sterile water and pressure up 30 cm H_2O exerted by manual compression of the rebreathing bag. Any leak detected as bubbles.
- Maintenance of normothermia crucial to postoperative respiratory function. Underbody warming, fluid warming and use of heat and moisture exchangers mandatory.

POSTOPERATIVE MANAGEMENT

- Basal drain placed at surgery.
- Ensure relaxant fully reversed (nerve stimulator).
- Extubate in the sitting position, breathing spontaneously.
- Administer humidified oxygen by face-mask.
- Restrict intravenous fluid.
- Use vasoconstrictor, not fluid, to treat epidural related hypotension.
- Never apply suction to a pneumonectomy drain. Most surgeons prefer to leave the drain clamped and to open it for 1 minute every hour to allow blood out. Drain removed after 24 hours.

COMPLICATIONS

- Haemorrhage into pleural space. Verify by unclamping drain. Replace volume lost. May need surgical exploration.
- Sputum retention, infection, acute lung injury and respiratory failure. This carries a high mortality after pneumonectomy. Treat with physiotherapy, antibiotics, ventilation and early tracheostomy.
- Infection in pneumonectomy space. This requires draining and may be associated with a bronchopleural fistula.
- Atrial fibrillation. Treat with beta-blockade ± amiodarone; digoxin usually ineffective.

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CROSS-REFERENCES

POSTOPERATIVE ANALGESIA

MATTHEW STAGG

Pain after thoracotomy is more intense than with any other incision. For this reason, regional block is commonly used, in conjunction with systemic analgesia if needed. All blocks except epidurals usually require additional systemic agents to achieve optimal analgesia. Blocks are usually used for the first three postoperative days. The catheter is then removed and systemic analgesia used. Chronic neuropathic pain is common after thoracotomy. Cryoanalgesia of intercostals nerves is no longer used as its use is associated with a high incidence of neuropathic pain.

SOURCES OF PAIN

- Chest wall and most of pleura via intercostal nerves.
- Diaphragmatic pleura via phrenic nerves.
- Mediastinal pleura via the vagus nerve.
- Shoulder joint via spinal nerves C5–C7.

AIMS OF ANALGESIA

- Reduce distress.
- Improve respiratory function and sputum clearance.
- Reduce complications and length of stay, and improve outcome.

ANALGESIC TECHNIQUES

REGIONAL BLOCKS

- Intercostal block
- Extrapleural block
- Intrapleural block
- Paravertebral block
- Epidural block

SYSTEMIC ANALGESIA

- Parenteral analgesics – opioids (usually PCA), paracetamol, non-steroidal anti-inflammatory agents (NSAIDs) and tramadol.

REGIONAL ANAESTHESIA

INTERCOSTAL NERVE BLOCK

- Simple to perform but 'single shot' therefore short duration of action.
- Does not control pain from diaphragmatic pleura, mediastinal structures and areas supplied by the posterior primary rami.

EXTRAPLEURAL BLOCK

- Indwelling catheter is placed in a pocket of retracted pleura so that the tip lies against a costovertebral joint.
- Local anaesthetic spreads to paravertebral space providing anaesthesia of both anterior and posterior primary rami.

INTRAPLEURAL BLOCK

- Local analgesic agent deposited between visceral and parietal pleura via indwelling catheter.
- Analgesic action due to widespread intercostal nerve block.
- Does not spread to paravertebral space.
- Analgesia unpredictable due to variable loss of drug into chest drains, binding with blood in thorax, and rapid systemic absorption.
- Cannot be used following pneumonectomy.

PARAVERTEBRAL BLOCK

- Percutaneously inserted catheter at one level allows considerable spread of drug between adjacent paravertebral spaces. Alternatively, multiple injections at different levels can be used as a 'one shot' technique usually coupled with extrapleural block for postoperative analgesia.
- Blocks both anterior and posterior primary rami.
- Provides good analgesia, possibly with less side-effects than epidural.
- Less easy to position accurately and maintain position compared to epidural.
- Main disadvantage is inferior reliability compared to epidural.

THORACIC EPIDURAL BLOCK

- Considered the gold standard.
- Height of required block necessitates thoracic approach (usually about T5–T6).
- Paramedian approach easier than midline.
- Use weak local anaesthetic solutions in combination with an opioid (usually 0.125% plain bupivacaine with fentanyl 2 mcg/mL by infusion).
- Provides excellent analgesia.
- Motor block rarely a problem.
- Urinary catheter required in most patients.
- Hypotension from sympathetic block must be treated with vasoconstrictor (such as norepinephrine infusion) rather than fluid provided the patient is not hypovolaemic.
- Patient-controlled epidural analgesia (PCEA) with boluses on top of background infusion better than infusion alone.

SYSTEMIC ANALGESIA

- If not using thoracic epidural, balanced analgesia with paracetamol, NSAID and opioid always required. This may be supplemented with tramadol.

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CROSS-REFERENCES

Bronchopleural fistula, Chapter 15
Lobectomy, Chapter 15
Pleurectomy, Chapter 15
Pneumonectomy, Chapter 15
Local anaesthetic toxicity, Chapter 30
Postoperative analgesia, Chapter 30

RIGID BRONCHOSCOPY

MATTHEW STAGG

Fibreoptic bronchoscopy has largely superseded rigid bronchoscopy for the diagnosis of lung disease. It is

used for therapeutic manoeuvres such as removal of a foreign body, stent insertion or debulking of airway tumour. Ventilation during bronchoscopy is achieved with a Venturi injector or ventilating bronchoscope. High-frequency jet ventilation is popular in some other countries but not used widely in the UK.

PREOPERATIVE ASSESSMENT AND INVESTIGATION

- Assess for tracheal obstruction or deviation with clinical examination (inspiratory stridor), chest X-ray (PA and lateral) or CT scan.

PREMEDICATION

- None needed.
- Continue cardiac and respiratory medication.
- Avoid sedatives if there is tracheal obstruction.

MONITORING

- Routine basic monitoring.

VENTILATION

VENTURI INJECTOR

- Usually used in adults.
- High pressure oxygen injected intermittently through injector port at proximal end of bronchoscope, entraining air.
- Proximal end of bronchoscope must be open to allow air entrainment during inspiration and egress during expiration. If upper airway is obstructed and egress is impossible, very serious barotrauma is likely.

VENTILATING BRONCHOSCOPE

- Used in infants and children in whom injector techniques are more likely to cause barotrauma.
- Glass lens covers proximal end of bronchoscope.

- T-piece circuit attached to side port and manual positive pressure ventilation started.
- Anaesthesia maintained with inhalational agents.

ANAESTHETIC TECHNIQUE

- Intravenous induction (adults), inhalational or intravenous induction (children).
- Short-acting muscle relaxant (suxamethonium, mivacurium).
- Maintenance of anaesthesia usually intravenous in adults (propofol and remifentanil) and inhalational (sevoflurane) in children.
- If bronchoscopy prolonged or followed by a surgical procedure, use longer acting muscle relaxant such as atracurium.

POST BRONCHOSCOPY MANAGEMENT

- Administer IPPV with oxygen by facemask and Guedel or laryngeal mask airway.
- Continue I/V anaesthesia and check for relaxant reversal (nerve stimulator).
- Waken sitting up or lying suppurative side down if secretions present.
- Be prepared to reintubate.

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Bronchial carcinoma, Chapter 1
Inhaled foreign body, Chapter 15
Mediastinal operations, Chapter 15
Preoperative assessment of pulmonary risk, Chapter 25

Cardiac surgery

16

AKBAR VOHRA

AORTIC VALVE SURGERY

AORTIC STENOSIS

Aortic stenosis (AS) can be either congenital (in which case the valve is abnormal and bicuspid in over 50% of cases) or acquired (usually from rheumatic involvement of a previously normal valve). In the absence of other valvular disease, AS is almost always congenital in origin. If it is of rheumatic aetiology, there is usually involvement of the mitral valve as well. The normal aortic valve area (AVA) is >2.0 cm². 'Critical' aortic stenosis has an AVA of <0.5 cm², 'severe' aortic stenosis has an AVA of <1.0 cm² and 'moderate' aortic stenosis has an AVA of 1.0–1.4 cm².

As aortic stenosis develops, there is a progressive increase in outflow obstruction to the left ventricle. Systolic pressures within the left ventricle rise and a pressure gradient develops between the left ventricle and the aorta. Increased systolic chamber pressure stimulates wall thickening and concentric ventricular hypertrophy. The consequences of this are twofold. Firstly, the ventricle relaxes poorly during diastole, so left ventricular end diastolic pressure (LVEDP) rises and higher filling pressures are needed to maintain cardiac output. The ventricle becomes increasingly dependent on atrial contraction to ensure diastolic filling and the atrium (in sinus rhythm) contributes up to 40% of LVEDV in AS compared to 10%–15% in normal patients. The sudden onset of atrial fibrillation (which suggests a rheumatic aetiology) can precipitate a major decrease in cardiac output. Secondly, the balance between myocardial oxygen supply and demand becomes precarious. This is because

increased myocardial bulk and high cavity pressures increase myocardial oxygen demand, whilst increased wall thickness and raised LVEDP predispose to subendocardial ischaemia. The relationship between diastolic time (determined by HR), LVEDP, and systemic diastolic pressure available for coronary perfusion (determined by cardiac output and systemic vascular resistance) is therefore critical. Coincident coronary artery disease is a serious added risk factor for these patients.

With aortic stenosis there is usually a long (can be up to 50 years or more) asymptomatic period and sudden death may be the first presenting feature. The most common symptoms are syncope, angina, dyspnoea and dysrhythmias. When symptoms finally occur, the stenosis is severe. Their significance, particularly signs of left ventricular failure, is ominous and if the stenosis is not surgically corrected death occurs within a few years.

The ECG will show LVH if aortic stenosis is significant, often with ST segment changes of left ventricular strain. Unless there is left ventricular failure (LVF) the chest X-ray will show a normal cardiac transverse diameter. If LVF has supervened, there will be cardiomegaly and lung field changes. Specialist investigation is by coronary angiography and ultrasound.

GENERAL ANAESTHETIC PRINCIPLES

Anaesthetic technique is similar to that described for coronary artery bypass grafting (CABG). The physiological objective is to maintain the basic haemodynamic state by carefully managing heart rate, filling pressure and systemic blood pressure. Give antibiotic prophylaxis.

Hypotension is very dangerous. Caused by low cardiac output, hypovolaemia or vasodilatation, it implies that a ventricle generating high intracavity pressures is being perfused by a low pressure arterial system. Immediate correction with an alpha agonist is needed whilst the underlying cause is remedied.

Tachycardia is dangerous. Myocardial ischaemia (sometimes acute LVF) results with reduced cardiac output from increasing dynamic impedance of stenosis. Treat cause (light anaesthesia, hypovolaemia). Give beta-blockers with caution because of the risk of reduction in cardiac contractility. Persistent

dysrhythmias affecting cardiac output may need synchronised cardioversion.

Moderate degrees of bradycardia are tolerated. Dynamic impedance of stenosis is reduced. If severe with very low diastolic pressures, use tiny doses of glycopyrollate and avoid overcorrection at all costs.

Preload on left ventricle must be maintained to ensure filling of hypertrophied ventricle.

Changes in afterload on left ventricle have little effect on valve pressure gradient and hence LV load, but the effect on systemic blood pressure in the aortic root significantly changes coronary perfusion.

MONITORING

Invasive arterial monitoring is mandatory from before induction. ECG monitoring must be able to detect left ventricular ischaemia and diagnose dysrhythmias; use leads V_5 and II. In practice, it may be difficult to interpret 'ischaemic' changes due to pre-existing ST abnormalities caused by LVH (strain pattern).

Central venous pressure (CVP) is a poor indicator of left ventricular filling when left ventricular compliance is reduced. A flotation catheter, however, may cause severe and persistent dysrhythmias as it passes through the right ventricle. Transoesophageal echocardiography is an excellent method for assessing ventricular filling and should be used in these patients. It is also important in helping to assess mitral valve function following valve replacement.

Persistent ischaemia in the face of appropriate corrective measures necessitates early institution of cardiopulmonary bypass. In the event of cardiac arrest, defibrillate immediately. Only internal massage is effective because of valve stenosis, and emergency bypass may be required. Do not commence anaesthesia unless bypass facilities are immediately available.

Intraoperative care, management of bypass and postoperative care are as described elsewhere for CABG.

AORTIC REGURGITATION

Aortic regurgitation may be acute or chronic. Chronic causes include rheumatic valve disease, connective-tissue disorders or congenital bicuspid valve. Acute aortic regurgitation is most commonly caused by infective endocarditis or trauma. The basic problem is

volume overload of the left ventricle caused by blood leaking through the incompetent aortic valve during diastole. The degree of regurgitation is determined by the size of the regurgitant orifice and the diastolic time interval. Systemic vasodilatation, increased inotropy and tachycardia all contribute to increased forward flow and may explain the phenomenon of mild exercise tolerance with symptoms at rest. Over a period of time, eccentric left ventricular hypertrophy, gross cardiomegaly and impaired oxygen supply result.

Mild to moderate degrees of chronic regurgitation are well tolerated and there is a long asymptomatic period. Symptoms, when they arise, are usually those of LVF or angina. The life expectancy of patients with significant aortic regurgitation is about 9 years. Sudden death is rare. In acute aortic regurgitation, there is a sudden volume overload of the left ventricle with a dramatic rise in the LVEDP. Ventricular dilatation enlarges the mitral valve annulus resulting in functional mitral regurgitation. Pulmonary oedema is marked and refractory. Very severe aortic regurgitation with gross distortion of the valve ring can result in dissection, which may involve the coronary arteries.

GENERAL ANAESTHETIC PRINCIPLES

Anaesthetic technique is as for CABG. Only severe aortic regurgitation is a major anaesthetic risk. Remember antibiotic prophylaxis.

Bradycardia allows time for back flow into the ventricle and increases regurgitant fraction. Treat carefully with glycopyrollate or very small dose of epinephrine, dobutamine or isoprenaline.

Tachycardia if mild is well tolerated because it increases dynamic impedance of reverse flow through valve.

Preload needs to be maintained to keep the dilated ventricle full.

Afterload needs to be kept low to enhance forward flow. A balance has to be found between good cardiac output and an aortic perfusion pressure adequate to perfuse the coronary arteries of the dilated ventricle.

Monitoring

As for aortic stenosis. In severe cases, use of a pulmonary artery flotation catheter allows cardiac output

to be optimised by afterload reduction, whilst maintaining preload by titrating fluid replacement to pulmonary artery capillary wedge pressure (PCWP). However, as with most cardiac cases, TOE is the easiest way of assessing ventricular filling and ejection and has superseded the use of the PAC.

Principles of intra- and postoperative management are as for CABG and ITU care.

CROSS-REFERENCES

Aortic valve disease, Chapter 2
Transoesophageal ECHO, Chapter 16
Elective surgery, Chapter 25
Premedication, Chapter 25

CARDIOPULMONARY BYPASS: PRINCIPLES, PHYSIOLOGY AND BIOCHEMISTRY

The objective of cardiopulmonary bypass (CPB) is to allow surgery on the heart and great vessels whilst the rest of the body is perfused with oxygenated blood and the products of metabolism are removed. Bypass necessitates anticoagulation with heparin (usually 3–4 mg kg^{-1}), and haemodilution is a consequence because of the need to prime the circuit with up to 2 L of fluid. Abnormal surface interactions between blood, air and plastics damage cells, particularly platelets, and denature proteins which can release pro-inflammatory agents. Air and particulate microemboli may enter the bypass circuit via suction, so blood filters are essential.

THE CPB CIRCUIT

This is shown diagrammatically in Figure 16.1. Venous cannulae are inserted into the right atrium, the venae cavae, or (more rarely) the femoral vein or pulmonary artery. The large-bore venous return line drains blood, under gravity, from the patient on the operating table to the reservoir of the bypass machine on the floor. The reservoir is either a rigid casing or a bag (soft-cell). Blood is then pumped through the oxygenator, where it is oxygenated and carbon dioxide is removed. In addition, a heat exchanger allows
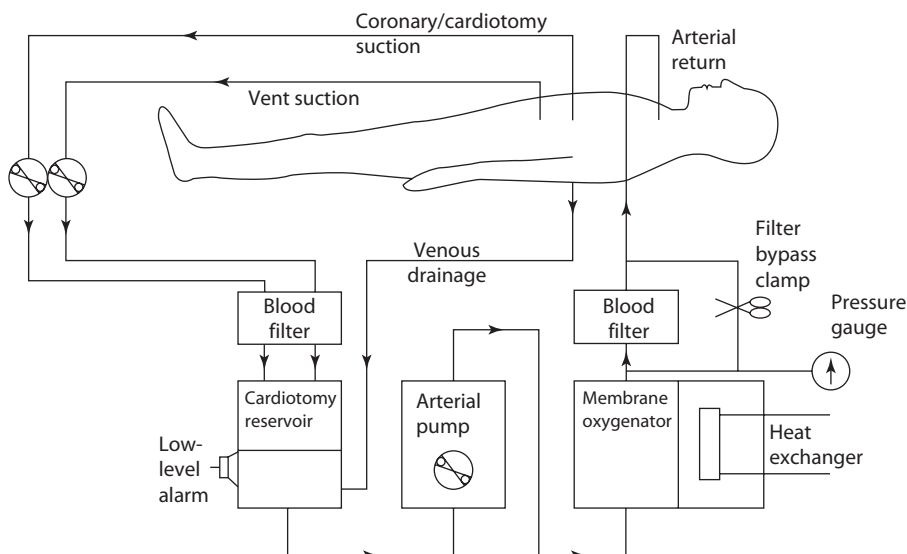
Figure 16.1 The cardiopulmonary bypass circuit.

heating and cooling of the blood. The pumps driving the flow may be compression roller devices or use centrifugal force (centrifugal pumps). The blood is returned to the body via an aortic or (more rarely) a femoral cannula after passing through a filter. The arterial cannula is always inserted first and is the last to be removed. There are a number of scenarios where femoral venous and arterial cannulation is the site of choice:

1. In patients in whom there is potential for difficulty with easy access to the heart via sternotomy with potential for damage to the (usually) right ventricle. This is the case in patients undergoing repeat sternotomy.
2. In patients undergoing complex mediastinal surgery where there is a potential for airway or circulatory loss.
3. Rewarming cardiac arrest patients that have become extremely cold.

In some of these cases the femoral artery and vein are cannulated without CPB being instituted (bypass on standby).

There are additional auxiliary roller pumps, which feed blood into the circuit. One (sometimes called the 'coronary sucker' or 'cardiotomy sucker') acts as a sucker to return blood in the operative field to the bypass circuit. The other (sometimes called the 'vent') aspirates gently from the left ventricle, or pulmonary artery to prevent ventricular distension, and from the aortic root when retrograde cardioplegia is used. During bypass when the heart is arrested and there is no effective ejection, blood draining from the bronchial and thesebian veins and retrogradely across the aortic valve collects in the left ventricle. Ventricular distension causes mechanical damage, impairs subendocardial perfusion and can result in subendocardial infarction.

Poor venous return is occasionally a problem and can result from kinks, air locks, drainage tube malposition, decreased circulating blood volume, sequestration of blood into body cavities or into the tissues or from vasodilator therapy. Venous return can also be improved by raising the height of the operating table to increase the hydrostatic gradient from the patient to the reservoir. Occasionally suction can be added to the reservoir to increase venous return, but this has to be performed with caution because the suction can cause damage to the venous wall. In addition, when the surgeon operating on the circumflex arteries rotates the heart, not only can venous return be impaired, but also venous drainage from the brain may be sufficiently obstructed

to cause jugular venous hypertension with an acute rise in central venous pressure. This obviously needs immediate correction. Complications of aortic cannulation are embolization (from air and wall debris), haemorrhage, dissection and malposition (abutting the aortic wall), and high arterial supply line pressures in the presence of normal flow rates can detect this. It is usually amenable to repositioning. Complications of venous cannulae are haemorrhage, reduction of venous return, arrhythmias and atrial or caval damage.

MANAGEMENT OF ANTICOAGULATION

The activated clotting time (ACT) should be kept three times greater than the baseline, or over 450 s using heparin. The patient must be anticoagulated adequately before the arterial return cannula is inserted, or a clot will form on its tip and within its lumen. At the end of bypass, the action of the heparin is reversed with protamine. Protamine is a potent vasodilator and decreases in blood pressure should be anticipated and treated. No blood must be returned to the pump via suction after this. If it is, clot formation can occur in the pump making an emergency return to bypass impossible and preventing the blood being used later for auto transfusion.

Aprotinin was frequently used for patients with a high risk of bleeding (500,000–2,000,000 KIU to the patient, 2,000,000 KIU to the pump and occasionally 500,000 KIU.h^{-1} infusion). Aprotinin was temporarily withdrawn due to suggested adverse events but is now being used again. Some units routinely use tranexamic acid, which is also effective at reducing bleeding (2 g bolus followed by infusion at 1 g/h, or 10 g boluses prior to sternotomy). Problems of graft occlusion associated with these two drugs are as yet inconclusive but at least one of them is routinely used to reduce bleeding in the perioperative period.

Anaemia management and preoperative iron therapy is gathering importance and attention. Patients found to be anaemic preoperatively should be treated with iron therapy if they are found to have iron depletion. Single shot IV bolus is the best method because compliance with oral therapy is poor. Recent data would suggest that the postoperative

transfusion threshold of 9 g/dL is associated with reduced morbidity and mortality.

THROMBOELASTOGRAPHY (TEG)

TEG is a method of evaluating the formation and breakdown of a clot. A small amount of blood is placed into the machine to obtain values for the following parameters:

- R time is the time to initial fibrin formation.
- K time is the speed to reach a certain level of clot strength.
- Alpha is the rapidity of fibrin build-up and cross-linking (clot strengthening).
- Maximum amplitude (MA) represents the ultimate strength of the fibrin clot.
- LY30 is the rate of amplitude reduction 30 minutes after MA.

It may be used to evaluate platelet function, plasma factors and activators/inhibitors of coagulation and serves as a guide to appropriate therapy for postoperative bleeding. However, standard cups will not indicate the level of platelet inhibition due to aspirin or clopidogrel. Specialised cups for aspirin and clopidogrel effect are available. Newer cartridge-based TEG machines are making the process easier to use.

TEMPERATURE, FLOW AND PRESSURE

There are many controversies surrounding the management of CPB. For example, some centres routinely use normothermic bypass whenever possible, whilst others invariably use hypothermic bypass. The advantages claimed for normothermic bypass are reduced bypass time (no cooling or warming needed), reduction in postoperative clotting dysfunction and reduced postoperative cooling (afterdrop) and shivering with its increased metabolic demand and tissue hypoxia. For normothermic bypass, flow rates of greater than 2.2 L min^{-1} m^{-2} are usually used. Hypothermic bypass reduces metabolism and oxygen consumption falls by a factor of 2.5 (known as the Q10), for every 10°C fall in temperature. Moderate hypothermia (25–30°C) is commonly used for adult coronary and valve work, with an associated reduction in flow rate. For some operations (e.g. on the aortic arch), circulatory arrest

is needed. The brain will tolerate approximately 1 h of arrest during deep hypothermia at 15–18°C. Even moderate hypothermia permits a brief (approximately 12 min) period of circulatory arrest, which may be life-saving if there is a catastrophic mechanical failure or circuit disruption. There is a general acceptance that maintaining temperature is good for clotting function and an increasing number of surgeons are tending to maintain higher temperatures (normothermia or drifting to 34/35°C) than previously.

Management protocols vary considerably in relation to perfusion pressure to be maintained on bypass. When the aorta is clamped and the heart protected by cardioplegia as far as the heart is concerned myocardial perfusion pressure is irrelevant. If the aorta is not cross-clamped, the heart is least likely to suffer ischaemic damage if it is kept empty, beating and well perfused. During intermittent cross-clamping techniques, the fibrillating ventricle consumes more oxygen than the beating but unloaded heart. At normothermia, with autoregulation, cerebral blood flow is maintained at 50 mmHg mean perfusion pressure. Under hypothermia, with reduced metabolic demand, lower perfusion pressures can be tolerated and many regard 40 mmHg as satisfactory. A number of retrospective studies indicate no relationship between hypotension during bypass and postoperative neurological dysfunction. There is a greater correlation with ascending aorta calcification. Typical UK practice is to fix the flow rate and then keep the mean perfusion pressure 50–80 mmHg. Pharmacological management of blood pressure centres on the use of alpha agonists (metaraminol, phenylephrine, norepinephrine) and smooth-muscle relaxants (sodium nitroprusside, glyceryl trinitrate and phentolamine). Infrared spectrometry is often used to give an indication of cerebral perfusion. Increase in flow or pressure may be performed to improve cerebral oxymetry saturations.

The use of pulsatile flow is controversial and outcome data on its benefits are lacking. Physiological models would suggest that such flow improves perfusion and oxygen uptake; achieving it requires intermittent roller pump action or external reservoir compression, both of which add complexity to the circuit. It is not commonly used.

BIOCHEMICAL AND HAEMATOLOGICAL CONTROL OF CPB

For adult patients the CPB is primed with 1.5–2 L of balanced salt solution (e.g. Gelofusin 1000 mL plus Hartman's solution 800 mL and 200 mL mannitol 10%), to which heparin is added. This causes significant haemodilution with a reduction in the total oxygen carrying capacity. On the other hand, by reducing viscosity, haemodilution improves blood rheology and prevents microcirculatory sludging during hypothermia. Most anaesthetists consider a haematocrit of >15% to be satisfactory for bypass. Attention to methods of reduction of anaemia of bypass is gaining attention. Circuit sizes can be reduced by minimising length of bypass tubing. In addition, blood can be drawn back into the circuit via the aortic pipe in order to reduce some of the crystalloid volume of the circuit (sometimes called wrapping).

Oxygen flow into the oxygenator should be sufficient to maintain an arterial PaO_2 of over 14 kPa. If this produces unacceptable hypocapnia, then carbon dioxide will need to be added to the oxygenator gas flow to reduce carbon dioxide washout. Many centres now use inline electrodes to monitor both arterial and venous blood gas status, the oxygen saturation in the venous line being used to confirm acceptable blood flow rates, with the A–V difference across the pump confirming satisfactory oxygenator function. Guidelines for minimal monitoring during bypass must be followed.

There is controversy over the optimum method of blood gas management during hypothermic CPB. The alpha stat method aims to achieve a pH of 7.4 and a $PaCO_2$ of 40 mmHg when blood drawn from the hypothermic patient is measured at 37°C: the pH stat method aims to achieve a pH of 7.4 and a $PaCO_2$ of 40 mmHg when blood drawn from the hypothermic patient is measured at the *in vivo* temperature. Alpha stat management is thought to preserve autoregulation and coupling of flow and metabolism in the brain better than pH stat, and therefore is currently gaining favour.

The most important electrolyte to monitor on CPB is potassium because correct levels optimize contractility and suppress dysrhythmias. High levels can be reduced by haemofiltration, diuresis, and insulin/glucose and countered by calcium. Low levels can be

corrected by giving incremental doses of 10–20 mmol of potassium chloride. These need correcting near the end of the bypass phase, just prior to coming off bypass.

TEMPERATURE MEASUREMENTS ON BYPASS

It is vital to measure the body temperature on bypass. The temperature of the nasopharynx is generally used to approximate to that of the brain. Thorough rewarming is essential. Normal central blood temperatures at the end of bypass after the patient has been rewarmed do not, however, represent the temperature in peripheral, poorly perfused tissues. After the discontinuation of CPB an 'afterdrop' is usually seen, which is caused by the opening up of cold, vasoconstricted tissue beds, particularly muscle. This can lead to post-bypass shivering with its high metabolic load.

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CONGENITAL HEART DISEASE (CHD)

Many patients with CHD are now surviving beyond childhood and presenting for revision surgery to regional centres. Common surgical interventions include revision surgery for pulmonary valve incompetence or stenosis. Right ventricular outflow obstruction at the sub-valvular or pulmonary artery level requiring new conduits and valve replacement are necessary. Residual atrial and ventricular septal defects are also relatively common. The usual problems of surgery for revision cardiac surgery apply. These patients are usually young adults and often have other medical syndromes.

Congenital heart disease first needs to be classified and the basic principles of anaesthesia for cardiac surgery explained. An understanding of the lesions and surgery are also helpful when anaesthetising children with heart disease for noncardiac surgery.

EPIDEMIOLOGY

The incidence is 4–10 per 1000 live births:

Ventricular septal defect (VSD)	20%
Atrial septal defect (ASD)	10%
Tetralogy of Fallot (TOF)	6%–10%
Transposition of the great arteries (TGA)	5%
Coarctation	5%

There is an association with other 'midline' abnormalities, e.g. tracheo-oesophageal fistula and imperforate anus. It can also occur as part of a number of syndromes including those with airway involvement, e.g. Down (40%) and Pierre–Robin. Congenital heart

disease may result from teratogenic exposure, e.g. maternal alcohol (25%) and rubella (35%).

SHUNTS

Blood moves between the oxygenated and deoxygenated sides of the circulation.

LEFT-TO-RIGHT

- ASD
- VSD
- Atrioventricular septal defect (AVSD)
- Patent ductus arteriosus (PDA)
- Partial anomalous pulmonary venous drainage (PAPVD)

RIGHT-TO-LEFT

- TOF
- Double outlet right ventricle (DORV)
- TGA
- Total anomalous pulmonary venous drainage (TAPVD)
- Pulmonary atresia (PA)
- Tricuspid atresia
- Truncus arteriosus

OBSTRUCTIVE LESIONS

- Aortic coarctation
- Aortic stenosis (valvar, subvalvar and supravalvar)
- Pulmonary stenosis
- Interrupted aortic arch

SINGLE VENTRICLE

- Congenital, e.g. HLHS, DILV
- Acquired as result of a surgical strategy, e.g. Glenn, Fontan

OTHER CONGENITAL HEART DISEASES

- Congenital complete heart block
- Inherited cardiomyopathies

ADULT CONGENITAL HEART DISEASE (ACHD)

Soon there will be more adults with CHD than children. This is because of the improvements in their care with better surgery and better facilities. These patients will present to regional ACHD centers for follow-up and appropriate interventional therapy including the management of pregnancy. They will present to the anaesthetist for a number of different procedures.

The most common attendees include:

- ASD or VSD 22%
- TOF 14%
- Complex lesions 13%
- LVOTO 12%
- TGA 10%
- RVOTO 8%
- Coarctation of the aorta (CoA) 7%
- Marfan syndrome 5%
- Congenitally corrected TGA (ccTGA) 4%
- Atrioventricular septal defect (AVSD) 3%
- Eisenmenger syndrome 3%

The most common procedure in patients with previous cardiac surgery as a child is usually surgery on the RVOT, pulmonary valve or the pulmonary artery. Some of these procedures can be performed with minimally invasive intervention in the catheter laboratory. Many will present for formal surgical correction.

GENERAL PRINCIPLES

Minor lesions can present as an incidental finding, such as a heart murmur on routine cardiac examination. Neonates form 25% of the practice, many of which have duct-dependent disease and are maintained on intravenous prostaglandin E1 (Prostin). These are often antenatally diagnosed with improvements in screening and imaging.

PALLIATION VERSUS REPAIR

Repair implies a return to normal physiology and normal life expectancy. Palliative surgery aims to improve quality and quantity of life, or allow growth so that a more definitive repair can be attained later.

DIFFERENCES BETWEEN CHILDREN AND ADULTS

Children are smaller and have immature physiology and pharmacokinetics, especially neonates. The cardiac pathology is more varied and there is less comorbidity. Palliative procedures and 'open' surgery are more common and cyanosis and pulmonary hypertension are more often present.

CPB IN CHILDREN

Haemodilution is greater (the smallest pump prime volume possible is around 300 mL) and multiple venous cannulae are often required. Aorto-pulmonary collaterals may be present, affecting emptying of the heart. Deep hypothermic arrest is more commonly used. Modified ultrafiltration (MUF) is used in small children to remove excess fluid and inflammatory mediators thereby reducing transfusion requirements and lung injury.

PREOPERATIVE ASSESSMENT

HISTORY – SPECIFIC POINTS

- Cardiac symptoms.
  - Failure to thrive, tachypnoea, poor feeding and sweating are all features of cardiac failure.
  - Squatting and blue lips may signify cyanotic spells.
  - Blackouts and chest pain may occur, although a history of pain is difficult to obtain in the younger child.
  - Drug history is important especially for cardiac medication and anticoagulant therapy.
  - Allergies.

EXAMINATION – SPECIFIC POINTS

- Cardiac signs
  - Shortness of breath, sweating, tachycardia, raised JVP and hepatomegaly in cardiac failure.
  - Blood pressure in all four limbs in the presence of a coarctation.
  - Saturation measured pre- and postductally.
  - Capillary refill time is useful, especially if the patient is acutely unwell.
  - Features of coexisting diseases or syndromes.
  - Assessment of the respiratory system and any end-organ damage. If airway difficulties are anticipated, a detailed assessment should be undertaken prior to anaesthesia.

INVESTIGATION

- ECG
- Echocardiogram
- Reports from catheter studies/cardiac CT/cardiac MRI
- CXR
- FBC
- Renal especially if taking diuretics or antihypertensive therapy
- Clotting
- Blood cross-match

ANAESTHETIC CONSIDERATIONS

During the preoperative visit, the child and parents are given a detailed explanation of the anaesthetic and postoperative care including:

- Requirement for premedication
- Type of induction
- Monitoring, including TOE if required
- Blood transfusion
- Postoperative ICU
- Analgesia and sedation

CONDUCT OF ANAESTHESIA

Intravenous or inhalational induction is used although inhalational is preferred in small children and neonates. Maintenance is usually with moderate to high dose fentanyl (10 to 50 mcg/kg) and inhalational agent. Both isoflurane and sevoflurane have ischaemic preconditioning properties. Long-acting muscle relaxants can be used for long cases where postoperative ventilation is expected. Inotropes may be required pre- or post-cardiopulmonary bypass, depending on the type of surgery.

MONITORING

Standard AAGBI monitoring and also:

- 3 or 5 lead ECG
- Pulse oximetry (pre- and postductal may be required)
- End tidal capnography (beware large arterial to end tidal gradient with low pulmonary blood flow states)
- Ventilation and agent monitoring
- Urine output
- Core and peripheral temperature
- Arterial line (site may be important if duct-dependent, Blalock Taussig (BT) shunt or in the presence of vascular abnormalities)
- Central venous line (usually right internal jugular or femoral)
- Surgically sited lines, e.g. LA or PA
- Near infrared spectroscopy (NIRS) if indicated

TOE

This is becoming increasingly the standard of care for paediatric bypass cases. Paediatric 8 Hz multiplane probes are suitable >3 kg. TOE is used to assess anatomy pre- and post-bypass, to look for air after cardiotomy, and to estimate cardiac function. Complications are rare.

SPECIAL CIRCUMSTANCES

NEONATES

Neonates often have duct-dependent lesions. They have immature physiology, pharmacology and haemostasis. The myocardium is sensitive to extracellular calcium concentration and they have a fixed, rate-dependent cardiac output. On bypass there is massive dilution of haemoglobin and clotting factors.

REPEAT STERNOTOMY

Congenital lesions are often repaired or palliated in stages. Scar tissue forms during healing and components of the heart may become attached to the back of the sternum and be damaged during reopening. Bleeding is expected and ventricular fibrillation can occur due to excessive diathermy near to the heart. The patient must be prepared for emergency initiation of bypass via the femoral vessels if the heart becomes damaged on opening. Externally placed defibrillation pads are used.

FAST-TRACK CARDIAC SURGERY

Simple surgery may be suitable for a shorter intensive care stay, including lesions such as ASD, VSD, and conduit replacements. Early extubation is performed, either in theatre or early on ICU and anaesthesia needs to be tailored to allow this.

CYANOTIC PATIENTS

Right to left shunts result in cyanosis and if this is chronic, patients can become polycythaemic and coagulopathic through a variety of mechanisms. There is a risk of thrombosis and cerebral infarction if the haematocrit is >60%. Avoiding long preoperative fasting and dehydration is important.

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Children and infants, Chapter 24
Neonates, Chapter 24

CORONARY ARTERY BYPASS GRAFTING

Coronary artery disease (CAD) is the leading cause of death in Western societies and coronary artery bypass graft (CABG) surgery comprises 50%–60% of most cardiac surgical programmes. The heart extracts

oxygen to a greater extent than any other organ with only minimal increases in oxygen extraction possible; therefore, any increase in oxygen demand must be met by increasing flow. In health this is done by autoregulation and in the absence of CAD maximal flow is four to five times as great as at rest. The coronary arteries arborize on the surface of the heart to form a mass of smaller epicardial arteries from which 'B' branches perforate directly through the myocardium to reach the endocardium. These vessels are subject to torsion and pressure during muscular contraction which in the left ventricle results in the majority of useful myocardial perfusion occurring during diastole. The only collateral circulation exists at subendocardial level and becomes of importance if there is a blockage in an epicardial vessel. Patients with classic CAD are asymptomatic at rest. As the severity of the stenosis increases, coronary flow reserve declines, resting blood flow is preserved by progressive vasodilation of the microcirculation, and the onset of angina of effort occurs with increased demand. CABG aims to bypass epicardial blockages using either the internal mammary artery or with vein grafts taken from the leg and so increase myocardial blood flow and oxygen delivery.

GENERAL ANAESTHETIC CONSIDERATIONS

PREOPERATIVE ASSESSMENT

History should concentrate on the symptoms of ischaemic heart disease, i.e. degree of angina pectoris (Canadian Heart Association Classification) and, when it occurs, previous infarction, exercise tolerance, shortness of breath and orthopnoea leading to functional debility (American Heart Association Classification). Diabetes mellitus, renal disease, hypertension, vascular disease and pulmonary disease are common associated problems. These are all added risk factors for CABG patients. Knowledge of perioperative medication is essential with antianginal agents and anticoagulant or platelet-inhibiting drugs being of particular importance. While the patient should continue with the usual antihypertensive and/or antidysrhythmic medication, platelet-inhibiting drugs, such as aspirin and clopidogrel, should be stopped one week prior to

surgery to avoid antiplatelet effects at operation. ACE inhibitors have been associated with low SVR perioperatively and should, therefore, be omitted on the day of surgery.

On examination, physical findings are often few in this group of patients but look for signs of right and left ventricular failure and check the arterial pulses. If there is a marked difference in right- and left-sided pulses, one should monitor from the strongest, as this will be the better reflection of aortic root pressure. If carotid bruits or stenosis are present, make a note to avoid jugular lines on that side.

Investigations involve routine preoperative tests which include urea and electrolytes, haemoglobin, chest X-ray, ECG, as well as more invasive procedures, e.g. cardiac catheterization. The latter can give information on left ventricular function and ejection fraction. A low ejection fraction, the presence of ventricular dys-synergy, high left ventricular end diastolic pressure (LVEDP) or pulmonary hypertension suggest a strong possibility of post-CABG myocardial dysfunction. All patients must have a baseline 12-lead ECG for postoperative comparison.

PREMEDICATION

The purpose is to reduce apprehension, fear and the stress of painful events, e.g. insertion of intra-arterial catheter prior to induction. Commonly used drugs include benzodiazepines (e.g. oral lorazepam, diazepam or temazepam) and intramuscular opiates (e.g. morphine). Metoclopramide and ranitidine may be used to reduce the volume and acidity of the gastric contents. Premedication should be supplemented with face-mask oxygen in a safe clinical environment. Remember antibiotic prophylaxis.

MONITORING

Establish good peripheral venous access, an arterial line and a multilead ECG (leads II and V5). Following induction, insert a central venous catheter (internal jugular or subclavian), temperature probes and urinary catheter. The use of a pulmonary artery catheter (PAC) varies from centre to centre, but one would be indicated in patients with abnormal left ventricular function, recent myocardial infarction or post-infarction sequelae, e.g. ventricular septal

defect, left ventricular aneurysm, mitral regurgitation. In some patients who may not need a PAC it is sensible to insert a PAC sheath to facilitate easy insertion of a PAC postoperatively if required. Transoesophageal echocardiography use is increasing, almost routine in some units and mandatory for mitral valve repair surgery. It appears to have superseded the use of the PAC.

ANAESTHESIA

The fundamental principle is to minimize myocardial ischaemia and prevent awareness. It is probably the experience of use of the available drugs which is of greater importance than the particular agent itself. Following preoxygenation, induction of anaesthesia needs to be smooth with cardiovascular stability, but simultaneously adequate to prevent the sympathetic response to laryngoscopy and intubation. Agents that have been used successfully for induction with or without opioid supplementation include: propofol, etomidate, ketamine and thiopental. Some centres use high-dose opioid techniques (fentanyl 50 mcg kg^{-1}) for the whole procedure, with very modest benzodiazepine supplementation. Shorter-acting agents such as remifentanil, alfentanil and sufentanil are also commonly used.

Probably the most commonly used induction technique in UK practice is a slow bolus of opioid (fentanyl 10–15 mcg kg^{-1}) followed by a small dose of induction agent. Maintenance is usually provided by an opioid infusion (e.g. remifentanil 1–3 mcg kg^{-1} min^{-1} or alfentanil 50 mcg kg^{-1} h^{-1}), combined with a low dose of either an inhalational or intravenous agent. Propofol by infusion can be used for maintenance at a rate of 5–6 mg kg^{-1} h^{-1} or as target controlled infusions (TCI) to achieve adequate serum levels during bypass. Of the inhalational agents, isoflurane has received much attention because its known vasodilator properties have been implicated in causing myocardial ischaemia through the coronary steal mechanism. However, this has been shown not to be of relevance at concentrations less than 1.5% and may even provide protection against myocardial ischaemia. Both enflurane and halothane have been and are still used for maintenance although halothane is not now available in the UK. Isoflurane is probably the most commonly used. If there is poor urine flow

in the presence of an adequate circulating fluid volume and blood pressure consider furosemide (bolus or infusion). Any of the currently available muscle relaxants may be used. The cardiovascular side effects of pancuronium may be beneficial in counteracting the bradycardia caused by opioids. Rocuronium is associated with very little cardiovascular effect and is becoming the relaxant of choice. Vecuronium is associated with an increased incidence of bradycardia.

During dissection of the internal mammary artery one should observe the blood pressure during insertion of the Chevalier retractor. There can be a genuine fall due to right ventricular compression or (if the arterial cannula is in the ipsilateral arm) an artefactual fall due to stretching of the subclavian artery. PEEP may interfere with the dissection of the mammary artery and surgeons will invariably ask for it to be avoided during this period. Adjust serum potassium to between 4.5 and 5.5 mmol L^{-1} prior to cessation of bypass.

In addition to the goals of providing anaesthesia and muscle relaxation, in the pre- and post-bypass periods, myocardial ischaemia must be minimized and this requires constant observation of the ECG. If ischaemic changes occur (which can be manifest by dysrhythmias and low cardiac output as well as by ST segment changes), always make sure immediately that the patient is ventilating well with a satisfactory FiO$_2$ and SpO$_2$. The mainstays of good cardiovascular management are meticulous attention to fluid balance (using information from direct visual inspection of the heart, central venous pressure, PAC [if present], left atrial catheter [if present] and surgical palpation of the pulmonary artery), and tight control of the cardiac output and blood pressure. The latter can best be achieved by the appropriate combination of vasodilators (glyceryl trinitrate, sodium nitroprusside, phentolamine), inotropes (enoximone, milrinone, dobutamine, epinephrine), vasopressors (norepinephrine, metaraminol) and an inhalational anaesthetic agent. In the absence of severe hypotension or tachycardia causing poor coronary perfusion, glyceryl trinitrate infusion can be used to treat ischaemic changes. Rapid elevations in diastolic coronary perfusion pressure are easy to achieve with short-acting alpha agonists (e.g. metaraminol), but these do of course also increase systolic work and may constrict vital microvasculature. The key to

post-bypass care is to optimize the intravascular volume by transfusion from the pump and then, if the cardiac output and blood pressure are still poor, to introduce an inotropic agent. TOE is invaluable for assessing ventricular function and identifying wall motion abnormalities.

Following the cessation of CPB and the heart taking over the circulation, the true cardiac performance is usually not seen for several minutes because it often has energy stores within the myocardium which accumulated whilst it was perfused and beating but with no load on full bypass. A heart that had good initial ventricular performance and was satisfactorily grafted can be expected to continue to beat well. The heart can, however, be disadvantaged by poor myocardial protection during CPB and/or by reperfusion injury. This can result in the 'stunned myocardium' (diastolic LV dysfunction) which functions badly and needs a period of hours to days to recover fully. Perioperative infarcts may also occur. In both these situations inotropes are usually necessary. If drug therapy shows no alleviation of the ischaemic picture (continued ST segment change, reduced cardiac output, elevation of pulmonary artery wedge pressure [PAWP], increasing acidosis), it is usually an indication for a balloon pump. This is the only treatment modality that simultaneously increases myocardial oxygen supply whilst decreasing demand. In the immediate post-bypass period calcium antagonists and beta blockers are relatively contraindicated and must be used with extreme caution since their negative inotropic effects are both magnified and unpredictable.

OFF-PUMP CORONARY ARTERY BYPASS SURGERY (OPCAB)

This involves the use of stabilising devices such as the Octopus 3 (Medtronic Inc, Minneapolis, MN). The grafts to the coronary arteries are performed on the beating heart with that area stabilised by the use of the Octopus, so named because it has eight suction pads which attach to the heart and fix that small area. The assistant blows carbon dioxide ($2–4$ L min^{-1}) on the site of coronary suturing, to ensure a relatively bloodless field. Patients may be turned to various head-down and lateral positions to optimise the view for the surgeon. Slow heart rates

($50–60$ beats min^{-1}) would be ideal in these patients. This technique avoids the complications associated with bypass. There is less haemodilution, reduction in clotting dysfunction and possibly fewer respiratory complications. Postoperative bleeding and reopening have not shown to be reduced. There have been mixed reports about reduction in postoperative renal dysfunction. Measures for reducing perioperative hypothermia are important.

There have been concerns about the quality of the bottom-ends of the coronary grafts with OPCAB surgery and good target vessels are imperative. Many surgeons would consider three grafts to be the threshold for OPCAB surgery. The patients are always heparinised but with lower doses (1.5 mg kg^{-1}). There is clearly a greater need for interaction between the anaesthetist and surgeon during the grafting. Combined general and epidural anaesthesia may be advantageous for providing slow heart rates. Some centres have used sole epidural anaesthesia for OPCAB surgery.

CROSS-REFERENCES

Coronary artery disease, Chapter 2
Cardiopulmonary bypass: principles, physiology and biochemistry, Chapter 16

MANAGEMENT OF SPECIFIC CONGENITAL CARDIAC LESIONS

ANAESTHESIA FOR LEFT-TO-RIGHT AND RIGHT-TO-LEFT SHUNTS

Shunts, whether left-to-right or right-to-left, are managed under anaesthesia by manipulating the relative systemic (SVR) and pulmonary (PVR) vascular resistances.

Raising SVR increases left-to-right shunt, whereas lowering SVR or raising PVR increases right-to-left shunt. General anaesthetic agents usually lower SVR. The SVR can be raised using vasopressor agents such as phenylephrine or metaraminol. PVR is raised by hypoxia, acidosis, cold, hypercarbia, excessive ventilation pressures and drugs such as norepinephrine and dopamine. PVR is kept to a minimum by

normoxia, hypocarbia and alkalosis, and drugs such as milrinone and nitric oxide, which are used as a treatment for pulmonary hypertension.

In addition, great care must be taken to avoid air or particulate emboli which may enter the arterial circulation. Severe shunts require endocarditis prophylaxis as per local guidelines.

ATRIAL SEPTAL DEFECTS

Several different types of ASD exist, the most common requiring closure is the ostium secundum defect. They commonly coexist with other cardiac lesions. Isolated ASDs usually present incidentally, usually in older children and adults and are generally well tolerated. Pulmonary hypertension may develop in later life due to longstanding high pulmonary blood flow and vascular damage.

ANAESTHETIC CONSIDERATIONS

The main issues are discussed above. Some are suitable for device closure in the catheter laboratory. Surgery is usually straightforward and the patient may be suitable for 'fast-tracking'. Occasionally rhythm problems may occur due to atrioventricular node damage.

VENTRICULAR SEPTAL DEFECTS (VSD)

Several different types of VSD occur according to the location within the ventricular septum (membranous or muscular) and the area of the ventricular cavity (inlet, outlet, apical, subvalvar) in which they lie. VSDs commonly occur in complex cardiac lesions. Small muscular VSDs may close spontaneously and small perimembranous VSDs may be occluded by tricuspid valve tissue and be insignificant or cause mild progressive aortic regurgitation. Larger lesions cause significant left to right shunting, pulmonary overload and heart failure and present early.

Over time the shunt can reverse due to chronic injury to the pulmonary vascular bed, leading to pulmonary hypertension and eventually Eisenmenger syndrome.

ANAESTHETIC CONSIDERATIONS

The main issues are discussed above. The pulmonary vasculature may be reactive. Very small babies may be unsuitable for early VSD repair and a band is used on the pulmonary artery to minimise shunting. Surgical repair of a VSD can result in damage to the ventricular conducting tissue from suturing in the patch, especially with perimembranous lesions. Temporary or even permanent pacing may be required. Postoperative cardiac dysfunction may occur, especially if the ventricle has been opened. Device closure in the catheter laboratory is becoming more common.

PATENT DUCTUS ARTERIOSUS (PDA)

Failure of the arterial duct to close is common, especially in preterm babies. In older children it can be incidental and asymptomatic. Larger ducts can cause severe heart failure and pulmonary hypertension. Diastolic blood pressure is usually low in the presence of a significant duct. In preterm babies, left-to-right flow can cause low cardiac output, failure to wean from ventilation and necrotising enterocolitis. Medical management involves treatment of heart failure and the use of indomethacin to close the duct.

In older children the duct can be occluded using a device in the catheter lab.

In younger children, or where there is a large lesion, surgical closure is performed via a thoracotomy.

ANAESTHETIC CONSIDERATIONS

The main issues are discussed above. Catastrophic bleeding can occur if the duct or nearby vessels are damaged; therefore, cross-matched blood should be available. In neonates, the duct can be difficult to distinguish from the aorta or pulmonary artery and these can be mistakenly ligated. Monitoring of postductal blood pressure, saturation and end tidal capnography should confirm that the duct has been ligated. Diastolic blood pressure will rise after ligation of a large duct.

POST-REPAIR

After correction of an ASD, VSD or PDA, children are generally well. Endocarditis prophylaxis is not required in the absence of a residual lesion.

TETRALOGY OF FALLOT (TOF)

This relatively common condition has the features of a VSD, a misaligned aorta over-riding the VSD, right ventricular outflow tract obstruction and right ventricular hypertrophy. A spectrum of outflow tract obstruction severity occurs, from absent pulmonary valve to pulmonary atresia. The presence of beta-adrenergic receptors in the outflow tract causes intermittent worsening of right to left shunting during pain and stress, which manifests as cyanotic spells. Typically children squat during a cyanotic spell to increase the SVR and reduce right-to-left shunting. Preoperative beta-blockers may be prescribed. Some children are asymptomatic and never become noticeably cyanosed.

Surgical repair involves closure of the VSD, and relief of the outflow tract obstruction. Sometimes pulmonary valve replacement or a patch to enlarge the valve is required. Surgical repair usually takes place around 6 months of age but severe cases may be managed with a BT shunt as a neonate. Some centres perform full neonatal repair.

ANAESTHESIA CONSIDERATIONS

The degree of shunting can be controlled as above. Some patients are cyanotic and prolonged fasting and dehydration should be avoided. Cyanotic spells occurring under anaesthesia can be managed with:

- Vasopressor therapy, e.g. phenylephrine 4 mcg/kg
- Fluid bolus
- Opiate analgesia
- Beta blockade, e.g. propranolol 0.1 mg/kg

Cyanotic spells can occur even if there is no history, especially during line insertion and surgical manipulation. An audible pulse oximeter is useful as a warning.

Surgical complications include damage to the conducting system and junctional ectopic tachycardia (JET). Postoperative complications include myocardial dysfunction, JET, and restrictive right ventricular physiology leading to pleural and pericardial effusions, low cardiac output and renal failure.

POST-REPAIR

Long-term complications include pulmonary regurgitation and ventricular dilatation and further surgery is usually required in early adulthood. Children who present for non-cardiac surgery post-repair should be well from a cardiac point of view, but may require endocarditis prophylaxis if prosthetic material is present within the repair or a residual valve gradient or VSD exists.

PULMONARY AND RVOT SURGERY

These are invariably patients presenting for repeat surgery following repair of TOF. They will be undergoing multiple surgeries and mortality and morbidity are particularly greater if the patient is undergoing more than the third repeat operation. The amount of adhesion of the heart to the posterior aspect of the sternum requires careful assessment with CT and MR scans. Some of these patients may need femoral–femoral bypass prior to sternotomy. All patients must have external defibrillation pads attached to enable intervention until the heart is fully exposed so that internal paddles can then be utilized if necessary. Otherwise, basic principles of cardiac anaesthesia are applicable.

Many of these patients are young in chronological age as well as mental capacity with learning disabilities, which can make anaesthetic management difficult, including issues with consent. Appropriate support in the postoperative period, including the help of relatives to facilitate ICU care, is important. Some of these patients have additional problems with mobility and musculoskeletal abnormalities. These need careful preoperative assessment.

TRANSPOSITION OF THE GREAT ARTERIES (TGA)

In this condition the great arteries arise from the wrong ventricle. It usually presents as desaturation when the arterial duct closes early in life. Reopening of the duct with prostin is necessary for mixing of oxygenated and deoxygenated blood and balloon atrial septostomy is often performed to improve mixing and to allow withdrawal of prostin. Variants include TGA with intact ventricular septum and TGA with VSD. Coronary anatomy can be variable.

Surgical repair is undertaken early in life, while the PVR remains high in the neonatal period. Delayed repair results in a left ventricle that cannot cope with

a systemic afterload. Surgery can be delayed in the presence of a VSD but accelerated pulmonary hypertension is a risk.

Surgical repair involves transection and switching of the great arteries, and transplanting the coronary arteries to the new aorta which is formed from the pulmonary artery stump. It is a long surgery and bleeding from extensive aortic suture lines is expected. Post-bypass ischaemia can occur due to coronary problems, and the left ventricle can have difficulty coping with the systemic afterload so left atrial pressure is usually continually monitored post-bypass and on ICU. Fluid and blood products must be carefully administered as they can result in elevated left atrial pressure. Often the chest is left open for a few days postoperatively.

POST-REPAIR

Cardiac anatomy is usually restored to normal, although sometimes pulmonary artery stenosis can occur.

CONGENITALLY CORRECTED TGA (CCTGA)

In patients with CCTGA, the atria receive venous blood from their appropriate veins but their outflow is into the 'wrong' ventricular chamber. Therefore, the congenitally correct left ventricle, receiving deoxygenated systemic venous blood, pumps into the pulmonary artery; and the right ventricle, receiving oxygenated pulmonary venous blood, pumps into the aorta. These patients may present in adulthood, with systemic ventricular failure or pulmonary hypertension.

COARCTATION OF THE AORTA

A coarctation of the aorta may present in the neonate as a duct-dependent obstruction to the circulation, or in later childhood due to systemic hypertension or a murmur. Collaterals develop in older children. The narrowing often occurs at the site of the arterial duct tissue.

Repair may be urgent or scheduled and usually involves a thoracotomy. Occasionally a long section of the aorta is involved and repair takes place on cardiopulmonary bypass with circulatory arrest.

ANAESTHETIC CONSIDERATIONS

Maintenance of arterial duct patency with prostin may be required in the neonate. The site of blood pressure monitoring depends on the level of the coarctation, head and neck blood pressure is measured proximal to the obstruction and femoral pulses may be impalpable.

During surgical repair the aorta is cross-clamped and fluid loading is necessary prior to unclamping. Long cross-clamp times can result in spinal cord ischaemia. Hypertension can develop rapidly post-repair and may need nitrate or nitroprusside therapy.

POST-REPAIR

Recoarctation is common and can often be managed with stenting in the catheter laboratory. Occasionally open repair may be necessary.

SINGLE VENTRICLE (FONTAN) CIRCULATIONS

In some situations a single ventricle circulation is created surgically where the ventricle supports the systemic circulation and pulmonary blood flow comes directly from the systemic veins. The single ventricle can be a right or a left ventricle. Lesions requiring a Fontan strategy include hypoplastic left heart syndrome and tricuspid atresia.

A single ventricle is created in stages by creating a superior vena cava to pulmonary artery communication (Glenn) at around 6 months of age, followed by a total cavo-pulmonary connection during early childhood when the inferior vena cava is also connected (Fontan). For hypoplastic left heart syndrome, early neonatal surgery is also performed.

Early complications include high systemic venous pressure leading to head and neck swelling and pleural and pericardial effusions. Late complications include ventricular failure, arrhythmias, thrombosis and protein losing enteropathy.

ANAESTHETIC CONSIDERATIONS

Preoperatively patients are often cyanotic, so long fasting times and dehydration must be avoided or thrombosis can occur in existing shunts. Because

surgery is staged, precautions must be taken for repeat surgery (see earlier). Pulmonary blood flow is passive and reversed during intermittent positive pressure ventilation and PEEP; therefore, minimal ventilator settings and an early return of spontaneous ventilation are optimal.

POST-REPAIR

An increasing population of single ventricle patients are presenting for non-cardiac surgery. Some patients will be taking long-term anticoagulant therapy. Occasionally a communication is left between the IVC and right atrium (fenestration) in which case paradoxical air embolus, desaturation and endocarditis remain a risk.

Careful positioning is essential to maintain systemic venous return as flow into the pulmonary arteries is passive. Over-ventilation and atelectasis must be avoided or spontaneously ventilating strategies used. These patients are usually best managed with local or regional anaesthesia. They should be operated at a regional ACHD centre where appropriate anaesthetic, cardiology and ICU expertise is available.

CROSS-REFERENCES

Atrial septal defect, Chapter 2
Tetralogy of Fallot, Chapter 2
Infants and children, Chapter 24
Neonates, Chapter 24
Cardiopulmonary bypass, Chapter 16

MINIMALLY INVASIVE CARDIAC INTERVENTION

Many procedures are performed in the catheter lab but now purpose-built hybrid labs are being used for more complex interventional procedures.

DIAGNOSTIC PROCEDURES

ACHD patients may require diagnostic tests such as diagnostic catheters and ECHO. The catheterization is performed to predominantly evaluate pressure changes in cardiac chambers and great vessels and also to evaluate the circulation for shunting.

ACHD patients with shunts will need evaluation of their shunts. Changes in oxygen saturation from the IVC, SVC, RA, LA, RV and PA can be evaluated. This enables assessment of the degree of mixing between right and left sides; sudden step increase in saturation would be seen. Ideally these patients should have evaluation under local anaesthesia in order to avoid the cardiovascular changes associated with GA. If performed under GA, they should be ventilated with 21%–23% oxygen whilst the oximetry readings are being taken.

ASSESSMENT OF PULMONARY HYPERTENSION

These patients can be a high-risk population depending on the degree of pulmonary hypertension. They can require assessment of pulmonary artery pressures (PAP) as well as responsiveness to treatment. Treatment responsiveness can be assessed with evaluation of reduction in PAP with the breathing of 100% oxygen, or response to incremental increase to up to 20 ppm of nitric oxide. These can often be performed under local anaesthesia with some sedation but in some cases require general anaesthesia. A facility for enabling the administration of NO is crucial for these patients. The biggest danger with NO assessment is that of decompensation during the reduction of NO, with pulmonary hypertension recurring to such an extent that RV failure can occur.

INTERVENTIONAL CARDIOLOGY PROCEDURES IN ACHD PATIENTS

A common group of ACHD patients that present are those that have previously had surgery for Tetralogy of Fallot. Their major problem is pulmonary valve stenosis or incompetence, pulmonary outflow restriction with calcification or narrowing of the RVOT or pulmonary artery. These can be corrected with minimally invasive endovascular procedures (valve replacement or stenting of the pulmonary artery) but may need open-heart surgery.

The procedure is performed using the femoral vein for access. Postoperative care may be provided in the ICU although the intervention appears to have minimal side effects.

RV function may be compromised in these patients and improvement in pulmonary flow may increase pulmonary pressures and be associated with pulmonary oedema in the postoperative period.

ASD CLOSURE

Some patients will present incidentally with de-novo ASD in adulthood. TIAs or strokes can be presenting features but some will have signs of pulmonary hypertension.

Device closure is ideal for these patients, often easiest under GA. The patients will have radiology and TOE assessment during placement. The size of the defect and the need for adequate tissues to allow for device placement are important. Most patients can be sent home the next day and do not require ICU postprocedure. Occasionally the device can dislodge into the cardiac chambers and require emergency cardiac surgical intervention to retrieve the device.

INTERVENTIONAL PROCEDURES IN NON-ACHD PATIENTS

TRANSCATHETER AORTIC VALVE IMPLANTATION (TAVI)

This procedure is performed for patients with aortic valve dysfunction often severe stenosis. The patients presenting for TAVI are currently older patients and those with significant comorbidity that make conventional valve replacement a more risky procedure. These patients require full evaluation as for any patient undergoing valve surgery. In addition, however, the ability to safely place the new valve endoluminally mandates that correct sizing and position of the coronary arteries be more fully assessed. The patients should have good-sized femoral arteries to enable easier access through this route. Occasionally, the femoral artery requires dilating or stenting prior to insertion of the TAVI placement device. If it is not possible to safely use the femoral approach, the patient will be considered for placement via the transaortic route using a mini-sternotomy, or a transapical approach via a mini-thoracotomy. These patients are being operated upon with local anaesthesia. However, many units prefer the use of general anesthesia. Full bypass back-up is important in these patients. The best place for these procedures is the hybrid catheter lab. The use of TOE with radiography is important.

The new valve, usually made of titanium, is placed at the aortic valve after prior balloon dilatation. It crushes the old valve as it opens. During this placement procedure, it is important to reduce cardiac ejection. Therefore, the patient is usually paced at 130–150 beats/min in order to reduce the systemic pressure to about 40 mmHg. Minimizing the use of contrast helps to reduce the risk of postoperative renal failure. Many of these patients are fit for discharge the next day. However, renal failure, stroke, bleeding and arrhythmias are causes of delay in discharge. Patients that have had a sternotomy or thoracotomy will clearly stay in the hospital for a longer period compared to those undergoing the procedure via the femoral route.

REFERENCE

Mollmann H, Kim W-K Kempfert J, Walther T, Hamm C. (2013). Complications of transcatheter aortic valve implantation (TAVI): How to avoid and treat them. *Heart* **10**: 1136.

CROSS-REFERENCES

Atrial septal defects, Chapter 2
Pulmonary hypertension, Chapter 2
Adult congenital heart disease, Chapter 2

MITRAL VALVE SURGERY

MITRAL STENOSIS

PHYSIOLOGICAL CONSIDERATIONS

Mitral stenosis continues to present regularly to specialist units, despite the success of antibiotic therapy against acute rheumatic fever. The history of the condition is a gradual deterioration over many years. By the time surgical intervention is required, these patients are classically dyspnoeic and exhibit a relatively fixed, low cardiac output, often in association with atrial fibrillation. The area of the normal adult mitral orifice is 4–6 cm^2 and the severity of mitral

stenosis is graded by reduction in this area into mild $(1.6–2.0 \ cm^2)$, moderate $(1.0–1.5 \ cm^2)$ and severe $(<1.0 \ cm^2)$. The narrowed mitral valve restricts flow between the left atrium and ventricle and the degree of stenosis thus limits both early diastolic ventricular filling and the contribution of atrial contraction to late diastolic ventricular filling. Increased left atrial work inevitably causes dilatation of the thin-walled left atrium. In the normal heart, the two phases of diastole are clearly distinguishable using echocardiography, but in mitral stenosis the echocardiographic pattern shows a plateau and loss of late diastolic filling because the dilated left atrium generates less force than normal. In the presence of atrial fibrillation, the uncoordinated movements of the left atrium no longer contribute to this second diastolic phase, reducing end diastolic ventricular volume by up to 30%. Duration of diastole is therefore of great importance in mitral stenosis, and excessive tachycardia compromises cardiac output, especially in the presence of atrial fibrillation. Many of these patients will be on long-standing digitalis therapy and this should be continued into the perioperative period, along with appropriate potassium supplementation. The primary purpose of this therapy is to control ventricular rate, rather than to enhance myocardial contractility. Mean left atrial pressure (LAP) is usually greater than 15 mmHg, and factors causing increased filling of the left atrium are liable to precipitate pulmonary oedema. The natural history of the condition is progression to pulmonary hypertension and, eventually, to cor pulmonale as a response to left- and then right-sided heart failure.

GENERAL ANAESTHETIC PRINCIPLES

The main areas of anaesthetic consideration for this group are heart rate, rhythm and preload. Anaesthetic technique is similar to that described for coronary artery bypass grafting (CABG) and must include antibiotic prophylaxis.

Bradycardia

- Causes a decrease in cardiac output due to a fixed stroke volume.

Tachycardia

- Decreases ventricular filling time and thus cardiac output.

- Increases LAP and may result in pulmonary oedema.

Atrial fibrillation

- Will further decrease ventricular filling by up to 30%.
- Is often long standing and, if so, will be highly resistant to cardioversion.
- May be induced by anaesthesia.
- Digitalis toxicity may be induced by intraoperative hypokalaemia.

Preload

- Hypovolaemia may result in reduced LAP, and thus a further reduction in left ventricular filling.
- Excessive fluid load may result in pulmonary oedema.

Afterload

- A large decrease in systemic vascular resistance (SVR) may cause severe hypotension due to a relatively fixed cardiac output.

MONITORING CONSIDERATIONS

The pressure gradient across the mitral valve means that pulmonary artery wedge pressure (PAWP) overestimates the left ventricular end-diastolic pressure (LVEDP). However, in the absence of significant changes in heart rate, PAWP trend measurements reliably track left-sided filling pressure. A reflex tachycardia following excessive blood loss will make this technique inaccurate, and in some cases it may be more appropriate to place a left atrial pressure catheter at operation for postoperative monitoring. TOE is helpful and probably considered mandatory in these patients. It helps with the assessment of atrial size, ventricular filling and assessment of the left atrial appendage for the presence of clot.

MITRAL INCOMPETENCE

PHYSIOLOGICAL CONSIDERATIONS

Mitral incompetence results in dilatation of both the left atrium and the left ventricle. During systole the regurgitant flow causes high pressure to

be transmitted to the left atrium and increases left ventricular work. In contrast to mitral stenosis, there is no obstruction to forward flow through the valve, with the exception of a combined stenotic and regurgitant lesion. Any increase in systemic vascular resistance will limit left ventricular forward ejection and thus encourage retrograde flow into the more compliant atrium. An increased afterload causes decreased forward flow and increased regurgitant flow, and in this respect these patients are sensitive to peripheral vasoconstrictors. The magnitude of regurgitant flow is determined by the size of the regurgitant orifice and the pressure gradient across it; the orifice size tends to parallel ventricular size. Increased preload causes left atrial dilatation and further stretching of the mitral valve orifice which may result in a decrease in ventricular forward flow due to an increased regurgitant flow into the atrium. In common with mitral stenosis, these patients may progress to pulmonary hypertension and cor pulmonale. It is important to realize that acute mitral incompetence may be due to posterior left ventricular papillary muscle damage induced by myocardial infarction. In this case, the left atrium may be small, making replacement of the valve technically difficult. This group is especially sensitive to increases in SVR. Patients with mitral regurgitation caused by coronary artery disease, including those with recent myocardial infarction, require extremely careful anaesthesia.

GENERAL ANAESTHETIC PRINCIPLES

Anaesthetic technique is similar to that described for CABG and must include antibiotic prophylaxis. The anaesthetic aims for these patients are centred on the maintenance of forward flow through the left ventricle.

Systemic vascular resistance

- An increased SVR increases the tendency for regurgitative flow, and vasoconstrictors should be avoided.

Preload

- A large increase in preload causes atrial distension and the relatively rapid onset of pulmonary oedema.

Heart rate

- Bradycardia reduces ventricular filling and increases the degree of regurgitation; however, a moderate tachycardia increases forward flow, and is thus preferable.

MONITORING CONSIDERATIONS

It has been suggested that faster, fuller and vasodilated are the principles on which forward flow in mitral regurgitation may be maintained. In these circumstances (especially when using potent vasodilators and with the dangers of vascular overfilling) a pulmonary artery catheter allows assessment of intravascular filling, measurement of cardiac output and evaluation of therapeutic intervention. In patients with high pulmonary artery pressures, evidence of tricuspid regurgitation should be looked for in the central venous pressure trace. Passive tricuspid regurgitation results from right ventricle dilatation in the face of increased afterload from pulmonary hypertension (PHT). There are few attractive therapies for this combination of PHT and right ventricular failure and attention should be paid to basic principles, including the avoidance of hypoxia, hypercarbia and acidosis. One promising therapeutic strategy for the treatment of PHT is the use of prostaglandin E_1 (prostacyclin), a potent dilator of pulmonary arterial smooth muscle. It has been noted that prostacyclin is also a systemic vasodilator. However, it has the theoretical advantage of having pulmonary endothelial first-pass metabolism, and may be considered a 'pulmonary-specific' vasodilator when given via the right atrium. Enoximone and milrinone may be used as they reduce systemic and pulmonary vascular resistance as well as improving inotropy.

TOE is deemed mandatory for mitral valve repair. It gives the surgeon assurance that the repair has worked. It ensures that there is no regurgitation but also that there is no evidence of severe stenosis due to overzealous repair. In addition, it is an easier way of assessing tricuspid valve function.

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CROSS-REFERENCES

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Cardiopulmonary bypass: principles, physiology and biochemistry, Chapter 16
TOE, Chapter 16

POSTOPERATIVE CARE OF ADULT PATIENTS AFTER CARDIOPULMONARY BYPASS

The increase in demand for cardiac surgery, caused mainly by coronary artery disease, has led to a marked rise in the number of patients presenting to postoperative ICUs. This has driven the development of specialized post-cardiac surgery care units (CSUs) with protocol-based care. The protocols used and the duration of postoperative ventilation, etc. vary considerably from one unit to another. However, the basic principles of postoperative care are common to all patients and are summarized below. For convenience, the postoperative period has been divided into transfer, early and late phases.

TRANSFER PERIOD

Following the completion of surgery, patients are usually transferred to their critical care bed within theatre and transported on it to the CSU. The distances involved vary enormously from one hospital to another but in most cases they are close to the operating rooms. This is a period of great potential instability and, unless the transfer distance is very short indeed, at the very least ECG and intra-arterial pressure monitoring should be continued. The development of modern transport monitors has made this a much easier task. We would recommend full monitoring in all patients.

THE EARLY POSTOPERATIVE PERIOD (0–6 H)

The essentials of the early postoperative care of these patients are summarized in Table 16.1.

Table 16.1 Care in the early postoperative period

- Analgesia and sedation
- Assisted ventilation ± PEEP
- Measurement of arterial blood gases, potassium, haemoglobin, etc.
- Adjustment of inspired oxygen concentration
- Maintenance of cardiac rhythm; treatment of arrhythmias
- Control of postoperative hypertension
- Haemodynamic monitoring
- Manipulation of cardiac filling pressures
- Maintenance of adequate cardiac output
- Maintenance of adequate renal perfusion and urine output
- Fluid management; colloid replacement, crystalloids
- Monitoring and treatment of clotting/platelet abnormalities
- Reduction of heat loss; assisted rewarming

ANALGESIA AND SEDATION

Adequate opioid analgesia is essential, but there is wide variability in the drugs and routes of administration used; fentanyl, alfentanil, pethidine and morphine are all popular. The use of high dose fentanyl (40–60 mg kg^{-1}) intraoperatively provides analgesia well into the postoperative period; alfentanil is a potent short-acting agent and is given by continuous infusion. Remifentanil appears to be associated with an acute withdrawal syndrome sometimes requiring higher initial doses of opioid at the cessation of infusion. Morphine is widely available, cheap and has useful additional sedative properties, but care should be taken in patients with poor renal function due to the accumulation of active metabolites. Epidurals are used at some centres. NSAIDs are not widely used; they all inhibit the renal protective actions of prostaglandins during hypotension and should be used with great caution in the early postoperative period. In some patients, despite adequate analgesia, additional sedation may be required; propofol, dexmedetomidine, midazolam and isoflurane (0.4%–0.6%) are popular and combine a short duration of action with acceptable haemodynamic stability. Shivering is an important complication in the early postoperative period as it markedly increases oxygen consumption

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and must be suppressed by adequate analgesia and sedation (e.g. pethidine 10–20 mg is interestingly quite effective).

ASSISTED VENTILATION

The duration of mechanical ventilation after cardiac surgery is very variable even in routine patients, varying from none (extubation on the operating table) to several hours. Improvements in surgical and anaesthetic techniques with a reduction in cardio-pulmonary bypass times, less hypothermia, epidur-als, etc. have led to a trend towards earlier extubation in many centres. Safety is an important consideration and it seems prudent to continue mechanical ventila-tion until adequate rewarming (at the least to central normothermia), adequate analgesia, haemostasis and haemodynamic stability have been achieved.

A degree of atelectasis is common after cardiac surgery, especially if the pleura has been breached during internal mammary dissection. Low levels of PEEP (2.5–5 cmH$_2$O) are widely used after cardiac surgery; they improve oxygenation in the presence of pulmonary oedema and may play a role in revers-ing atelectasis. The adverse haemodynamic effects of PEEP are exacerbated by hypovolaemia.

Most modern intensive care ventilators incorpo-rate the facilities for weaning in the form of synchro-nized intermittent mandatory ventilation (SIMV) with inspiratory assist. Mandatory breaths are grad-ually reduced to zero and, if adequate ventilation is maintained, then the patient is extubated. Many factors may precipitate more prolonged periods of mechanical ventilation. These include: previous lung pathology, haemodynamic instability, persistent pulmonary oedema, delayed neurological recovery and pulmonary infection.

MONITORING

Table 16.2 lists the basic monitoring required in the postoperative period.

HAEMODYNAMIC MANIPULATION

Skilled haemodynamic monitoring and manipula-tion are essential after cardiopulmonary bypass. In UK practice, most routine patients do not have

Table 16.2 Basic monitoring after cardiopulmonary bypass

- ECG (rate, rhythm, ST changes)
- Intra-arterial blood pressure
- Central venous pressure
- FiO$_2$, respiratory rate, airway pressures, tidal and minute volumes
- Arterial blood gases
- Pulse oximetry
- Serum potassium
- Blood loss
- Haemoglobin, clotting screen, platelet count
- Blood sugar
- Core and peripheral temperatures
- Chest X-ray
- Urine output

pulmonary artery catheters inserted and the ade-quacy of cardiac output has to be estimated from a combination of arterial blood pressure, urine output, peripheral rewarming and the absence of a metabolic acidosis. Early hypotension is most commonly due to hypovolaemia exacerbated by the vasodilatation that occurs during the rewarming phase. Central venous pressures should be maintained by the infusion of colloids, including blood, if indicated. The pressure required is variable, but in the absence of significant ventricular dysfunction, pulmonary hypertension, etc., a level of 5–8 mmHg is a reasonable starting point. Hypertension after cardiopulmonary bypass is also common and should be treated to avoid excessive left ventricular workloads, suture-line disruption and bleeding. Having ensured adequate analgesia and sedation, nitrodilators (glyceryl trinitrate, sodium nitroprusside) form the mainstay of treatment.

Arrhythmias are common after cardiac surgery and require aggressive treatment because of their effects on cardiac output and blood pressure. When they occur, always quickly ensure that the patient has not become acutely hypoxic from a simple cause such as ventilator disconnection. Hypokalaemia is a com-mon contributing factor and serum potassium should be maintained at 4.5–5.0 mmol L^{-1}. Magnesium levels should also be maintained at normal to high normal levels. Following cardiopulmonary bypass, ventricu-lar function is often impaired and many patients have relatively fixed stroke volumes. Persistent brady-cardias should be avoided by making use of pacing,

isoprenaline or dobutamine infusions, for example, to maintain the heart rate; rates of 60–100 beats min^{-1} are usually considered optimal.

The absolute indications for the instigation of more complex haemodynamic monitoring in the form of pulmonary artery wedge pressure measurements and thermodilution cardiac output measurements are difficult to define. Failure to rewarm, oliguria, worsening metabolic acidosis, low venous oxygen saturations and persistent hypotension despite apparently adequate filling pressures are all good indications. Pericardial tamponade should be high on the list of suspected causes if these conditions develop in the postoperative period. Transoesophageal echo (TOE) should be considered at an early stage. Mixed venous oxygen saturation is also a good indicator of perfusion.

Inotropic agents such as enoximone, milrinone, dobutamine and epinephrine are indicated if the cardiac index is low despite adequate filling. A low systemic vascular resistance occurring after prolonged bypass may require the use of norepinephrine. Hydrocortisone (100 mg) followed by 50 mg 6 hourly may help to counteract the SIRS response to surgery and enable earlier weaning of norepinephrine infusion.

RENAL FUNCTION

Although acute renal failure requiring dialysis is rare after uncomplicated cardiac surgery, degrees of oliguria are common. Although there is little scientific evidence, dopamine at 'renal' doses (e.g. 3 mg kg^{-1} h^{-1}) is still used both intra- and postoperatively. Dopexamine has not been found to be useful. It is important to maintain a diuresis in the postoperative period so as to reverse the anaemia from haemodilution caused by the bypass prime and the cardioplegia. Good volume status, good cardiac output, good oxygen delivery, avoidance of acidosis and high mean blood pressure are the mainstay for management. If oliguria persists despite an adequate cardiac output, vascular volume and blood pressure, small doses of a loop diuretic are indicated. It is not unusual to see a doubling of serum creatinine in patients that have a history of renal dysfunction. Short-term renal replacement therapy with haemofiltration may be necessary in some patients, particularly if there is severe acidosis or hyperkalaemia.

FLUID MANAGEMENT

Cardiac filling pressures should be maintained by the infusion of colloid solutions, including the modified gelatins, albumin solutions and blood. Free water is required for the formation of urine, and in the absence of pulmonary oedema our routine practice is to give all patients 1 mL kg^{-1} h^{-1} of crystalloid (dextrose saline, saline or Hartmann's solution). Although circulating hypovolaemia is common, tissue dehydration is rare in the immediate postoperative period, the usual problem being an increase in total body water.

THE LATE POSTOPERATIVE PERIOD (AFTER 6 H)

In a routine case, following extubation, the activities described above are continued, but the emphasis shifts towards preparing the patient for return to the HDU or ward. Most patients require oxygen to be administered by face-mask to maintain arterial saturations above 95%. Oral, intramuscular or rectally administered analgesics may be prescribed and the patient undergoes regular chest physiotherapy. Where postoperative complications develop, or if the patient's progress is slow, the intensive care administered during the first few hours is continued. In these circumstances every effort must be made to look for correctable causes of the failure to progress. Observing the deterioration closely but without intervention achieves nothing.

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Postoperative pain management, Chapter 30

POSTOPERATIVE CARE OF PAEDIATRIC PATIENTS AFTER CARDIOPULMONARY BYPASS

After cardiac surgery, most children are kept sedated and ventilated in intensive care for a few hours to several days, the goal being to minimise cardiac work and oxygen requirements. In some circumstances, it is possible to expedite extubation and intensive care, (fast-tracking) especially after uncomplicated surgery. Good analgesia is required. Sedation is usually achieved with a combination of opioid analgesia and midazolam infusions. Sometimes muscle relaxants are needed, especially if the chest remains open or if the patient is unstable. Organ dysfunction after cardiac surgery is not confined to the cardiovascular system, especially after surgery for complex lesions or where there has been a complication.

CARDIOVASCULAR SYSTEM

Even in stable patients careful attention to myocardial function is required and myocardial work should be kept to a minimum. Systemic blood pressure needs to be controlled to allow normal organ perfusion without hypertension which can disrupt suture lines.

Low cardiac output syndrome is common after cardiac surgery and inotropes may be required, commonly a combination of milrinone and epinephrine is used. Milrinone is a phosphodiesterase inhibitor with venous, arterial and pulmonary artery dilating properties. As well as its inotropic action, it is also a lusitrope, promoting active diastolic relaxation without increasing myocardial oxygen demand. It is useful in fast heart rates, such as those seen in younger children and is usually commenced in the operating theatre as a loading dose of 50 mcg/kg followed by a maintenance infusion at 0.3–0.75 mcg/kg/min.

Modified ultrafiltration (MUF) is a technique used after separation from cardiopulmonary bypass to remove water and inflammatory mediators in smaller patients. This reduces myocardial oedema and improves cardiac function.

Rhythm disturbances can be seen after cardiac surgery. Heart block can occur after trauma to the conducting tissue and may need pacing. Tachydysrhythmias also occur and can be managed with magnesium sulphate and anti-arrhythmic therapy. Bleeding after cardiac surgery can lead to hypovolaemia or to cardiac tamponade if concealed. Pulmonary hypertension is associated with some cardiac lesions and may require inhaled nitric oxide.

RESPIRATORY SYSTEM

A chest X-ray is performed soon after cardiac surgery to check the position of the endotracheal tube and chest drains, and to examine the lung fields. Pulmonary oedema and haemorrhage can occur in the early postoperative period and may need special ventilatory parameters. Bleeding and inflammation can lead to pleural effusions and a chylothorax can occur if there has been damage to lymphatic tissue in the chest. Damage to the phrenic nerve or recurrent laryngeal nerve can cause respiratory compromise.

CENTRAL NERVOUS SYSTEM

Rarely brain injury can result from cardiac surgery, due to low cardiac output states, emboli or bleeding. Deep hypothermic cardiac arrest states are a particular risk. The use of near infrared spectroscopy (NIRS) aims to warn of impending neurological injury and it is often continued into the postoperative period. After aortic cross-clamping for coarctation repair, there is a risk of spinal cord ischaemia.

RENAL

Urine output needs to be monitored throughout the case and in the postoperative period. Oliguria is common, particularly after MUF. If urine output falls below 0.5 mL/kg/h, diuretic therapy can be used. Low cardiac output may lead to renal dysfunction, sometimes requiring renal replacement therapy, usually peritoneal dialysis or haemofiltration. Maintenance intravenous fluid is introduced slowly to avoid fluid overload. Electrolyte levels need monitoring.

GASTROINTESTINAL SYSTEM AND NUTRITION

Gastrointestinal smooth muscle tone is reduced in the early postoperative period due to anaesthesia,

opiate therapy and cardiac bypass. A nasogastric tube is used to minimise gastric distension. Enteral nutrition is commenced as soon as reasonable. In neonates with low cardiac output syndrome, mesenteric ischaemia or necrotising enterocolitis can occur.

HAEMATOLOGY

Chest drain output needs to be recorded and severe bleeding may require re-exploration of the chest either in the intensive care unit or in theatre. Clotting factors and platelets are consumed on cardiopulmonary bypass and may require supplementation on ICU. Residual heparin may require protamine administration. A thromboelastograph (TEG) can guide coagulation management.

INFECTION

Routine antibiotics are continued in the postoperative period. Invasive lines and chest drain sites are monitored for infection and removed as soon as practical.

EXTRACORPOREAL LIFE SUPPORT (ECMO)

Occasionally very sick patients require ECMO postoperatively while cardiac function recovers. This is undertaken at a few specialist centres.

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Infants and children, Chapter 24
Neonates, Chapter 24
Fluid and electrolyte balance, Chapter 30
Postoperative pain management, Chapter 30

REGIONAL ANAESTHESIA AND CARDIAC SURGERY

The majority of clinical experience with spinal opioids in cardiac anaesthesia has been with intrathecal morphine although epidural opioids have also been used. Typically, 0.03 mg kg^{-1} morphine diluted with 10 mL normal saline is administered to the lumbar CSF using a 25G bevelled or a 24G Sprotte needle. Clonidine (50–100 μg mL^{-1}) may be added to the solution.

The advantages claimed for intrathecal morphine in cardiac surgery are

- Excellent analgesia, which persists into the postoperative period.
- Reduced vasodilator use in the ICU.
- Less respiratory depression when compared to intravenous opioid use.
- Reduced hormonal stress response to surgery.
- Reduction in cardiac ischaemia and arrhythmias.

Epidural analgesia may be a better option when compared to single shot spinal anaesthesia because it may be continued for a number of days postoperatively. It is usually inserted at T2/3 level to provide blockade of the cardiac sympathetic fibres. The timing of insertion is debatable. Some institutes insert them the day before in order to reduce the concern of a bloody tap. Logistically this is problematic because of the need for appropriate sterility and staffing during insertion. It is more logical to insert these on the day of the operation in the anaesthetic room in an adequately premedicated patient with full invasive monitoring. It also allows it to be used in the unstable patient in whom more time may be given to stop any preoperative anticoagulants. As heparin is not a fibrinolytic agent, there should not be a problem with its administration an hour after the epidural has been inserted. A 16G Tuohy needle and catheter are inserted at the T2/3 level with the patient in the sitting or left lateral position. We have used a combination of local anaesthetic (bupivacaine or ropivacaine) and opioid (diamorphine or fentanyl) to initiate and maintain the anaesthesia. With such high epidurals it is mandatory to monitor ascending block towards the phrenic nerve (C3, 4, 5). This may be performed with the epidural scoring scale for arm movement (ESSAM Score). This utilises handgrip, wrist flexion and elbow flexion (C5-T1) to assess ascending motor blockade.

It should be noted that, although intrathecal morphine results in a reduction in the patient's demand for analgesia, there have been few controlled studies comparing this technique with other methods of analgesia and no studies have conclusively proven

that intrathecal morphine provides better analgesia. Furthermore, although intrathecal morphine is associated with a modest improvement in respiratory parameters such as peak expiratory flow rate and post-operative arterial carbon dioxide tension, it has not been shown to reduce time to extubation or ICU stay.

Many anaesthetists will not utilise regional anaesthesia because it is deemed to add a new potential for complications, ranging from the undesirable to the life-threatening. Spinal and epidural anaesthesia are procedures that require a significant amount of skill, and are associated with a small failure rate, particularly in the elderly, as well as the rare complications of infection and neurological sequelae. Post-spinal headache does not appear to be a problem in the cardiac surgical patient; urinary retention is not an issue owing to the necessity for catheterization. Backache attributable to dural puncture has not been reported. In common with IV opioids, up to 20% of patients will suffer from nausea and/or vomiting, and a much smaller number experience pruritus which is often confined to the facial dermatomes but which may be severe.

The two most important problems associated with the use of spinal opioids in cardiac surgery are respiratory depression and the potential for spinal haematoma formation. Respiratory depression is characteristically delayed following the use of hydrophilic drugs such as morphine. It is believed to be due to slow rostral spread of the drug by bulk flow in the cerebrospinal fluid, which acts on the respiratory centre in the floor of the fourth ventricle many hours after administration. Several papers have attested to the fact that the phenomenon does not occur after the first 24 h, during which time close respiratory monitoring is clearly required. This prerequisite is easily provided in the post-cardiac surgery patient, as it is usual practice to nurse these patients either in an ICU, HDU or step-down unit during this time. The respiratory depression associated with intrathecal morphine may be precipitated by the concomitant use of opioids by other routes (including premedication drugs), which should be given with caution, if at all. It is easily reversed with a carefully titrated dose of intravenous naloxone, which is insufficient to antagonize analgesia.

The controversy surrounding the use of spinal and epidural blocks in patients with abnormalities of coagulation reaches its zenith in cardiac surgery, as the patient is required to be fully anticoagulated. Spontaneous epidural haematomata in the presence of a coagulopathy, and haematomata following axial blocks, although rare, have been reported in the literature, and can lead to irreversible neurological damage. However, several large series from the 1980s have demonstrated the safety of axial blockade in patients who have subsequently been heparinized for vascular surgery. Indeed, there have been no reports of epidural haematoma following intrathecal morphine for cardiac surgery. Our own current practice is to insert epidurals on the day of the surgery. We have had no problems in our patients over the past 17 years in over 3000 patients (unpublished data).

Notwithstanding the above argument, it is generally accepted that the pre-existence of an iatrogenic or other coagulopathy is an absolute contraindication to regional anaesthesia, although, if there were very strong indications, some anaesthetists might proceed in a patient receiving low-dose heparin or antiplatelet drugs. The other contraindications to spinal puncture – local infection, spinal deformity, neurological disease, raised intracranial pressure and patient refusal – also apply.

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SEQUELAE OF CARDIOPULMONARY BYPASS

During cardiopulmonary bypass (CPB), normal physiology and biochemistry are significantly altered by changes in blood pressure and flow, temperature and haemodilution. The blood is in contact with abnormal surfaces in the oxygenator, heat exchanger, reservoir, tubing, cannulae and filters. These factors can lead to systemic and cerebral complications. Fortunately, the incidence of serious morbidity is low (0.5%–1%) and CPB is regarded as a safe procedure in the majority of patients. There is, however, a much higher incidence of more minor and subtle effects which are usually temporary and which the patient may not notice.

Blood flow, pressure and temperature are abnormal during CPB. At the onset of CPB using a crystalloid prime there can be a sharp drop in the blood pressure. This is caused by the lower systemic flow and the sudden fall in blood viscosity as crystalloid is pumped into the circulation. Subsequently during bypass the systemic vascular resistance gradually increases towards the normal range. A further reduction may be seen when the cross-clamp is removed, particularly in patients receiving blood cardioplegia.

Although the endocrine response cannot be separated from that due to anaesthesia and surgery, during CPB there is a generalized increase in serum catecholamine levels in excess of those seen in operations not utilizing CPB. There is no pulmonary metabolism of norepinephrine; renin secretion is increased, and with this follows angiotensin activation and aldosterone secretion. Vasopressin levels increase considerably during CPB and remain elevated for up to 48 h following surgery. These increased levels of catecholamines, angiotensin and vasopressin, together with local tissue vasoconstrictor agents, lead to arteriolar constriction. A mild hyperglycaemia may be seen following CPB, due to increased gluconeogenesis, peripheral insulin resistance, a decrease in serum insulin and raised ACTH and cortisol levels.

Total body water is increased at the end of CPB, the extra water being contained in the extracellular and extravascular spaces. Haemodilution and the increased capillary permeability resulting from activation of inflammatory mediators are the major factors causing this fluid shift.

DAMAGING EFFECTS OF THE CPB CIRCUIT

The exposure of blood to abnormal surfaces during CPB causes platelet activation and aggregation, the net effect being a reduction in platelet numbers and impairment in function of those that remain. Platelet damage is probably the most important factor in the bleeding diathesis associated with CPB. Proteins are denatured by contact with foreign surfaces, and this can lead to activation of various clotting and fibrinolytic cascades with consumption of clotting factors, microcoagulation, fibrin generation and complement activation. The complement cascade results in the production of powerful anaphylotoxins which increase capillary leakage, mediate leucocyte chemotaxis and facilitate leucocyte aggregation and enzyme release. There is mechanical damage to leucocytes and erythrocytes from the shear stresses caused by turbulence from the pumps, suckers, abrupt changes in velocity of blood flow and cavitation around the cannula tip. Damage to blood produces fibrin micro-emboli, aggregates of denatured protein and lipoproteins and platelet and leucocyte aggregates. Particulate emboli in spilt blood are aspirated by suckers and returned to the bypass circuit. There can be significant air emboli during aortic cannulation, during filling of the beating heart after removal of the aortic cross-clamp and during discontinuation of CPB despite meticulous de-airing techniques. In about 1 in 1000 procedures, a critical incident will occur from malfunction of the extracorporeal circuit.

SPECIFIC ORGAN DAMAGE ASSOCIATED WITH CPB

HEART

CPB has only a minor effect on cardiac dysfunction unless there has been inadequate myocardial

protection or perfusion. Post-bypass cardiac function is more closely related to the preoperative condition of the heart and the success of surgery.

LUNGS

Abnormalities of lung function following CPB are frequent, with clinical manifestations of atelectasis and pulmonary oedema. Acute lung injury leading to adult respiratory distress syndrome (ARDS) occurs in less than 1% of patients. A reduction in FRC with an increased A–a difference may persist for up to 10 days. Sputum retention and ineffective coughing, which contribute to pulmonary morbidity, are consequences of the surgery and postoperative care rather than the CPB.

KIDNEYS

Renal dysfunction occurs to some degree in 1%–4% of patients following CPB. It is usually due to acute tubular necrosis and, although potentially reversible, is associated with a high mortality. Factors associated with an increased risk of renal failure are pre-existing renal impairment, long bypass times and low cardiac output. Drugs such as aminoglycoside antibiotics may be contributory.

GASTROINTESTINAL TRACT

Gastrointestinal tract complications develop in less than 2% of patients following CPB, but the associated mortality is high. The most common problem is upper gastrointestinal bleeding and is maximal on the tenth postoperative day. Most units use prophylactic H2 antagonists to reduce this risk. Hyperbilirubinaemia has been reported in up to 20% of patients. Rare complications are ischaemic bowel and ischaemic pancreatitis.

NEUROLOGICAL

These can be divided into global, focal or neuropsychological complications. Global damage often presents as a prolonged depression of conscious level unrelated to sedation and is seen in up to 3% of patients. In serious cases there are frequently signs of widespread neurological dysfunction present. Patients in coma for over 24 h have a high mortality, and poor prognostic signs include extensor posturing, the absence of motor responses and seizures. Choreoathetosis is a rare but serious complication occurring almost exclusively in paediatric patients who have had total circulatory arrest. Sensorineural hearing loss is often missed clinically, but up to 13% of patients have been reported to have a hearing loss of greater than 10 dB following CPB.

Focal events or strokes (defined as a focal CNS deficit of relatively sudden origin that lasts for more than 24 h) are the major cause of persisting neurological disability following cardiac surgery. They are usually seen as an acute hemiparesis or visual field defect and have been reported as occurring in 1%–6% of patients after coronary artery bypass grafting. Approximately 70% of cardiac-related strokes occur intraoperatively and 30% in the early postoperative period. Acute focal deficits due to air emboli usually resolve steadily over the first 24 h. Membrane oxygenators have been shown to produce fewer micro-emboli than bubble oxygenators (rarely used now), and there are fewer microvascular occlusions seen in the retinal microcirculation when using a membrane oxygenator as compared with a bubble oxygenator.

The most important risk factors for cerebral damage during CPB are increasing age, a previous cerebrovascular event, pre-existing carotid or cerebrovascular disease, aortic atherosclerosis, valve surgery, left ventricular thrombus, poor preoperative cardiac function, the occurrence of microemboli and long bypass times. Current evidence suggests that the best way to reduce the sequelae of CPB is to perform the surgery meticulously and expeditiously, with minimum suction of shed blood, using a membrane oxygenator and a 40 mm main arterial filter. Aortic assessment with transaortic sonography can identify areas of calcification in the ascending aorta. Avoidance of cannulation of these areas may be associated with a reduction in neurological sequelae. Judicious de-airing of the open heart prior to restoring flow is important. Insufflation of CO_2 (2–4 L min^{-1}) into the mediastinum may help to displace air, and thus nitrogen.

Postoperative confusional state is seen in up to 30% of patients. This is particularly seen in patients that are elderly, have high alcohol intake, are heavy smokers or have pre-existing psychiatric problems. It can present as a manic state with aggressive behaviour, or a hypomanic state where they appear

very withdrawn. These patients may be treated with haloperidol boluses given regularly 6 to 8 hourly, or as needed boluses.

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THORACIC AORTA SURGERY

AORTIC DISSECTION

Aortic dissections are characterized by an intimal tear followed by a longitudinal separation within the media of the wall which extends parallel with the lumen. It usually presents acutely with severe anterior or posterior chest pain. Depending upon position and progression, dissections can cause aortic valve incompetence, and interruption of the coronary, cerebral, spinal, subclavian, mesenteric, renal or femoral arteries. Clinical presentation may be related to these secondary effects. Young patients could have an associated connective tissue disorder such as Marfan syndrome. The major classification is into types A (involving the ascending aorta) and B (distal to the origin of the left subclavian).

Type A occurs in 65%–70% of cases and presents at a slightly younger age (50–55 years) than type B (60–70 years). Both types are 2–3 times more common in men and associated with hypertension more than 50% of the time. In type A an intimal tear is always present whereas a tear is seen in 90%–95% of type B. Aortic regurgitation is more common in type A (50%) versus 10% in type B although the latter is more often associated with hypertension on admission and atherosclerosis. The mortality is >90% in type A but only about 40% in type B.

ANEURYSMAL DILATATIONS

These are usually asymptomatic until they leak or produce symptoms due to compression on surrounding structures such as the superior vena cava, left main bronchus or lung. Intimal deterioration can occlude smaller arteries; paraplegia, for example, may be the presenting symptom. There is often a history of hypertension and diabetes together with aneurysmal dilatation of the abdominal vessels.

Although clinical presentation and plain chest X-rays may suggest a diagnosis, accurate diagnosis depends upon special investigations such as aortography, CT scan, MRI scan and echocardiography.

ANAESTHETIC CONSIDERATIONS

PREOPERATIVE

Perform a full neurological assessment and record any deficits. Reduce hypertension with the use of vasodilators (e.g. sodium nitro-prusside, nitrates and beta-blockers). Catheterize and check renal function.

Insert two large-bore cannulae and an arterial line in the arm least affected by the lesion. Order a minimum of 4–8 units of blood and clotting factors. Ensure early institution of invasive monitoring. The choice of artery may in part depend on surgical factors. Occasionally these patients may require bypass. Femoral artery cannulation may be indicated. Mortality increases by 1% every hour in patients presenting with acute type A dissection. Continued or suddenly increasing pain may indicate further dissection and the need for immediate surgery.

PERIOPERATIVE

Antibiotic prophylaxis is essential. Provide renal protection – furosemide and mannitol are used on some units. The anaesthetic technique is as for coronary artery bypass grafting.

Type A with involvement of ascending aorta only

- Full cardiopulmonary bypass (CPB) is necessary with cardioplegia for myocardial protection. Replacement of the aortic valve and reimplantation of the coronary arteries may be required in addition to grafting of the ascending aorta.

Type A with involvement of aortic arch

- As above, but in addition operation on the cerebral vessels requires total circulatory arrest at <18°C.

Type B with involvement of the descending aorta

- Does not require CPB and is approached via a left thoracotomy. A double-lumen tube is preferred to allow deflation of the left lung.

All of these operations necessitate cross-clamping of the aorta. When the clamp goes on there may be proximal hypertension, requiring the use of vasodilators. Unclamping produces a sudden fall in left ventricular afterload and systemic BP. Fluid loading and/or vasoconstrictor agents will be required.

Some of the type B dissections may be corrected with the use of endoluminal stents. It involves an approach through the femoral or iliac arteries with radiology for ensuring correct placement. It can be performed under local anaesthesia and sedation. Arterial monitoring is helpful. Some problems of open surgery do not apply, leading to a reduction in perioperative morbidity

POSTOPERATIVE

Ensure stable haemodynamics. Monitor and preserve renal function wherever possible. The incidence of renal failure is 5% and is related to preoperative renal function and the cross-clamp ischaemic time. Central and peripheral neurological function need careful observation, although there is little that can be done to affect the course of intraoperative damage from ischaemia or embolization. The incidence of paraplegia is 5%–10%. Postoperative hypotension should be avoided since it may contribute to the incidence of late-onset paraplegia.

ENDOLUMINAL STENTING OF AORTA

This involves the use of stents specifically designed for each patient on the basis of spiral CT scans. Not all patients are suitable for this form of surgery. A stent is passed through the femoral route and placed in the aorta under X-ray control. It avoids the need for bypass or one-lung anaesthesia. It has minimal effect on the cardiovascular system because cross-clamping of the aorta is avoided. These are often performed in theatre under general anaesthesia but can be performed under local anaesthesia with sedation. Purpose-built hybrid theatres are now available in most major cardiovascular centres and allow for better facilities for the management of these patients. These patients require direct arterial monitoring but central venous monitoring may not always be necessary. Occasionally the left subclavian artery is occluded. This may, on rare occasions, necessitate carotid to axillary artery bypass surgery. Patients can be sent to the critical care unit for postoperative monitoring.

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CROSS-REFERENCES

Diabetes mellitus, Chapter 6
Hypertension, Chapter 2
One-lung anaesthesia, Chapter 28
Blood transfusion, Chapter 30

TRANSOESOPHAGEAL ECHO (TOE)

A highly specialized skill, it requires extensive experience and formal assessment. Full accreditation can be achieved through the British Society of Echocardiography. An in-depth coverage is beyond the scope of this book but a concise introduction is appropriate covering indications, contraindications and an introduction to the basic views.

It is accepted that TOE is mandatory for all but the most straightforward of cardiac surgical procedures, and some hospitals, including the authors', perform TOE on all patients without contraindications. It provides rapid evaluation of cardiac function and in some cases can identify previously unknown abnormalities, particularly mitral valve regurgitation, in patients presenting for CABG surgery.

TOE is particularly valuable in valve surgery, confirming adequacy of repaired valves and correct seating of replaced valves. Identification and quantification of operative complications such as an otherwise undetected paravalvular leak is essential as it enables discussion with the surgeon to determine if a small leak can be left alone or requires further surgical attention, even if this means going back onto CPB.

INDICATIONS FOR TOE

1. Evaluation of cardiac and aortic structure and function in situations where the findings will alter management and TTE is non-diagnostic or TTE is deferred because there is a high probability that it will be non-diagnostic.
   - Detailed evaluation of the abnormalities in structures that are typically in the far field such as the aorta and the left atrial appendage.
   - Evaluation of prosthetic heart valves.
   - Evaluation of paravalvular abscesses (both native and prosthetic valves).
   - Patients on ventilators.
   - Patients with chest wall injuries.
   - Patients with body habitus preventing adequate TTE imaging.
   - Patients unable to move into left lateral decubitis position.
2. Intraoperative TOE.
   - All open heart (i.e. valvular) and thoracic aortic surgical procedures.
   - Use in some coronary artery bypass graft surgeries.
   - Noncardiac surgery when patients have known or suspected cardiovascular pathology which may impact outcomes.
3. Guidance of transcatheter procedures.
   - Guiding management of catheter-based intracardiac procedures (including septal defect closure or atrial appendage obliteration, and transcatheter valve procedures).
4. Critically ill patients.
   - Patients in whom diagnostic information is not obtainable by TTE and this information is expected to alter management.

CONTRATINDICATIONS TO TOE

It is important that the patient understands the potential complications of TOE and, as with all

perioperative procedures, the risk of TOE needs to be carefully balanced against the risk of not performing it. In the case of TOE, some surgeons may not wish proceed to surgery without it.

There are relatively few absolute contraindications – oesophageal stricture, tumour, perforation or diverticulum and active upper GI bleed.

Relative contraindications need to be considered cautiously on an individual patient basis. They include:

- A history of radiation to the neck and mediastinum, recent upper GI surgery or bleed.
- Oesophageal varices, Barrett's oesophagus, active oesophagitis or peptic ulcer disease.
- Coagulopathy or thrombocytopaenia.
- Significant neck restriction (severe cervical arthritis, atlantoaxial dysfunction).
- History of dysphagia.

INTRAOPERATIVE TOE COMPLICATIONS

Severe complications from TOE are rare and generally it is a well-tolerated perioperative procedure. Intraoperative TOE is associated with:

- Mortality 0%–1.2%
- Major bleeding 0.03%–0.8%
- Endotracheal mal-position 0.03%

- Minor pharyngeal bleeding 0.1%
- Severe odynophagia 0.1%

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Vascular surgery

REDMOND P TULLY

ABDOMINAL AORTIC RECONSTRUCTION: ELECTIVE OPEN REPAIR

This is a major surgical procedure aimed at reducing the mortality from rupture of an aneurysm, or the symptoms of claudication in occlusive disease. Outcome depends on age and coexisting disease. Mortality is around 5%, with perioperative myocardial infarction being the principal cause.

Most patients are elderly with significant comorbidities, carrying major risks of perioperative cardiopulmonary morbidity and mortality. The procedure involves aortic cross-clamping with resultant haemodynamic and ischaemic complications. The incision may be midline, transverse or rarely a left retroperitoneal approach. In 90% of patients, a tube graft or a bifurcation graft is inserted below the origin of the renal arteries. Additional procedures including femoral crossover grafts or embolectomies are sometimes required to improve lower limb perfusion. With the increasing popularity of endovascular aneurysm repair, open elective procedures have become less common and many are confined to aneurysms which are anatomically unsuitable for EVAR.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

GOALS

- To identify and evaluate comorbidities
- To optimise the patient's condition where possible
- To make informed decisions for best management

Take a full history and examination and consider all risk factors/predictors, functional capacity assessment and scoring systems. In high-risk patients, the multidisciplinary decision may be not to offer surgery. Alternatively, surgery may be deferred until the procedure is indicated on the basis that the balance of benefits and risks has changed.

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INVESTIGATIONS

- FBC, urea and creatinine, electrolytes, glucose
- Group and save (cell salvage should be available)
- 12 lead ECG and echocardiography
- Stress cardiac evaluation (exercise ECG or dobutamine stress echo)
- Chest X-ray and pulmonary function tests
- CPET

PREOPTIMISATION

Ideally the patient should be seen 1 month preoperatively and risk factors assessed. Explanation concerning the procedure, management and postoperative care is important. Surgery should take precedence over time taken for lifestyle changes since delay could increase risk of aortic rupture.

PERIOPERATIVE MANAGEMENT

The aim is to maintain the balance of myocardial and end-organ oxygen supply and demand – avoid tachycardia, hypotension, hypoxia and hypothermia.

PREINDUCTION

- Reassure patient, maintain a calm environment
- Peripheral IV access with large bore cannulae
- 5 lead ECG: leads II and V5 with ST segment analysis
- Arterial line
- Thoracic epidural corresponding to the upper level of the incision

INDUCTION AND MAINTENANCE

- Avoid haemodynamic instability, especially at induction, laryngoscopy, intubation, cross-clamp on and release, extubation.
- Maintain with inhalational agents or TIVA. Remifentanil infusions are often used as they have the benefit of assisting with rapid control of cardiovascular parameters. Inhalational agents may confer myocardial protection and ischaemic preconditioning. Vasopressors or vasodilators may be required.

- The epidural will supplement anaesthesia intraoperatively although the vasodilation produced may worsen hypotension caused by cross-clamp release or bleeding. It is therefore often commenced only after the graft has been placed and the cross-clamp released.

MONITORING

- Routine AAGBI guidelines.
- Invasive arterial blood pressure and regular blood gases.
- Central venous catheter insertion postinduction.
- Cardiac output monitoring may guide filling status but be aware of its limitations in major vascular surgery. An oesophageal Doppler trace may be difficult to obtain during aortic cross-clamping. Uncalibrated pulse contour analysis is inaccurate when a clamp is applied. Changes in vascular tone may require regular recalibration of thermodilution-based devices to ensure accuracy. Trans-oesophageal echocardiography may be indicated in selected cases.
- Core temperature monitoring.
- Near patient coagulation testing, e.g. thromboelastography (TEG) may be helpful.

SPECIFIC POINTS

- Warming mattress, forced air warming blanket, humidify inspired gases, warm intravenous fluids. Warming the lower limbs during cross-clamping is not advised.
- Antibiotic prophylaxis as per local guidelines.
- Give heparin (5000 units) prior to aortic cross-clamping.
- Supine position – ensure potential pressure areas are padded. Table break may be required to improve surgical access.
- Aortic cross-clamping produces a marked increase in left ventricular afterload. Severe systemic hypertension and an increased left ventricular end-diastolic pressure may precipitate myocardial ischaemia. Consider regional technique with sympathetic block to reduce the response to cross-clamping or increasing depth of anaesthesia. Remifentanil

is especially useful at this point. Vasodilators such as glyceryl trinitrate or sodium nitroprusside may be needed.

- Aortic cross-clamp release may cause profound hypotension and myocardial ischaemia. Consider fluid preloading, controlled cross-clamp release by the surgeon, rapid fluid infusion guided by cardiovascular monitoring, administration of a vasopressor.
- Blood loss is usually 2–4 units. The need for blood transfusion can be reduced by intraoperative cell salvage, normovolaemic haemodilution or early administration of clotting factors. This may be guided by thromboelastography.

POSTOPERATIVE MANAGEMENT

- ICU admission to optimise the cardiovascular, renal and respiratory systems.
- Epidural analgesia is usually preferred; however, patient-controlled opioid analgesia alongside regular paracetamol infusion may be used.

PERIOPERATIVE COMPLICATIONS

SURGICAL

- Ischaemic limb due to embolisation
- Haemorrhage from graft anastomosis
- Ischaemia of the gastrointestinal tract
- Graft or wound infection
- Return to theatre may be required with all the attendant risks

MEDICAL

- Myocardial ischaemia
- Kidney injury
- Respiratory failure

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CROSS-REFERENCES

Vascular surgery – general principles, Chapter 17
Preoperative assessment of cardiac function, Chapter 25
Preoperative assessment of pulmonary function, Chapter 25
Cardiopulmonary exercise testing, Chapter 25

ABDOMINAL AORTIC RECONSTRUCTION: EMERGENCY REPAIR

A ruptured or leaking aortic aneurysm is fatal if untreated; emergency repair is the only chance of survival. Emergency surgery in a specialist unit is required because perioperative morbidity and mortality is very high. Overall mortality is over 60%. For those surviving to surgery, it is around 40%, compared to 5% for elective procedures. In certain patients, surgical intervention may be inappropriate. Scoring systems are used to predict mortality but do not replace overall clinical judgement. Effective analgesia and nursing care should be provided if surgery is not appropriate to ensure death is peaceful and dignified. Poorer outcomes are more likely with advanced age (>76 years), raised creatinine (>190 micromoles/L), Hb <9 g/dL, loss of consciousness and an ischaemic ECG (Hardman index).

The clinical presentation ranges from severe abdominal/lumbar pain with a palpable pulsatile abdominal mass, to complete cardiovascular collapse and sudden death. If cardiovascularly stable, a CT scan of the abdomen is performed, whereas the severely compromised patient is taken directly to theatre. Emergency EVAR is an alternative to open repair, but requires CT assessment. When the

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rupture is retroperitoneal, the haemorrhage may be slowed by a tamponade effect.

PREOPERATIVE ASSESSMENT, INVESTIGATIONS AND PREOPTIMISATION

Assessment, investigation and preoptimisation are carried out simultaneously. Ultimately, the need is for control of blood loss by aortic cross-clamp application. A second experienced anaesthetist is required to assist, although the procedure may need to commence whilst awaiting their arrival.

Resuscitation requires large bore IV access. Limit fluid resuscitation before cross-clamp application and accept a low blood pressure (e.g. 70 mmHg). Excessive fluid resuscitation risks greater haemorrhage with clot dislodgement and dilutional coagulopathy. If the blood pressure is adequate for myocardial and cerebral perfusion, then no fluid should be given. Blood may need to be administered to avoid a dilutional anaemia. Ensure adequate pain relief.

Formal detailed assessment and investigations are rarely possible, but attempts should be made to establish the severity of any coexisting disease and establish a medication history as anticoagulation may need to be reversed.

INVESTIGATIONS

- FBC, urea and creatinine, electrolytes, glucose, coagulation.
- Acid-base and lactate give an indication of the degree of shock.
- Cross-match blood, FFP, platelets. Activate the major haemorrhage protocol. Emergency cell salvage may be used to minimise transfusion requirements.
- 12 lead ECG may differentiate aneurysm leak/rupture and massive myocardial infarction.

PERIOPERATIVE MANAGEMENT

The aim is to ensure anaesthesia whilst attempting cardiovascular stability and normothermia.

PREINDUCTION

- Preoxygenation and induction in theatre.
- 5 lead ECG: lead II and V5, ST segment analysis.
- Urinary catheter.
- Arterial line insertion should not delay commencement of surgery. Central line insertion later unless other large access is not possible.
- Blood products and rapid infusers with fluid warmers; if cross-matched blood is not yet available, group O negative blood should be available.
- Have available a vasopressor (metaraminol, noradrenaline) and an inotrope (adrenaline).
- Position with both arms out for access.
- During skin preparation and draping, pre-oxygenation is continued.

INDUCTION AND MAINTENANCE

Rapid sequence induction (modified to minimise hypotension if needed). Ketamine is a suitable induction agent. As soon as the airway is secured with an endotracheal tube, instruct the surgeon to proceed. Anticipate cardiovascular collapse as the abdominal tamponade effect is lost. Ventilate with an inhalational agent in 100% oxygen. Anaesthetic requirement may be low until cross-clamp. Once the aorta has been cross-clamped, better haemodynamic stability may occur, facilitating the insertion of invasive monitoring lines.

INTRAOPERATIVE MONITORING

- As per AAGBI guidelines with:
  - ECG – leads II and V5 with ST segment analysis
  - Invasive arterial blood pressure - serial blood gases
  - Central venous catheter insertion post induction
  - Haemodynamic/cardiac output monitor, depends on availability and preference
- Core temperature monitoring
- Near patient coagulation testing, e.g. TEG

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SPECIFIC POINTS

- Continued haemorrhage is common. Large volumes of blood may be needed resulting in a dilutional coagulopathy. Early administration of FFP and platelets is recommended guided by TEG. Blood product administration should not wait until laboratory tests confirm deranged function.
- Aortic cross-clamp application leads to an increase in blood pressure proximal to the cross-clamp. Hypertension may be managed by an increase in anaesthetic (inhalational or remifentanil) or the use of a vasodilator (GTN).
- Aortic cross-clamp release may cause severe hypotension due to the reduction in afterload along with severe ischaemic reperfusion injury and metabolic acidosis. Controlled cross-clamp release and fluid loading reduce the impact. Further fluid and vasopressor/inotropic support is guided by invasive and/or oesophageal Doppler monitoring.
- Warming mattress, forced air warming blanket, humidify inspired gases, warm intravenous fluids.
- Antibiotic prophylaxis as per local policy.
- Supine position; ensure potential pressure areas are padded, a table break may be required to improve surgical access.
- Insert a nasogastric tube; postoperative ileus is common.

POSTOPERATIVE MANAGEMENT

- ICU for continued ventilation to optimise cardiovascular, renal and respiratory systems and ensure maintenance of normothermia and blood coagulation.
- Analgesia by intravenous opioid infusion with conversion to patient-controlled analgesia when awake. Regional block may be considered after correction of coagulopathy.
- Close monitoring of renal function is required as renal failure is common.

EMERGENCY ENDOVASCULAR REPAIR

If the anatomy of the aneurysm is suitable, endovascular repair of a ruptured aneurysm is possible. There is no difference in mortality between the approaches; however, extrapolation of the lower morbidity of EVAR in elective patients means that this approach may be favoured in patients with poor prognostic factors. Patient considerations are the same as open repair; however, the procedure may be carried out under local anaesthetic infiltration. An occlusion balloon may be deployed in the aorta to provide haemodynamic stability. Complications of emergency EVAR include postoperative abdominal compartment syndrome, which may require decompressive laparostomy.

PERIOPERATIVE COMPLICATIONS

SURGICAL

- Uncontrolled haemorrhage
- Ischaemic limb due to embolisation
- Ischaemia of GI tract or spinal cord
- Intra-abdominal hypertension (intra-abdominal pressure [IAP] ≥12 mmHg) and abdominal compartment syndrome (IAP ≥20 mmHg).

MEDICAL

- Myocardial infarction
- Kidney injury
- Respiratory failure
- Coagulopathy, DIC

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CROSS-REFERENCES

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Emergency surgery, Chapter 25
The elderly patient, Chapter 25

ABDOMINAL AORTIC RECONSTRUCTION: ENDOVASCULAR ANEURYSM REPAIR (EVAR)

INTRODUCTION

Endovascular surgery provides an alternative to conventional open repair. The technique can be used to repair thoracic and abdominal aneurysms. The advantages of decreased stress response, smaller incision, decreased fluid shifts and avoidance of aortic cross-clamping are reflected by a decrease in short-term mortality and morbidity. Mortality rates for open repair and EVAR subsequently converge (years) reflecting an increase in long-term complications such as endoleak in patients receiving EVAR. Patients have the same high-risk profile as those for open surgical repair.

Not all aneurysms are anatomically suitable for endovascular repair. New devices (e.g. fenestrated and branched grafts) have become available which have allowed some patients where EVAR would previously have been contraindicated to be included.

Anatomical aspects in decisions regarding suitability for EVAR include:

- Aneurysm neck. Length >15 mm for standard repair although specialised grafts are available for shorter necks. Should be relatively thrombus- and calcium-free. Angulation should be less than 60 degrees.
- Distal aorta. Adequate landing zone length and diameter.
- Ileo-femoral vessels. Relatively disease-free with good run off.
- Femoral artery diameter. Should exceed 7 mm to enable the collapsed stent to be inserted although low profile devices are available if smaller.

The procedure may be performed in the radiology suite or operating theatre and is carried out under fluoroscopic guidance, jointly by the vascular surgeon and radiologist. Hybrid theatre suites are increasingly being provided for this procedure. Surgical incisions are usually made to access the groins although percutaneous techniques are possible. A guide-wire is inserted under fluoroscopic guidance; the stent-graft delivery system is then inserted over the guide-wire and advanced to the diseased segment where it is deployed. Preparation must always be made for open repair (<2% incidence) and potential complications, including major haemorrhage.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Goals, assessment, risk factors, predictors, functional capacity, investigations: as for open-repair.

PERIOPERATIVE MANAGEMENT

Maintain the balance of myocardial and end-organ oxygen supply and demand by ensuring haemodynamic stability – avoid tachycardia, hypotension, hypoxia and hypothermia. The risk of contrast-induced nephropathy should be minimised with appropriate fluid management, maintenance of renal perfusion and reduced contrast time.

The possibility of conversion to open repair should be remembered and the patient should be aware of this. Anaesthesia is required for surgical access of the femoral arteries, and options include:

- General anaesthesia
- Tegional anaesthesia (spinal, epidural or CSEl) ± sedation
- Local infiltration anaesthesia with sedation

The procedure can be between 1½ and 5 hours. Spinal anaesthesia alone may be considered depending on the graft system used, its complexity and the anatomical complexity of the patient. A CSE is appropriate if the procedure may reasonably outlast the duration of a single shot spinal blockade. General anaesthesia may be preferred if the case is known to be complex. If local infiltration is chosen, this group will already be a select cohort, with predicted ease of access, fewer likely complications and lower BMI.

PRE-ANAESTHESIA

- Reassure patient, maintain a calm environment.
- Large bore IV cannulae.
- 5 lead ECG: lead II and V5, ST analysis.
- Arterial line under local anaesthetic.

INDUCTION AND MAINTENANCE/ REGIONAL ± SEDATION

Avoid haemodynamic instability, especially at induction, laryngoscopy, tracheal intubation and with the potential effects of regional anaesthesia. Maintenance with inhalational anaesthesia or TIVA. Sedation may be carried out with propofol or midazolam.

INTRAOPERATIVE MONITORING

- As per AAGBI guidelines
- Invasive arterial pressure
- Urinary catheter
- Core temperature monitoring
- Cardiac output monitoring may be used

SPECIFIC POINTS

- Antibiotic prophylaxis as per local guidelines.
- Heparinisation (5000 units) after the femoral artery is exposed.
- Hypertension caused by aortic occlusion during balloon deployment to seal the stent at the neck is usually transient and self-limiting.
- Potential remote site surgery.
- Occult bleeding from sheath ports accidentally left open is possible – have a high index of suspicion for this in case of haemodynamic disturbance.

POSTOPERATIVE MANAGEMENT

- Specialist care on a vascular ward is usually adequate although ICU may be required. Monitor renal function.
- Analgesia: paracetamol; opioid requirements will be minimal if intraoperative local anaesthetic infiltration or regional block have been used.

- Avoid NSAIDs.
- Early mobilisation is encouraged.

PERIOPERATIVE COMPLICATIONS

SURGICAL

- Endoluminal leaks from either the proximal or distal anastomosis
- Maldeployment of stent
- Graft thrombosis
- Graft migration
- Arterial rupture
- Aortic injury from instruments, occlusion or embolisation
- Distal organ embolisation and ischaemia: lower limb and renal
- Conversion to open repair
- Graft infection

MEDICAL

- Acute kidney injury; contrast induced nephropathy or ischaemia caused by mechanical obstruction of the renal arteries by the graft
- Myocardial ischaemia
- Postimplantation syndrome: fever, leucocytosis, raised CRP, rarely DIC and shock; usually self-limiting
- Sepsis

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CROSS-REFERENCES

ANAESTHESIA IN VASCULAR SURGERY: GENERAL PRINCIPLES

Vascular surgery includes the major operations of aortic reconstruction, carotid endarterectomy, arterial grafting of ischaemic legs and amputation of the limb if it is not salvageable. The most common underlying pathological condition is arterial atherosclerosis, which may also cause aneurysm formation.

Ruptured abdominal aortic aneurysm (AAA) usually presents with abdominal or back pain and cardiovascular collapse. Unruptured aneurysms are frequently asymptomatic, although they can cause vague symptoms due to stretching and direct pressure. Carotid artery disease may present with transient ischaemic attacks (TIA) or stroke. Peripheral arterial occlusive disease causes ischaemic symptoms.

AAA refers to an aorta diameter of ≥30 mm. They occur in 10% of men and 3% of women over the age of 65. The most important risk factor is smoking, but it is also more common in those with hypertension, hyperlipidaemia and a family history. A national screening programme using ultrasonography has been established for all men from their 65th birthday. Once the aneurysm diameter reaches 55 mm, surgery is usually offered. The risk of rupture increases exponentially as the size of the aneurysm increases. Emergency surgery for rupture carries a mortality risk of around 50%, compared with a mortality of around 5% for elective open repair.

Carotid endarterectomy (CEA) aims to reduce the risk of cerebral ischaemic events and is usually offered to those who are symptomatic with carotid artery stenosis >70%, within 2 weeks of presentation.

The most common symptom of peripheral vascular disease (PVD) is intermittent claudication, occurring when blood flow is reduced by ≥75% (this corresponds to a ≥50% reduction in arterial diameter as seen on an angiogram). The presence of permanent rest leg pain indicates that blood flow is reduced by ≥90% (which corresponds to a ≥70% reduction in arterial diameter as seen on an angiogram). These patients require urgent treatment.

ASSESSING PERIOPERATIVE RISK

RISK FACTORS AND PREDICTORS

The American College of Cardiology (ACC) and American Heart Association (AHA) have produced guidelines for evaluating cardiac risk in non-cardiac surgery. These use clinical factors in addition to functional capacity and surgical risk stratification to predict cardiac risk. All vascular procedures have an elevated cardiac risk. The majority of patients are elderly, M:F 5:1. Younger patients often have a family history, are smokers or have a history of diabetes mellitus. Most vascular patients are ASA 3 or above.

A preoperative history of cardiac ischaemia is common, but cardiac failure is a more important predictor for postoperative morbidity and mortality. Several prediction tools exist to quantify risk: Goldman, Detsky and Lee. Lee's Revised Cardiac Index uses six predictive factors to determine perioperative cardiac risk: high risk surgery, ischaemic heart disease (IHD), congestive cardiac failure (CCF), renal insufficiency (RI), insulin dependent diabetes mellitus (IDDM) and cerebrovascular disease (CVD). Advanced age (>70 years) and hypertension are also recognised factors.

COMORBIDITIES

- *IHD* – Perioperative myocardial infarction occurs mostly in the first postoperative week, with the greatest risk on the third postoperative day. Coronary stenting and coronary artery grafting should only be done prior to non-cardiac surgery if these interventions are deemed necessary irrespective of the proposed vascular operation.

Perioperative management of a patient with a pre-existing stent should be a multidisciplinary decision because of the balance of risk between haemorrhage and thrombosis.

- *Heart failure* – Diastolic or systolic dysfunction.
- *Chronic kidney disease* – Serum Cr >170 micromoles/L indicates a high-risk patient.
- *Diabetes mellitus* – Diabetic patients comprise a large number of those undergoing peripheral arterial reconstruction. Macro- and microvascular complications result. Amputation of toes and limbs is commonly undertaken to control infection, which may be adversely affecting diabetic management. Good glycaemic control is essential in the perioperative period.
- *Cerebrovascular disease* – A marker of atherosclerosis, procedure may be complicated by a perioperative stroke.
- *Hypertension* – Left ventricular hypertrophy requires higher filling pressures to achieve adequate cardiac output and can progress to heart failure. In brain and kidneys, autoregulation of blood flow is shifted to the right. The widespread increase in arteriolar resistance means that patients manifest exaggerated intraoperative haemodynamic changes.
- *Chronic obstructive pulmonary disease* (*COPD*) – Perioperative nebulisers and physiotherapy should be considered.

FUNCTIONAL CAPACITY

- Medical history including metabolic equivalents (METs) and scoring systems (e.g. the Duke Activity Status Index) have been used to assess maximum physical activity. Exercise can be limited by claudication and mobility, although if a patient can climb two flights of stairs, equivalent to >4 METs, they may not require further testing.
- Cardio-pulmonary exercise testing (CPET) is the gold standard for combined assessment of functional capacity of both cardiac and respiratory systems. CPET helps determine an individual's physiological reserve in order to

predict their ability in coping with the stress of surgery and postoperative recovery. It also aids decision making with regards to whether surgery should proceed and procedure choice. Current opinion considers a patient to be high risk if their anaerobic threshold (AT) is <11 mL/kg/min.

CONCURRENT MEDICATION

Patients frequently take multiple medications such as anticoagulants, cardiac medications and broncho-dilators. Most drugs should be continued periopera-tively. However, there are some that require further consideration:

- Clopidogrel should be stopped 7 days before planned surgery if a neuraxial technique is planned.
- Warfarin needs to be stopped at least 3 days prior to planned surgery with an INR check.
- Heparin (infusion or LMWH): consider timing of neuraxial block in relation to this.
- ACE inhibitors may be omitted on the day of surgery to minimise intraoperative hypotension.
- Statins must be continued as cardiac risk increases if stopped.
- Beta-blockers should be continued.

OTHER CONSIDERATIONS

- *Hypothermia* – Inadvertent hypothermia (core temperature <36.0°C) should be avoided in order to reduce the complications of infection, delayed healing, bleeding, arrythmias and protracted hospital stay. Preoperative warming should be considered. Intraoperatively, active patient warming should be carried with the use of fluid warmers and forced air blankets.
- *Remote location* – An increasing number of vascular procedures, such as angioplasty and stenting of vessels, involve interventional radiology. In some hospitals, anaesthesia is provided in the radiology suite to facilitate this. Appropriate facilities and support must be available in these environments which

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may be remote from the theatre complex. Purpose-built hybrid theatres with the facility to allow complex interventional radiology and open surgery to both be performed provide a solution to this problem and allow more combined radiological and surgical procedures to be performed.

- *Governance* – The Royal College of Anaesthetists has published guidelines for the provision of anaesthetic services pertaining to vascular anaesthesia. These suggest specific resources and standards required to deliver high quality care for vascular patients. The importance of adequate preoperative assessment and the involvement of the anaesthetist in multidisciplinary planning of a patient's treatment is emphasised. There is increasing scrutiny of outcomes in vascular surgery with all centres being required to submit data to the National Vascular Registry and associated quality improvement programs. There is pressure for a smaller number of higher volume units to reduce variation in care and patient outcome.

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CAROTID ENDARTERECTOMY (CEA)

INTRODUCTION

Carotid artery disease causes symptoms of cerebral ischaemia, which may reverse within 24 hours (transient ischaemic attack; TIA) or last >24 hours (stroke). It is usually caused by atheromatous disease leading to narrowing at the carotid bifurcation, reducing cerebral blood flow and initiating platelet or clot embolism into the distant cerebral vessels.

The aim of CEA is to reduce the risk of cerebral ischaemic events, with greater benefit seen in patients with symptomatic disease. It is a prophylactic operation but carries a high morbidity (6%) and mortality (3%) due to the patient population and perioperative complications. The majority of deaths are due to stroke or MI.

An incision is made along the anterior border of the sternocleidomastoid muscle, the carotid and internal jugular vessels are dissected and cross-clamps applied sequentially to the common, internal and external carotid arteries. Following the endarterectomy, the artery may be closed directly or, more commonly, increased in diameter by a patch angioplasty.

In symptomatic patients, there is good evidence that surgery should be performed in those with carotid stenosis >70%. UK guidelines suggest the surgery should take place within two weeks of development of symptoms (TIA or stroke). In asymptomatic patients, the operation may not be as beneficial.

Surgery may be carried out under local (LA) or general anaesthesia (GA). It was previously presumed that LA would lead to better outcome; however, the general anaesthesia versus local anaesthesia for carotid surgery trial failed to demonstrate this.

At the time of cross-clamping there is a risk of cerebral hypoperfusion and ischaemia, with blood flow to the ipsilateral hemisphere being dependent on

collateral supply from the Circle of Willis. To prevent this, a shunt may be inserted between the internal carotid artery and the common carotid artery. Shunt complications include kinking, embolic complications and damage to the arterial wall. The need for shunt insertion is determined either by surgical preference or from the estimated cerebral perfusion, guided by cerebral monitoring. There is no ideal cerebral monitor.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

History and examination, risk factors, scoring systems and functional capacity are important. Document preoperative neurological status. Hypertension is commonplace and carries the risk of perioperative haemodynamic instability with reduced cerebral perfusion and potential haemorrhage.

INVESTIGATIONS

- FBC, urea and electrolytes, glucose, group and save
- ECG; other cardiac investigations as indicated

Whilst preoptimisation may be possible in some cases, others must undergo surgery without such delay.

PERIOPERATIVE MANAGEMENT

The aim is to ensure adequate cerebral and myocardial perfusion via maintenance of blood pressure, PaO_2, $PaCO_2$ and normothermia. Blood pressure must be most closely controlled during cross-clamping with mean arterial pressure being kept either at baseline or even augmented to 20% above baseline. Adequate venous drainage should be ensured.

- Reassure patient
- Peripheral IV access
- Monitoring as per AAGBI guidelines
- 5 lead ECG: lead II and V5 with ST segmental analysis
- Arterial line

ANAESTHETIC TECHNIQUE

- General anaesthetic
- General anaesthetic with local anaesthetic/ block

- Deep plus superficial cervical plexus block
- Superficial cervical plexus block alone

INDUCTION AND MAINTENANCE

Take care to prevent or minimise haemodynamic instability, in particular, at high-risk times such as induction, laryngoscopy, intubation, cross-clamp application, surgical stimulation of carotid sinus and extubation. This may be achieved using an inhalational agent and muscle relaxant or TIVA. Attenuation of pressure responses may be achieved with opiate boluses or a remifentanil infusion. The aim is to reduce $CMRO_2$ and therefore O_2 demand. Avoid nitrous oxide because it increases cerebral metabolic rate and cerebral blood flow, and impairs CO_2 response of the cerebral vasculature. It also worsens air emboli. An endotracheal tube is used, either regular or reinforced. Lack of access to the airway intraoperatively must be remembered. Supplementation with local anaesthesia may be used. Hypotension must be avoided as it risks cerebral hypoperfusion – vasopressors may be required.

GA ADVANTAGES

- Provides more controlled operating conditions.
- Avoids need for patient compliance.
- Reduces $CMRO_2$.
- Reduces catecholamine release and stress response of surgery.
- Allows greater cardiovascular control/ pharmacological manipulation.

GA DISADVANTAGES

- Reduces CBF.
- May lead to more frequent, unnecessary shunt use with associated complications.
- Risks failure to detect cerebral ischaemia post–cross-clamp insertion.
- Haemodynamic fluctuations associated with induction, laryngoscopy and extubation.

An alternative technique is to use superficial cervical plexus block in combination with GA.

CAROTID ENDARTERECTOMY UNDER LOCAL ANAESTHESIA

Local anaesthesia requires blockade of the C2/3/4 dermatomes. Some cover of cranial nerve V (mandibular branch) may be needed for surgical retraction. Supplemental local infiltration into the carotid sheath may be required.

C2-4 cover may be achieved by:

- Superficial cervical plexus block alone (most common and provides adequate analgesia in most patients)
- Deep cervical plexus block (increases risk of complications)
- Combination of the above

Sedation must be carefully titrated to avoid losing the ability to perform neurological monitoring of the patient. Remifentanil is ideal. It reduces the need for local anaesthetic supplementation by the surgeon. The infusion can be stopped before cross-clamping and reliably allow neurological examination. If neurological function becomes disturbed after application of the cross-clamp, the blood pressure should be augmented and high flow oxygen administered initially. This may resolve the symptoms. The surgeon will likely proceed to insert a shunt.

LA ADVANTAGES

- Allows direct cerebral function monitoring
- Reduces unnecessary shunt insertion
- Avoids the haemodynamic instability associated with induction, laryngoscopy, intubation and extubation
- Preserves cardiovascular and cerebrovascular autoregulation

LA DISADVANTAGES

- Poor access to airway
- Requires patient compliance
- May be claustrophobic, uncomfortable and stressful for patient
- May provide inadequate analgesia requiring supplementation, sedation or general anaesthesia
- Conversion to GA is likely to be hurried and uncontrolled

- Possible inadvertent injection into the vertebral artery or subarachnoid space, phrenic nerve block with respiratory compromise and haematoma formation (greater risk with deep cervical block)
- Potential greater stress response, increasing cardiac and $CMRO_2$ consumption with possible ischaemia

INTRAOPERATIVE MONITORING

- As per AAGBI guidelines
- Direct arterial pressure
- Cerebral function
- Core temperature
- Haemodynamic if clinically needed

CEREBRAL FUNCTION MONITORS (CFM)

An awake patient is the best for monitoring cerebral function during cross-clamping. Under GA there is no ideal way of monitoring adequacy of cerebral perfusion but options may include:

- Transcranial Doppler of the middle cerebral artery (MCA)
- Near infrared spectroscopy (NIRS) measuring oxygen saturation of the cerebral hemisphere at risk
- Processed EEG: bispectral index or compressed spectral array

SPECIFIC POINTS

- Antibiotic prophylaxis as per local policy.
- Heparin 5000 IU or 100 U/kg bolus prior to cross-clamping.
- Reverse Trendelenburg with head turned away from operating side, shoulders raised with support between shoulder blades, head ring.
- Surgical stimulation of the carotid sinus may cause bradycardia plus hypotension and rarely asystole. This can be treated by removing the surgical stimulus, administration of vagolytic drugs or by injection of lidocaine around the carotid sinus nerve. Postoperatively, this may result in hypotensive episodes.

POSTOPERATIVE MANAGEMENT

- Close monitoring with extended recovery for several hours and then specialist vascular ward care or ICU with close observation of neurology and blood pressure.
- Analgesia: LA and paracetamol.

PERIOPERATIVE COMPLICATIONS

SURGICAL

- Haematoma and oedema: airway oedema is very common, additional haematoma can cause airway compromise necessitating re-exploration.
- Stroke.
- Nerve damage: hypoglossal nerve > recurrent laryngeal nerve > superior laryngeal nerve, marginal mandibular nerve and great auricular nerve injuries (most likely due to traction, usually transient).

MEDICAL

- Myocardial ischaemia.
- Labile BP: postoperative hypertension may occur, possibly due to baroreceptor dysfunction. Marked hypertension may injure the myocardium and lead to hyperperfusion syndrome. Hypotension is also a risk.
- Hyperperfusion syndrome: leads to a significant increase in cerebral blood flow following removal of the stenosis. This can cause: headache, hypertension, seizures, neurological deficit, cerebral oedema, subarachnoid haemorrhage, intracranial haemorrhage and death. Requires emergency treatment with antihypertensive therapy in a critical care environment.

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Preoperative assessment or cardiac function, Chapter 25

Preoperative assessment of pulmonary function, Chapter 25

Vascular surgery – general principles, Chapter 17

LEG REVASCULARISATION AND AMPUTATIONS

Peripheral revascularisation surgery is classed as having elevated risk with perioperative cardiac complications being the most frequent. The greatest risk is in patients requiring emergency surgery for proximal arterial occlusion with limb threatening ischaemia, where time is limited for preoperative management. With chronic limb ischaemia, the 12-month mortality is around 35%. Surgery, although relatively non-invasive, is usually prolonged and is carried out on patients with significant comorbidities. Almost all patients with significant peripheral vascular disease are current or previous cigarette smokers. In severe cases, acute limb ischaemia is a preterminal event and surgery may be inappropriate.

Acute limb ischaemia presents with a pale, pulseless, paraesthetic leg, is usually caused by an embolism and is managed with surgical embolectomy, often under local anaesthetic. Further surgery to improve the arterial blood supply may include angioplasty, thrombolysis, stenting, and bypass grafting; this is usually carried out under general anaesthesia.

Ideally, revascularisation should be performed within 6 hours of the onset of critical ischaemia.

Chronic limb ischaemia, presenting with symptoms of claudication, is due to atheromatous disease. These patients are more likely to have an established collateral circulation. The surgical procedures commonly encountered are femoro-popliteal or femoro-femoral bypass grafting.

Patients may present for repeated procedures in an attempt to salvage ischaemic limbs, sometimes culminating in progressive proximal amputation in the following sequence: (1) toes, (2) forefoot, (3) below knee, (4) above knee. Risk increases with the number of procedures carried out. Many patients are diabetic, with infected and necrotic tissue disturbing blood sugar control.

PREOPERATIVE ASSESSMENT, INVESTIGATION AND PREOPTIMISATION

Preoperative goals will depend on the urgency of surgery. For emergency procedures, investigations are likely to be limited to blood tests and an ECG.

A full history should be taken and appropriate examination performed. When time is available, and if appropriate, risk factors and predictors can be more formally assessed, as can functional capacity.

Chronically ischaemic limbs may be infected and the patient may have systemic signs of sepsis.

Anticoagulant drugs are often prescribed either chronically to reduce risk or they may have been given acutely to try to improve circulation to the ischaemic limb (anti-platelet agents or heparin). This may contraindicate regional anaesthesia, especially if surgery is too urgent to wait for the effect to be reversed.

INVESTIGATIONS

- FBC, urea and electrolytes, glucose, group and save, coagulation studies
- 12 lead resting ECG; consider other tests as appropriate

PERIOPERATIVE MANAGEMENT

The aim is to ensure anaesthesia with cardiovascular stability, avoiding hypoxia, hypercarbia, hypothermia and hyper/hypoglycaemia.

The choice of anaesthetic technique includes:

- General anaesthesia (GA) – (inhalational agent or TIVA)
- Regional anaesthesia (RA) – single shot spinal, CSE or femoral and sciatic nerve blocks
- Combined general and regional anaesthesia

GA may be preferred due to uncertainty over the duration of surgery; however, regional anaesthesia and nerve blockade have the advantage of reducing opioid requirements postoperatively. Regional techniques are preferred if the patient has severe respiratory disease and may even be used when anticoagulant drugs would normally contraindicate them if the risk of GA is considered higher than the risk of epidural haematoma. A Cochrane review has not demonstrated any difference in mortality or myocardial infarction rate between neuraxial anaesthesia and GA. The rate of pneumonia was, however, lower with neuraxial anaesthesia.

PREANAESTHESIA

- Reassure patient, maintain a calm environment
- IV access
- Arterial line
- Discuss RA techniques if relevant

INDUCTION AND MAINTENANCE

Aim to maintain cardiovascular stability, especially at induction, laryngoscopy, intubation and extubation.

Maintain with inhalational agent or TIVA with propofol and remifentanil. The airway may be maintained with an ETT or laryngeal mask with IPPV.

INTRAOPERATIVE MONITORING

- As per AAGBI guidelines with:
  - 5 lead ECG: lead II, V5 with ST analysis
  - Invasive arterial blood pressure monitoring may be indicated
- Core temperature monitoring
- Urinary catheter
- Haemodynamic cardiac output monitoring may be indicated

SPECIFIC POINTS

- Blood loss is usually minimal apart from patients on anticoagulants.
- Warming mattress, forced air warming blanket, humidify inspiratory gases, warm intravenous fluids.
- Heparin (5000 units) bolus for reconstructive procedures.
- Diabetic patients will require intraoperative glucose control.
- Antibiotic prophylaxis as per local guidelines.
- Supine; ensure potential pressure areas padded/protected.
- Prolonged procedures under regional anaesthesia may require sedation or conversion to GA, plans for which should be in place.

POSTOPERATIVE MANAGEMENT

- Although high risk patients, the majority return to the vascular ward.
- Analgesia includes regular paracetamol; opiates (once regional block has worn off). Avoid NSAIDs.

PERIOPERATIVE COMPLICATIONS

SURGICAL

- Persistent ischaemia
- Progression to gangrene with localised or systemic infection
- Repeat surgery
- Poor wound healing

- Reperfusion injury following revascularisation of a critically ischaemic limb may cause hyperkalaemia, cardiac arrhythmias or arrest, myoglobinuria, and AKI
- Compartment syndrome

MEDICAL

- Myocardial ischaemia
- Rhabdomyolysis
- Kidney injury
- Respiratory failure
- Unstable diabetic control

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Preoperative assessment of cardiac function, Chapter 25
Preoperative assessment of pulmonary function, Chapter 25
The elderly patient, Chapter 25

Ophthalmic surgery

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ROGER MARTIN SLATER

ANAESTHESIA IN OPHTHALMOLOGY: GENERAL PRINCIPLES

Anaesthetists should be aware of all the nonsurgical factors in respect of the safe running of the ophthalmic operating schedule. Their roles and responsibilities may be:

- Preoperative assessment lead.
- Administering local anaesthetics with or without sedation.
- Providing general anaesthesia for adults and children.
- Administering intravenous antibiotics, steroids, intraocular pressure reducing medications.
- Monitoring, preventing and management of adverse events.

RECENT ADVANCES IN OPHTHALMIC SURGERY

- The change to day care under local anaesthesia for the majority of patients.
- The screening of patients by nurses trained in preoperative assessment, with medical anaesthetic input as required.
- Over 90% of cataract surgery is now performed under local anaesthetic without sedation.
- Preoperative fasting is generally not necessary for procedures under local anaesthesia unless sedation is also to be administered, in which case local policy should be followed.

SPECIFIC PREOPERATIVE INVESTIGATIONS

- Clotting profile should be checked within 24 hours of surgery for patients on anticoagulants.
- Electrolytes on the day of surgery for patients on dialysis.
- If a peribulbar anaesthesia is planned, axial length measurement should be recorded; high myopes with axial length >26 mm, are more likely to have staphylomas (posterior bulge of sclera diagnosed by ultrasound B scan) and are more at risk of perforation of the globe.

LOCAL ANAESTHETIC TECHNIQUES USED FOR OPHTHALMIC SURGERY

- Topical anaesthesia alone, or with intra-cameral local anaesthetic.
- Sub-conjunctival anaesthesia.
- Sub-Tenon's anaesthesia.
- Peribulbar (extraconal) anaesthesia.
- Retrobulbar (intraconal) anaesthesia.

ADMINISTRATION OF LA

- Sub-Tenon's blocks for complex procedures and peribulbar or retrobulbar blocks should only be performed by a trained anaesthetist or ophthalmologist.
- Appropriately trained/regulated and indemnified nonmedical staff may administer subconjunctival or sub-Tenon's blocks for selected ambulatory cataract surgery provided monitoring is appropriate.

CONTRAINDICATIONS TO LOCAL ANAESTHESIA INCLUDE

- Patient refusal; local sepsis; trauma or perforated globe; grossly abnormal coagulation; severe reaction (e.g. allergy) to local anaesthetic; patient confusion or inability to communicate; uncontrolled tremor; inability to adopt acceptable positioning.

SEDATION

- Useful to relieve anxiety but should not be used to compensate for inadequate LA block.
- Should facilitate but not complicate surgery.
- Complications include restlessness, sneezing with eye blocks, airway obstruction.
- Ensure patient understands that despite sedation they may be aware of parts of the surgical procedure.

COMPLICATIONS OF LOCAL ANAESTHETIC BLOCKS

MINOR

- Subconjunctival haemorrhage, chemosis. Common with sub-Tenon's block.

MAJOR

- Orbital haemorrhage: rare complication of sharp needle injection and sub-Tenon's block. Bleed at orbital apex can cause a compartment syndrome with potential compression of optic nerve and central retinal artery. Such a retrobulbar haemorrhage is an ophthalmic emergency. Features are of pain, proptosis, ecchymoses of eyelids and reduction of vision. Treatment is medical with acetazolamide and mannitol, and if needed: surgical decompression.
- Globe perforation: generally occurs with sharp needle techniques. Suspect if severe pain on local anaesthetic injection. Intraoperative signs are of hypotony, reduced red reflex due to vitreous haemorrhage. Vitreoretinal surgical input is required.
- Brain stem anaesthesia (confusion, convulsions, cardiorespiratory compromise) is usually associated with a retrobulbar block using a long needle.
- Nerve injury: optic nerve can be damaged by direct trauma, ischaemic damage from haemorrhage, excessive pressure externally or excess local anaesthetic volume. Other nerves may be damaged by direct needle trauma.

- Muscle palsy leading to diplopia and ptosis. If lasts long term may be due to muscle damage from intramuscular injection of local anaesthetic, local anaesthetic myotoxicity, surgical trauma, antibiotic injection.

ORBITAL REFLEXES

- *Oculo-cardiac reflex* – Trigemino-vagal reflex. Can produce bradycardia or other abnormal rhythms. Produced with eyeball pressure, extraocular muscle traction, orbital haematoma, ocular trauma, eyelid retraction. Can occur in the enucleated orbit. Periocular anaesthesia may prevent these.
- *Oculo-respiratory reflex* – Shallow breathing, slow rate, seen in squint surgery.
- *Oculo-emetic reflex* – Trigemino-vagal. High incidence after squint surgery. Peri-ocular anaesthesia may prevent it.

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CATARACT SURGERY

A cataract is opacity of the lens which occurs when the lens protein becomes denatured causing a significant reduction in sight. The causes may be metabolic, infective, vascular or traumatic (Table 18.1).

Table 18.1 Underlying causes of cataracts

Congenital	Aquired
Down syndrome	Age related
Myotonic dystrophy (age 20 > 90%)	Diabetes mellitus
Hypothyroidism	Trauma
Hypocalcaemia	Irradiation
Galactosaemia	Drugs (steroids)
Hurler syndrome	
Phenylketonuria	
Lowe syndrome	
Pierre Robin syndrome	
Neurofibromatosis type 2	
Intrauterine infection (rubella, toxoplasmosis, varicella)	

PROCEDURE

PAEDIATRIC CATARACT

- Early surgery to maximise visual outcome.

ADULT CATARACT

- Phacoemulsification (via a small incision) is the preferred method of cataract surgery but extracapsular surgery is occasional necessary.
- Intraocular lens implant is routine.

PATIENT CHARACTERISTICS

- The elderly with coexisting medical conditions (hypertension, ischaemic heart disease, diabetes mellitus, chronic obstructive pulmonary disease)
- Neonates and young children with associated congenital syndromes

PROBLEMS

- Airway not accessible because of the operating microscope
- Immobile, centrally located eye with dilated pupil and uniform intraocular pressure (IOP) provide optimal surgical conditions
- Variable length of surgery from 10 to 90 min

PREOPERATIVE ASSESSMENT

- Assessment and optimisation of associated or coexisting medical conditions as required.
- ECG required if pulse irregular or abnormal, recent chest pain, uncontrolled hypertension or syncope.
- Obtain recent INR result if on warfarin (proceed if within therapeutic range or lower).
- Assess suitability for local anaesthesia.
- Assess suitability for day-case surgery.

PREMEDICATION

- Local anaesthetic cream in children
- Atropine 0.02 mg/kg intramuscularly in neonates

LOCAL ANAESTHESIA

- Local anaesthesia is the anaesthetic of choice for routine cataract surgery in adults

MONITORING FOR LOCAL ANAESTHESIA

- Patient communication with hand holding
- Pulse oximeter
- Clinical observations
- Intravenous access when sharp needle local anaesthetic techniques used

TECHNIQUES OF LOCAL ANAESTHESIA

- Thorough knowledge of orbital anatomy required.
- *Topical anaesthesia*: 1% amethocaine or 0.4% oxybuprocaine provide good surface anaesthesia of the globe without the complications of a regional block and are frequently used alone for phacoemulsification cataract surgery. May require intraoperative supplementation with intracameral lidocaine 1%. Does not provide akinesia and may fail to provide an adequate sensory block.

- *Sub-Tenon's block*: following topical anaesthesia with proxymetacaine, then povidone-iodine instillation, 3–4 mL of local anaesthetic solution (2% lidocaine plain or a 2% lidocaine and 0.5% bupivacaine mixture, with hyaluronidase 15 IU/mL [unless there is a history of allergy]) is introduced to the sub-Tenon's space through a small incision in the inferior nasal quadrant of the eye using a blunt sub-Tenon's cannula. Ocular compression is not routinely required with this technique. This provides excellent anaesthesia and akinesia without the potential complications of sharp needle techniques. However, scleral explants and previous retinal surgery may make sub-Tenon's block difficult or impossible. Ocular pemphigoid and Stevens–Johnson syndrome are contraindications to sub-Tenon's block because of the need for preservation of the cornea.
- *Peribulbar block*: introduced to avoid complications of retrobulbar blocks. A 25-gauge 25 mm needle is used to place 5–10 mL of the anaesthetic solution (as for sub-Tenon's) outside the muscle cone with the needle tip no further back than the equator of the globe. One or two injections are used, infero-lateral and medial to the globe. With larger volumes of anaesthetic, ocular compression may be required to lower IOP.
- *Sedation*: it is vital to have full cooperation from the patient under local anaesthesia. However, the use of small doses of intravenous benzodiazepines (0.5–1.0 mg midazolam) in anxious patients is beneficial.

GENERAL ANAESTHESIA

- Routine monitoring for general anaesthesia (AAGBI guidelines).
- Technique aims to provide stable conditions avoiding any rises in IOP with an immobile central eye.
- The surgery is not stimulating apart from the sub-conjunctival antibiotic injection at the end.

INDUCTION

- Intravenous in adults with propofol provides a low IOP.

- Intravenous or inhalation in children depending on preference.

AIRWAY MANAGEMENT

- Controlled ventilation with low dose atracurium or rocuronium and alfentanil 0.01 mg/kg or an infusion of remifentanil 0.1 microgram/kg/min is appropriate.
- A reinforced laryngeal mask is ideal unless contraindicated (risk of high inflation pressures) as it minimises coughing on emergence.
- Neonates require intubation.

MAINTENANCE

- Controlled ventilation with volatile agent or TIVA
- Antiemetic prophylaxis if patient susceptible to PONV

EMERGENCE AND RECOVERY

- Antagonise residual neuromuscular block and extubate awake
- With small incision surgery some coughing is not a problem

POSTOPERATIVE MANAGEMENT

- Little postoperative pain, simple oral analgesia is adequate.
- Eye requires padding while corneal reflex is diminished due to local anaesthetic block.
- Routinely managed as day case.

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CORNEAL TRANSPLANT

The cornea is the most important focussing element of the eye. Scarring causes impaired focussing. Endothelial cells maintain a clear cornea. Endothelial cell loss occurs with age and with inflammation inside the eye and following eye trauma/surgery. Corneal graft surgery places a new cornea with a full complement of endothelial cells.

PROCEDURE

PENETRATING KERATOPLASTY

- A full thickness disc of cornea is cut out and replaced with donor cornea that is sutured in place.

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LAMELLAR KERATOPLASTY

- The inner layers of the cornea are left intact and the donor cornea is sutured on top.

PATIENT CHARACTERISTICS

- Atopy associated with the corneal condition of keratoconus
- Associated connective tissue disorders such as rheumatoid arthritis, SLE, Wegener's granulomatosis, sarcoidosis
- Paediatric corneal disease in Down, Alport, Marfan, Goldenhar syndromes, fetal alcohol syndrome, myotonic dystrophy, achondroplasia

PROBLEMS

- Airway not accessible under the operating microscope.
- Sudden changes in choroidal blood volume and in episcleral venous pressure can compromise the operating field by marked swings in intraocular pressure.
- Less suitable for local anaesthesia, in view of risks of coughing and hypertensive responses.

PREOPERATIVE ASSESSMENT

- Identification and optimisation of associated or coexisting medical conditions.
- Routine grafts may be managed as day-case procedures.

ANAESTHETIC MANAGEMENT

- Routine monitoring as per AAGBI guidelines.

LOCAL ANAESTHESIA

- For uncomplicated corneal grafts.
- May be appropriate in medically compromised patients.
- Technique used must provide akinesia, IOP control and last for the duration of the surgery (up to 90 min).

- Paralysis of orbicularis orbis is needed to prevent squeezing of the eye.
- *Peribulbar anaesthesia*: 10 mL of a mixture of 2% lignocaine and 0.5% bupivacaine with hyaluronidase 150 units given as an inferolateral and a medial caruncle injection. Ocular compression for 10–15 min prior to surgery to reduce IOP.
- *Sub-Tenon's anaesthesia*: 5 mL 0.5% bupivacaine with hyaluronidase 75 units provides lasting anaesthesia and akinesia of the globe. Orbicularis orbis is blocked with a medial caruncle injection of 2–3 mL of the same anaesthetic solution. Ocular compression to reduce IOP.
- *Sedation*: as with all intraocular surgery patient cooperation is required, but small doses of intravenous benzodiazepine (midazolam 0.5–1.0 mg) or low dose propofol TCI (target blood conc. 0.5–1 µg/mL) can make the procedure more comfortable for the patient. With sedation, oxygen supplementation and careful monitoring are vital as there is limited access to the patient. Careful attention to comfort and warmth ensure a relaxed and still patient.

GENERAL ANAESTHESIA

INDUCTION

- Total intravenous anaesthesia (TIVA) with target-controlled infusion (TCI) of propofol and a remifentanil infusion provides excellent operating conditions.
- TIVA also provides rapid and smooth emergence from anaesthesia, with reduced postoperative nausea and vomiting.
- Alternatively, a balanced technique using intravenous induction with propofol, fentanyl or remifentanil infusion, muscle relaxant and volatile agent is suitable.

AIRWAY MANAGEMENT

- A reinforced laryngeal mask, unless contraindicated, provides ideal emergence conditions.

- With an open eye the airway must be secure and a tracheal tube should be used if there is any doubt.

MAINTENANCE

- Nondepolarising relaxant (e.g. atracurium or rocuronium) is used for controlled ventilation and to ensure an immobile central eye.
- Monitor carefully to avoid sudden movements.
- Moderate hyperventilation reduces IOP.
- Antiemetic prophylaxis with ondansetron 4 mg intravenously.

EMERGENCE AND RECOVERY

- Antagonise residual neuromuscular block with neostigmine and glycopyrrolate.
- Extubate awake.

POSTOPERATIVE MANAGEMENT

- Antiemetic prescribed in case of PONV.
- Regular paracetamol, NSAIDs, PRN codeine (max 240 mg/24 h).
- Pain may be due to raised IOP, acetazolamide may be required.

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CROSS-REFERENCES

Ophthalmic surgery – overview, Chapter 18
Rheumatoid disease, Chapter 8
Total intravenous anaesthesia, Chapter 28
Day-case surgery, Chapter 25
Artificial airways, Chapter 26
Infants and children, Chapter 24

INTRAOCULAR PRESSURE

Intraocular pressure (IOP) is the tension exerted by the contents of the globe on the corneoscleral envelope. Its importance in health is in maintaining the corneal curve and refractive index of the eye.

NORMAL VALUES

- 10–20 mmHg (may differ by 5 mm between eyes).
- Diurnal variation of up to 5 mmHg – higher in the morning.
- There is an increase in IOP with age.
- There is a positive correlation between IOP and axial length of the globe.

PHYSIOLOGY

- The factors that contribute to the IOP in the intact eye are the vitreous volume, the aqueous volume and the choroid plexus volume.
- The vitreous volume is usually constant.
- The aqueous volume is maintained by a balance between production and drainage.
- Aqueous is produced from the ciliary epithelium by a mixture of secretion and ultrafiltration.
- Aqueous drains through the trabecular network into the canal of Schlemm and the episcleral veins into the orbital venous system.
- The volume of the choroid plexus is maintained by autoregulation within physiological variations of systolic pressure.

PATHOLOGY

- Glaucoma is a condition caused by chronically elevated IOP, which results in progressive damage to the optic nerve (ischaemia and infarction) with sight-threatening visual field defects.
- This may be open-angle, when the trabecular meshwork becomes nonporous or closed-angle, when the iris obstructs the iridocorneal angle.

MEASUREMENT OF IOP

- The IOP is measured indirectly by applanation tonometry. The principle of this is that pressure on the cornea flattens a standard area, or the extent of the flattening is measured.
- A noncontact method is to use a puff of air to flatten the cornea.

FACTORS AFFECTING IOP

ARTERIAL PRESSURE

- Autoregulation in the retinal and choroidal circulations maintains a constant blood flow over a range of perfusion pressures.
- A sudden rise in arterial pressure may cause a transient rise in IOP but an increase in aqueous outflow and decrease in production will bring the IOP back to normal.
- A reduction in arterial pressure below 90 mmHg reduces the choroidal blood volume and the IOP. IOP will drop to zero at a systolic pressure of 50–60 mmHg.

VENOUS PRESSURE

- Changes in choroidal blood volume and IOP are more closely related to changes in venous than arterial pressure.
- Venous congestion will cause a rise in IOP due to an increase in choroidal blood volume and a decrease in episcleral vein drainage with consequent reduction in aqueous drainage.
- Venous pressure is increased by coughing, straining, vomiting, the Valsalva manoeuvre, obstructed airway, positive pressure ventilation and obstructed venous drainage of the head.
- Head-up tilt reduces venous pressure. A 15° head-up tilt reduces the IOP to the same extent as a reduction in $PaCO_2$ to 3.5–4.0 kPa.

GAS EXCHANGE

- A rise in PCO_2 due to hypoventilation results in dilatation of the choroidal blood vessels and a rise in IOP.
- A decrease in PCO_2 causes constriction of the choroidal vessels and a fall in IOP.
- Hypoxia may dilate intraocular vessels and increase IOP while hyperoxia may constrict vessels decreasing IOP. However, the changes seen during anaesthesia will have little effect on the IOP.

GENERAL ANAESTHESIA

- Physical interventions by the anaesthetist may raise the IOP.
- Pressure on the eye by the facemask will raise IOP.
- Laryngoscopy and intubation can markedly raise IOP by raising the systolic pressure. Various methods have been used to obtund this reflex. The use of propofol with an opiate such as alfentanil successfully reduces this rise.
- Insertion of a laryngeal mask causes a lesser rise in IOP.
- Extubation with coughing or gagging on the tracheal tube causes a rise in IOP.
- There is minimal coughing and straining in patients recovering with a laryngeal mask in situ.

ANAESTHETIC AGENTS

- All intravenous induction agents apart from ketamine will lower IOP.
- Propofol produces the most marked reduction in IOP.
- Ketamine does not lower the IOP and may increase it.
- Inhalation anaesthetics all lower the IOP if there is a normal or low PCO_2.

- Intravenous opioids and benzodiazepines will lower the IOP.
- The nondepolarizing muscle relaxants will reduce the IOP by reducing extraocular muscle tone.
- Suxamethonium will cause a rise in IOP that lasts up to about 10 min. However, if it is given following a sleep dose of propofol the IOP does not rise to above the resting value due to the reduction in IOP caused by the propofol.
- The effect of suxamethonium is due to contraction of the extraocular muscles.

SURGICAL INTERVENTIONS

- Chronic administration of drugs topically or systemically to lower IOP in chronic glaucoma. These work by affecting aqueous production or drainage, or by constriction of the pupil (Table 18.2).
- To treat acute glaucoma or lower IOP perioperatively, systemic agents such as 20% mannitol, 30% urea solution or oral glycerol may be used. These are osmotic diuretics and shrink the vitreous volume.
- The lid speculum and traction sutures may press on the eye during surgery and may raise the IOP.
- Intraocular gas bubbles such as sulphur hexafluoride (SF_6) will expand if the patient

is anaesthetised using nitrous oxide. The resultant rise in IOP may compromise perfusion of the optic nerve and retina.

LOCAL ANAESTHESIA

- In a peribulbar block, the volume of the local causes an intraorbital pressure increase; this leads to a transient rise in IOP.
- Sub-Tenon's block does not cause a significant increase in IOP but may cause inflammatory response in the conjunctiva and risks blocking the surgical drainage route.
- Ocular compression following a peribulbar block results in a soft eye which is preferred during glaucoma surgery.
- Once the local anaesthetic is effective, the anaesthetic has spread out in the tissues and the extraocular muscles are relaxed resulting in a lowering of IOP.

PROCEDURE TO TREAT GLAUCOMA (CHRONICALLY RAISED IOP)

SURGICAL TRABECULECTOMY

- A small hole is made through the sclera and into the anterior chamber drainage angle to allow aqueous humour to flow directly to the subconjunctival area, forming a drainage bleb.

Table 18.2 Drugs for treatment of chronic glaucoma

Drug	Method of action
Miotics	Constrict pupil and open up trabecular
• Carbachol	meshwork to increase drainage of aqueous.
• Pilocarpine	Given topically.
Beta-blockers	Reduce rate of aqueous formation. Given
• Timolol	topically.
• Betaxolol	
Carbonic anhydrase inhibitors	Reduce rate of aqueous formation.
• Acetazolamide (oral)	
• Dorxolamide (topical)	
Alpha$_2$-adrenoreceptor stimulant	Reduce rate of aqueous formation.
• Brimonidone (topical)	
Prostaglandin analogues	Increases uveoscleral outflow of aqueous.
• Latanoprost (topical)	

- Need to avoid exacerbating any pre-existing ischaemia of the optic nerve head; therefore, take care with the peribulbar blockade.
- Options are either general anaesthesia (as for cataract surgery; procedure duration 60–90 min), peribulbar block with care to avoid raised IOP or subconjunctival–intracameral local anaesthesia.

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CROSS-REFERENCES

Ophthalmic surgery – overview, Chapter 18
Artificial airways, Chapter 26
Total intravenous anaesthesia, Chapter 28

OPHTHALMIC TRAUMA

Ophthalmic trauma may result in a spectrum of injury from superficial laceration of the eyelid or cornea to complete disruption of the globe with extrusion of the contents. It may be impossible to assess the extent of the injury until the time of surgery. It may be associated with other injuries. There may be a foreign body present. It is one of the leading causes of monocular blindness.

PROCEDURE

- Exploration and anatomical repair in the first instance
- Removal of foreign body if present

PATIENT CHARACTERISTICS

- All age groups
- May have coexisting medical conditions
- Present as an emergency
- Trauma is likely to delay gastric emptying

PROBLEMS

- Open eye with full stomach situation
- Delay before surgery for penetrating eye injury may increase risk of loss of contents of globe and increase risk of infection
- Associated trauma, particularly of head and neck
- Danger of extrusion of globe contents at induction

PREOPERATIVE ASSESSMENT

- Assessment of associated injuries as resuscitation and urgent surgery may be required for non-ophthalmic problems
- If surgery is within 24 h of injury, treat as a full stomach
- Assess airway for rapid sequence induction
- Assess and optimise coexisting medical conditions if time allows

PREMEDICATION

- Sedation and antiemetics may be required especially in tearful children (caution with metoclopramide and risk of extrapyramidal effects in children/young adults)
- Local anaesthetic cream over venepuncture site in children
- Do not attempt to empty stomach preoperatively in children as crying, struggling or vomiting will increase intraocular pressure (IOP)

ANAESTHETIC MANAGEMENT

- Routine monitoring as per AAGBI guidelines

LOCAL ANAESTHESIA

- Local anaesthesia only appropriate in simple lid laceration without tear duct involvement.

GENERAL ANAESTHESIA

INDUCTION

- There is a conflict of possible full stomach and the need for rapid sequence induction with the need to protect the eye from a rise in IOP.
- Suxamethonium causes a transient rise in IOP.
- Laryngoscopy and intubation also produce a significant rise in IOP.
- *Modified rapid sequence technique*: may be used in experienced hands. Obtain intravenous access. Preoxygenate for 5 min without pressing on eye with facemask. Give alfentanil 0.02 mg/kg, propofol 3 mg/kg, rocuronium 0.6 mg/kg and apply cricoid pressure. Intubation can be performed at 60 seconds without coughing.
- *Standard rapid sequence technique*: intubation with suxamethonium, if inexperienced or anticipated difficult intubation. Use of propofol as induction agent (markedly lowers IOP) will protect against rise in IOP due to suxamethonium. Administration of lidocaine 1.0 mg/kg prior to induction will attenuate rise in IOP due to laryngoscopy and intubation.

MAINTENANCE

- Use balanced technique or TIVA.
- Careful monitoring of relaxation to ensure immobile patient.
- Antiemetic prophylaxis.
- Analgesia with NSAID or opiate.

EMERGENCE AND RECOVERY

- Antagonism of neuromuscular block
- Extubate awake

POSTOPERATIVE MANAGEMENT

- Antiemetics prescribed for postoperative nausea and vomiting.

- Moderate postoperative pain may require NSAID or opioid analgesia on first postoperative day.

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CROSS-REFERENCES

Emergency surgery, Chapter 25
Trauma, Chapter 30
Infants and children, Chapter 24
Airway and aspiration risk, Chapter 26
Ophthalmic surgery – overview, Chapter 18

ORBITAL AND OCULOPLASTIC SURGERY

Umbrella term for a variety of operations on the lids, orbit and lachrymal apparatus:

- Removal of tumours of the orbit
- Removal of the eye (with or without prosthesis insertion)
- Dacrocystorhinostomy (tear duct surgery)
- Ptosis and other eyelid surgery
- Eyelid cancers and reconstructions
- Decompression of the orbit in thyroid eye disease

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PATIENT CHARACTERISTICS

- Children with congenital tumours: retinoblastoma, dermoid cyst, orbital teratoma and capillary haemangioma
- Congenital ptosis may be associated with underlying muscle disease
- The elderly with eyelid skin cancers
- Associated medical conditions such as muscular dystrophies, thyroid disease

PROBLEMS

- The oculo-cardiac reflex commonly triggered during orbital manipulations.
- Preoperative counselling prior to eye removal.
- Risks of intra- and postoperative bleeding.
- Severe postoperative pain may be a sign of bleeding into the orbit.
- Decompression surgery is close to the cribriform plate, risk of CSF leak or cerebral damage.
- Following skin cancer resection under local anaesthesia (Mohs surgery), oculoplastic reconstruction cannot be postponed because patient has an open defect.

PREOPERATIVE ASSESSMENT

- Assess thyroid function prior to orbital decompression surgery.
- Check for undetected myasthenia gravis in patient with ptosis (Tensilon test).
- Assess fitness for anaesthesia in the elderly with skin cancers.
- Anaesthetists often consulted about safety of stopping aspirin prior to oculoplastic procedures to avoid periorbital haematomas.

PREMEDICATION

- Anxiolysis if required (e.g. temazepam 10–20 mg orally in adults, midazolam 0.5 mg/kg orally in children)
- Local anaesthetic cream to venepuncture site in children

- Anticholinergic prophylaxis for oculocardiac reflex is best given intravenously at induction

ANAESTHETIC MANAGEMENT

- Routine monitoring as per AAGBI guidelines

LOCAL ANAESTHETIC

- As many procedures involve skin or surface structures only can use local and regional techniques to provide adequate anaesthesia and analgesia.
- External DCR (dacrocystorhinostomy) can be performed under local anaesthesia with sedation.
- However many procedures are long (1–3 hours); therefore, continuous sedation ensures patient comfort with small doses of midazolam (0.5–1.0 mg) or low dose propofol TCI (target blood conc. 0.5–1 µg/mL).
- Local anaesthetic injections around the eye in the presence of sedation may result in vigorous sneezing (sternutatory reflex).
- Peribulbar blocks prior to removal of the eye often attenuate the oculocardiac reflex.

GENERAL ANAESTHESIA

INDUCTION

- Intravenous or inhalation as appropriate.
- Careful protection of the unaffected eye.
- Prophylaxis against oculocardiac reflex with glycopyrrolate 0.01 mg/kg intravenously at induction.
- Acetazolamide (500 mg IV) at induction for orbital decompression to reduce intraorbital pressure.
- Airway management – south-facing endotracheal tube and throat pack may be required where there is a risk of bleeding from the sinuses in orbital decompression and DCR.
- Personal experience is of a higher incidence of difficult intubation in patients requiring DCR for stenosed tear ducts.
- Dexamethasone (8 mg IV) often used in decompression and orbital tumour resection to reduce postoperative oedema.

MAINTENANCE

- Total intravenous anaesthesia (TIVA) using propofol and remifentanil infusions provides excellent conditions and reduces PONV.
- Alternatively, a balanced technique with fentanyl or remifentanil by infusion, muscle relaxant and volatile agent.
- Ventilation to normal ETCO$_2$.
- Maintain normotension or slight hypotension (traditional hypotensive anaesthesia is not indicated because this may mask orbital bleeding).
- Prophylactic antiemetic (ondansetron 0.01 mg/kg intravenously).
- Analgesia with paracetamol 15 mg/kg intravenously, diclofenac 2 mg/kg (latter for eye removal surgery only; contraindicated where there is a risk of orbital haemorrhage).
- Peribulbar block at the end of surgery to reduce postoperative pain following eye removal and implant surgery.

EMERGENCE AND RECOVERY

- Antagonise neuromuscular block with neostigmine and glycopyrrolate.
- Extubate/remove laryngeal mask when awake.
- Try to avoid coughing with intravenous lidocaine 0.25 mg/kg.
- If using a laryngeal mask, do not apply head bandage until LMA removed because of risk of displacement and airway obstruction.
- Following enucleation of the orbit, may require intravenous opioids in recovery and long-acting oral opioids for 1–2 days.
- The oculoemetic reflex following orbital surgery may result in prolonged PONV, anticipate with multimodal antiemetic therapy.

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Hyperthyroidism, Chapter 6
Myasthenia gravis, Chapter 3
Muscular dystrophies, Chapter 3
Ophthalmic surgery – overview, Chapter 16
Infants and children, Chapter 24

STRABISMUS CORRECTION

Misalignment of the visual axes may result in double vision (diplopia), loss of visual acuity (amblyopia) and loss of binocular vision. This occurs in about 5% of children and treatment usually requires surgical correction. Adults may require strabismus surgery due to thyroid eye disease, trauma, sixth cranial nerve palsy and following strabismus surgery as a child.

PROCEDURE

- Surgery takes 30–90 min and is routinely managed as day case.

RECESSION

- Weakening an extraocular muscle by moving its insertion on the globe.

RESECTION

- Strengthening an extraocular muscle by removing a short piece of tendon or muscle.

ADJUSTABLE SUTURES

- Used in some adult patients to allow final adjustments to be made with eye movements postoperatively.

PATIENT CHARACTERISTICS

- The most common ophthalmic operation carried out in children.
- Infantile strabismus needs early surgery (6–12 months) for best visual outcome.
- Rare association with primary muscle diseases and malignant hyperthermia.
- Adult patients may have associated medical conditions such as connective tissue disease, thyroid eye disease and amyloidosis.
- Associated rare conditions: Stickler syndrome, craniosynostosis, Mobius syndrome, incontinentia pigmenti.

PROBLEMS

- Airway not accessible because of microscope position.
- Avoid suxamethonium.
  - Can trigger malignant hyperthermia in susceptible patients.
  - Can cause tonic contracture of extraocular muscles. This interferes with forced-duction test.
- Oculo-cardiac reflex
- Topical adrenaline often used to reduce bleeding and may be absorbed systemically – watch dose in small children.
- High incidence of postoperative nausea and vomiting (PONV).

PREOPERATIVE ASSESSMENT

- Identification, investigation and optimisation of associated syndromes or coexisting medical conditions.
- Assess suitability foy-case surgery.

PREMEDICATION

- Anxiolytics if required (e.g. temazepam 10–20 mg orally in adults, midazolam 0.5 mg/kg orally in children).
- Local anaesthetic cream to venepuncture site in children.

- Anticholinergic prophylaxis for oculocardiac reflex is best given intravenously at induction.

ANAESTHETIC MANAGEMENT

- Routine monitoring as per AAGBI guidelines.

LOCAL ANAESTHETIC

- May be used in cooperative adult patients for uncomplicated surgery.
- Topical anaesthesia with 1.0% amethocaine allows optimal adjustment of muscle sutures at the time of surgery. The oculocardiac reflex is not blocked and ECG monitoring and possible use of anticholinergics is required.
- Sub-Tenon's or peribulbar blocks (as for cataract surgery) are suitable and provide some protection from the oculocardiac reflex.

GENERAL ANAESTHESIA

INDUCTION

- Intravenous or inhalation as appropriate.
- Prophylaxis against oculocardiac reflex with glycopyrrolate 0.01 mg/kg intravenously at induction.
- Airway management – reinforced laryngeal mask unless contraindicated by patient factors.

MAINTENANCE

- Total intravenous anaesthesia (TIVA) using propofol and remifentanil infusions provides excellent conditions and reduces PONV.
- Nondepolarising relaxant (e.g. atracurium or rocuronium) if intubation required. A small dose (half intubating dose) required for maintenance with TIVA to ensure a central immobile eye.
- Alternatively, a balanced technique with fentanyl or remifentanil by infusion, muscle relaxant and volatile agent.
- Ventilation to normal $ETCO_2$.
- Prophylactic antiemetic (ondansetron 0.01 mg/kg intravenously).

- Analgesia with diclofenac 2 mg/kg and/or paracetamol 15 mg/kg intravenously.
- 1–2 mL 0.5% bupivacine sub-Tenon's at the end of surgery gives good postoperative analgesia if adjustable sutures not used.
- Alternatively, codeine phosphate 1 mg/kg oral or intramuscularly for more extensive bilateral surgery.

EMERGENCE AND RECOVERY

- Reverse neuromuscular block with neostigmine and glycopyrrolate.
- Extubate/remove laryngeal mask when awake.

POSTOPERATIVE MANAGEMENT

- Topical amethocaine at the end of surgery for immediate postoperative pain.
- Moderate postoperative pain on first day, oral paracetamol usually sufficient.
- Antiemetics prescribed, but PONV may be delayed until after discharge.

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CROSS-REFERENCES

VITREORETINAL SURGERY

Normal vision requires transparency of the vitreous body and integrity of the retinal layers. These may become disrupted by disease processes and by trauma, resulting in the separation of the photosensitive layer of the retina from the pigment epithelium with visual loss. Diabetes mellitus, myopia and increasing age pre-dispose to retinal detachment. In type 1 diabetes and sickle cell anaemia, abnormal blood vessel growth on the retina can cause vitreous haemorrhages.

PROCEDURE

- Two types of operation:
  - External approach (cryo-buckle procedure): location of retinal holes and treating them externally with cryotherapy whilst observing with indirect ophthalmoscope. Traction sutures placed around the recti muscles may stimulate the oculocardiac reflex when pulled up. A silicone sponge or solid explants may be sutured to the globe.
  - Internal approach (vitrectomy): three tiny holes are made in the sclera so that instruments can enter the eye; the retina is then treated directly with electrocautery and laser coagulation. Often agents are used to tamponade the retina such as air, air-gas mixtures (SF_6 or C_3F_8) and silicone oils.
- V-R surgery is urgent when the macula is still attached at presentation (macula-on) and in whom subretinal fluid is likely to rapidly extend. Once the macula has detached, the procedure can be done within 7 days.
- Retinal detachment is frequently associated with areas of weakness in the contralateral eye that could predispose it to the same condition; therefore, laser treatment may be required.

PATIENT CHARACTERISTICS

- Children with retinopathy of prematurity
- Adult patients may have associated medical conditions, diabetes mellitus, Marfan syndrome
- Other associated conditions: SLE, sickle cell disease, Stickler syndrome.

PROBLEMS

- Remote airway and limited access to patient due to vitrectomy equipment
- Operating theatre in darkness for much of the surgery
- Surgery can be lengthy
- Oculocardiac reflex
- Midricaine (procaine, atropine and adrenaline mixture for prolonged pupil dilatation) subconjunctival injection may cause cardiovascular effects
- Laser frequently used and anaesthetist has to wear goggles

PREOPERATIVE ASSESSMENT

- Assessment and optimisation of associated medical conditions

PREMEDICATION

- Perioperative management of diabetes if required
- Anxiolysis with oral temazepam 10–20 mg if required

ANAESTHETIC MANAGEMENT

- Routine monitoring as per AAGBI guidelines
- Audio alarms and anaesthetic machine illumination important in a dark theatre

LOCAL ANAESTHESIA

- Suitable in cooperative adults for simple detachment and vitreous surgery or in the medically compromised.
- Sub-Tenon's anaesthesia or two injection peribulbar block (as for corneal graft surgery) provide good operating conditions.

- If the procedure is lengthy, sub-Tenon's top up can readily be given by the surgeon.
- Patient comfort important during lengthy surgery and sedation with small doses of midazolam (0.5–1.0 mg) or low dose propofol TCI (target blood conc. 0.5–1 µg/mL) can be considered.
- With sedation, oxygen supplementation and careful monitoring are vital as there is limited access to patient.

GENERAL ANAESTHESIA

- General anaesthesia is the preferred technique for prolonged and complicated surgery.

INDUCTION

- Total intravenous anaesthesia (TIVA) with target controlled infusion (TCI) of propofol and a remifentanil infusion provides excellent operating conditions and rapid and smooth emergence from lengthy anaesthesia, with greatly reduced postoperative nausea and vomiting.
- Alternatively, a balanced technique using intravenous induction with propofol, fentanyl boluses or remifentanil infusion, muscle relaxant and volatile agent is suitable.

AIRWAY MANAGEMENT

- A secure airway with either a reinforced laryngeal mask or south facing endotracheal tube.

MAINTENANCE

- Muscle relaxation (e.g. atracurium or rocuronium). Monitor carefully to avoid sudden movement of patient during surgery. A remifentanil infusion may also avoid sudden movement and take the place of continuous muscle relaxation.
- Ventilate with oxygen in air and avoid nitrous oxide (nitrous oxide will expand a gas bubble and can result in a dangerous rise in intraocular pressure in the closed eye).

- Careful positioning and padding of patient.
- Consider active warming and intravenous fluids for prolonged procedures or in at-risk patients.
- Note that cryotherapy may be particularly stimulating.
- Consider prophylaxis against the oculocardiac reflex with glycopyrrolate 0.01 mg/kg intravenously (note: local anaesthetic eye block usually attenuates this reflex).
- Antiemetic prophylaxis with ondansetron 4 mg intravenously.
- Sub-Tenon's bupivacaine by surgeon provides intraoperative and postoperative analgesia.

EMERGENCE AND RECOVERY

- Antagonise neuromuscular block with neostigmine and glycopyrrolate.
- Extubate awake, coughing is usually minimal.

POSTOPERATIVE MANAGEMENT

- Regular paracetamol, NSAIDs, PRN codeine (max 240 mg in 24 hours). Intravenous opioids may be required in recovery. Local anaesthesia as part of the general anaesthetic technique can improve patient comfort.
- PONV can be a problem especially after buckling procedures (local anaesthetic block usually reduces this).
- Some patients need to posture (prone) postoperatively to ensure the gas bubble is in the best position to close the retinal break; this may be for 5–10 days.

- Over the following weeks where a gas bubble is present, the patient should be warned about:
  - Avoiding nitrous oxide in general anaesthesia
  - Atmospheric pressure changes during aircraft flights

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ENT surgery

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ROSS MACNAB, KATHERINE BEXON,
SOFIA CLEGG AND ADEL HUTCHINSON

ANAESTHESIA FOR ENT SURGERY: GENERAL PRINCIPLES

A whole spectrum of patients, from the very young to the old, ASA 1 to ASA 4, can present for ENT surgery.

PREOPERATIVE

- Detailed history and examination (especially airway assessment and relevant pathology).
- Assessment of comorbidities, previous anaesthetic history.
- Smoking and alcohol consumption.
- Screening for obstructive sleep apnoea.
- Relevant and targeted investigations.
- Preoperative endoscopic airway examination.
- Review of airway imaging: CT, MRI scans.
- Preoptimisation if possible, e.g. nutritional status, respiratory system.

PREMEDICATION

- Avoid sedative medication if there is any suggestion of airway compromise.
- Consider gastric acid prophylaxis.

PERIOPERATIVE MANAGEMENT

- Skilled multidisciplinary team
- Team brief and WHO checklist
- Establish IV access
- Airway equipment
  - Laryngoscopes with a variety of blades
  - Video laryngoscopes, fibreoptic scopes, optical stylets (Bonfils).
  - Variety of ET tubes standard, preformed, reinforced, MLT, laser tubes.
  - Supraglottic airway eevices.
  - Bougies, stylets, exchange catheters and other airway adjuncts.
  - Jet ventilation equipment.

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- Cricothryroidotomy/tracheostomy equipment.
- Equipment for LA topicalisation of airway.
- Difficult airway trolley.
- Preoxygenation
  - Face mask
  - Nasal oxygenation during efforts securing a tube
  - THRIVE (Transnasal Humidified Rapid-Insufflation Ventilatory Exchange)
- Monitoring:
  - Standard monitoring
  - Neuromuscular monitoring
  - Core temperature
  - Invasive monitoring may be required depending on comorbities, injuries; extent, complexity and duration of surgery
- Induction:
  - IV or inhalational induction or awake intubation
  - Titrate to patient response; rapid sequence if warranted
  - Rocuronium is a good choice, with sugammadex available to reverse if required
  - Sevoflurane or desflurane
  - Remifentanil, alfentanil, fentanyl
  - Steroids for swelling and antiemesis
  - Antibiotics

SAFE USE OF A THROAT PACK

A throat pack is used to prevent blood and debris contaminating the airway or being swallowed. Throat packs are often inserted to:

- Absorb material created by surgery in the mouth
- Prevent fluids or material from entering the oesophagus or lungs
- Prevent escape of gases from around tracheal tubes
- To stabilise artificial airways

If a throat pack is retained after surgery it can lead to obstruction of the airway and result in significant morbidity or mortality. It is classed as a 'never event' by NHS England.

The National Patient Safety Agency (NPSA) safer practice alert NPSA/2009/SPN001-reducing the risk of retained throat packs after surgery 28th April 2009 recommends:

- Label or mark patient either on the head or, exceptionally, on another visible part of the body with an adherent sticker or marker.
- Label artificial airway (e.g. tracheal tube or supraglottic airway).
- Attach pack securely to the artificial airway.
- Leave part of pack protruding.
- Formalised and recorded 'two-person' check of insertion and removal of pack must be performed and insertion and removal recorded on theatre swab board.

MAINTENANCE

- Oxygen and air with inhalational agent or TIVA.
- Remifentanil infusion.
- Head-up position and relative hypotension can reduce bleeding and aid surgical field. Careful neck extension to improve access to neck and glottis.

EXTUBATION

- Throat pack *must* be removed and confirmed by team members at sign out.
- Airway clear of secretions and blood.
- Assessment of airway oedema: presence of leak around tube when cuff is down is suggestive that airway is not overly oedematous.
- Plan for extubation using the DAS guidelines:
  - Stratify to low or high risk
  - Awake/deep extubation, advanced extubation techniques or delay extubation/ surgical airway.

POSTOPERATIVELY

- Depending on patient and surgery, return to day-case facility, head and neck ward, HDU or ICU.
- Analgesia is not a particular problem: use a multimodal approach with LA, paracetamol, NSAIDs and opiates.
- Antiemetics.

COMPLICATIONS

Important complications relate to airway compromise. A level of high vigilance must be maintained by all staff.

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Difficult airway prediction, Chapter 26

LARYNGOSCOPY AND MICROSURGERY OF THE LARYNX

KATHERINE BEXON

The laryngeal inlet is the narrowest part of the upper airway. For safe management, the anaesthetist and surgeon must share responsibility. The anaesthetist needs to understand the nature and types of pathologies commonly encountered and the surgical operating requirements. Surgical assessment can provide valuable information about altered anatomy and assist in planning airway management.

PATIENT CHARACTERISTICS

- Usually older with significant comorbidities.
- Squamous cell carcinoma of the larynx is associated with longstanding cigarette and alcohol consumption and disabling cardiorespiratory and hepatic disease.
- Cigarette consumption causes airway hyper-reactivity.

PREOPERATIVE ASSESSMENT

- Routine review of associated medical conditions.
- Clinical evaluation of respiratory distress and of the presence of stridor.
- ENT appraisal of the airway by indirect laryngoscopy and/or fibreoptic nasendoscopy.
- Appropriate investigations determined by the history and examination.
- Radiological imaging (CT or MRI) to determine the extent and nature of the pathology.

PREMEDICATION

- Warn patient possible temporary breathing difficulty postoperatively.
- Not usually necessary (may compromise airway narrowing).
- Antisialogogues decrease secretions but make secretions thicker.
- Patients with stridor require oxygen during transport. Heliox can mitigate symptoms by improving gas flow.

THEATRE PREPARATION AND MONITORING

- Routine minimal monitoring with neuromuscular monitoring where appropriate.
- Transnasal humidified rapid-insufflation ventilatory exchange (THRIVE) can be useful for preoxygenation and for postoxygenation until a definitive airway has been established.
- A difficult intubation trolley may be required and should be available.

- Patient position is with a head-ring ± sandbag beneath the shoulders, with due care as excessive neck extension may cause injury.

ANAESTHETIC TECHNIQUE

The choice of anaesthetic technique depends on the condition of the patient at the time of surgery and the individual preferences and expertise of the anaesthetist.

A main consideration is sharing the airway with the surgeon. Prevention of hypoxic brain damage is more difficult when the airway is abnormal. Induction of general anaesthesia in a patient with a compromised airway may result in total obstruction. Generally supraglottic lesions block the view of the laryngeal inlet, glottic lesions narrow it and subglottic lesions cause narrowing beyond the view during direct laryngoscopy. Neoplastic lesions can be friable and prone to bleeding during instrumentation, obscuring the view further. The anaesthetist must predict and plan for the likely condition of the airway at recovery and anticipate any continued bleeding.

- Tracheal intubation is usually required.
- Muscle relaxants may not be needed for tracheal intubation and are usually only administered when assisted ventilation and/or intubation are guaranteed.
- Awake fibreoptic intubation is an option.
- Surgery on the vocal cords (for biopsies or cord stripping) usually requires the patient to be fully relaxed but assessment of vocal cord movements may need to be considered at some stage.
- Manipulation of the larynx can cause a hypertensive response and cardiac arrhythmias and may result in laryngeal oedema with implications for the end of the procedure.
- A rapid return of consciousness and protective reflexes is important.
- Aspiration of blood and surgical debris is possible.
- In severely narrowed airways an urgent tracheostomy performed under local anaesthesia may be the best option but this can be difficult in a hypoxic, distressed, uncooperative patient.
- Cardiovascular stability, avoidance of awareness and a smooth, rapid recovery are essential.
- An intravenous induction is suitable for most patients followed by maintenance with oxygen, nitrous oxide and an inhalational agent ± opioid.
- Total intravenous anaesthesia (TIVA) is a useful alternative and can avoid the use of muscle relaxants. Hypotension can be a problem in elderly patients.

AIRWAY MAINTENANCE

Tracheal intubation is the standard airway management. A microlaryngoscopy tube is a cuffed tube of normal length for adults but of small diameter creating high resistance and sometimes difficulty ventilating obese patients. Sizes 4.0–6.0 mm I.D. are available.

The need for optimal access to the vocal cords has led to the following alternative methods.

- *Tubeless anaesthesia*: there is no tracheal tube between the vocal cords. One method uses intubation lower down the airway, e.g. cricothyroid puncture (usually with jet ventilation). Another relies on the patient breathing spontaneously with local anaesthetic spray to the vocal cords allowing a lighter plane of anaesthesia. Close cooperation between surgeon and anaesthetist is essential.
- *Venturi jet ventilation*: injected driving gas entrains room air. Injectors typically use oxygen as the driving gas and are firmly attached to a rigid bronchoscope to limit backlash. Ventilation is most effective when the bronchoscope tip is well aligned with the trachea. There is a risk of barotrauma.
- *Tracheal catheter*: inserted through the cords down to the carina to deliver anaesthetic gases. These increase aspiration risk and can move with the driving gas and cause soft tissue injury.
- *High frequency oscillation*: uses smaller tidal perturbation at higher rates (60–100/min).

It requires specialized ventilators usually via a catheter below the vocal cords. The risk of barotrauma is less than with venturi ventilation.

It is important to always consider maintenance of a clear conduit for inspiration and expiration and to be aware that routine ventilatory monitors, e.g. tidal volume, airway pressures and $ETCO_2$ are inaccurate. Hypoventilation and awareness is a risk, particularly in patients who are obese or have poor lung compliance. Anaesthetic inexperience can contribute to this and TIVA may help provide depth of anaesthesia.

LASER SURGERY

Lasers are used for excision of various laryngeal lesions. They allow precise incisions with an extremely fine zone of coagulation leading to less postoperative bleeding, minimal tissue reaction and reduced postoperative oedema. Carbon dioxide (the most common) or Nd-YAG lasers are available for ENT surgery. Safety training for theatre staff is mandatory and local policies exist to prevent direct and incidental burns. The use of laser safety glasses is mandatory.

FIRE PRECAUTIONS AND TREATING A FIRE

Specially designed, single use disposable laser-resistant tracheal tubes with double cuffs filled with saline are recommended. The surgeon applies saline-soaked gauze pads to further protect the cuffs and surrounding soft tissue. Wet towel drapes are used around the surgical field. Traditionally it has been suggested that the inspired oxygen concentration should be less than 30% before use of the laser. More recently it has been proposed that the expired oxygen concentration should be less than 30% as the majority of the respiratory cycle is spent in expiration. If tubeless anaesthesia is used, there may be inadvertent laser damage to tissues lower down the airway. Utilising low energy lasers in intermittent rather than continuous stimulation modes should further minimise the risk of fire.

If fire does occur, the first priority is to limit injury to the patient. It has been suggested that extubating the patient without disconnecting the fresh gas flow may be preferable to disconnecting the breathing circuit then removing the tube as the FRC acts as a combustible reservoir of oxygen. Saline flushing with a syringe can be used to cool tissues and limit secondary damage. The original tracheal tube will need to be replaced; metallic tubes are available and should be readily on hand. Reactive oedema occurs rapidly. Oxygen administration is resumed as soon as this is considered safe. The patient should be treated as having an inhalational burn injury and transferred to ICU.

POSTOPERATIVE COMPLICATIONS

- *Immediate and early*: transient respiratory distress, sore throat, pain, hoarseness, laryngospasm, bleeding, stridor, laryngeal oedema, aspiration of blood and surgical debris. Early discharge should only be allowed for carefully selected patients where risks are considered minimal.
- *Late*: tissue scarring and progression of the original pathology. Where radiation is used in the treatment of malignancy there is increased risk of reactive laryngeal oedema.

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Awareness, Chapter 30

MAJOR RECONSTRUCTIVE SURGERY

SOFIA CLEGG

Reconstructive procedures are conducted to repair defects or correct deformities caused by:

- Cancer surgery of the head and neck
- Congenital cranio/maxillofacial deformities
- Acquired facial deformities
- Secondary to radiotherapy treatment

The operations can be lengthy, lasting up to 18 hours with free flap reconstruction. The patients range from children (potentially with associated congenital cardiac defects) through young, healthy adults to elderly, frail patients with an array of medical comorbidities. Major reconstructive surgery involves a multidisciplinary team, starting from diagnosis until long after the patient has left the hospital. The team involves anaesthetists, surgeons, specialist nurses, speech and language therapists, physiotherapists, dieticians and oncologists.

Single stage reconstructive surgery with pedicled or microvascular free flap (tissue ± bone) is now commonplace. The need for intermaxillary and complex external fixation has decreased following mandibular reconstruction with titanium plates and internal fixation.

Any form of airway surgery can be a challenge. Not only can the patient's airway be difficult to manage, but we also need to share this space with the surgeons. Clear communication is vital. The head of the patient is often the other side of the theatre to the anaesthetic machine (remote anaesthesia) and the surgical drapes will make diagnosing a disconnection, occlusion or misplaced ETT and bleeding more difficult.

PATIENT FACTORS

Patients often have a long history of heavy cigarette smoking and alcohol consumption. There is a high rate of coronary artery disease, respiratory disease and peripheral vascular disease. The patient may be malnourished, either as a secondary consequence of the tumour, chronic alcohol consumption or previous chemoradiotherapy. The preoperative clinic is the time to fully discuss potential risks and complications in these high-risk patients and should involve the full multidisciplinary team. Patients are at high risk of postoperative cognitive dysfunction (POCD) and delirium especially those that drink more than 5 units of alcohol per day.

Patients presenting for craniofacial deformities can range from young children with associated syndromes and congenital defects to healthy young adults. The former group requires specialist attention and is beyond the scope of this chapter.

PREOPERATIVE ASSESSMENT

AIRWAY

A thorough preoperative assessment is vital, in particular a comprehensive understanding of the pathology and any previous procedures. Previous surgery may make the airway more or less challenging. History and examination is imperative and importantly a review of any recent anaesthetics. Airways can change quickly, tumours can grow slowly or very fast, airways can become oedematous and tumours friable. Radiotherapy oedema can last up to six months.

Look for potential problems with bag-mask ventilation or difficulties with laryngoscopy or both. Include in the history any symptoms of dysphagia, dysphonia, stridor, difficulty lying flat, snoring, OSA and pain. Examination should include a standard airway assessment but also palpate external tissues of the head and neck exposed to radiotherapy or previous surgery (can be rigid or 'woody'). Locate the cricothyroid membrane and closely observe neck movements, mouth opening and tongue mobility. Radiotherapy exposed tissue often feels solid and will therefore not be malleable after induction, the cricothyroid membrane can often feel indistinct. Nasendoscopy may be beneficial depending on the pathology.

Airway management can be split into three sections – bag mask ventilation, visualising the glottis and passing the endotracheal tube. It is important to review the CT/MRI scan but to be wary that the scan could be 4–6 weeks old.

Indicators of difficult bag mask ventilation:

- Previous radiotherapy ± surgery
- Edentulous
- Beard
- Obese
- Facial deformity or asymmetry
- Poor neck extension
- OSA
- Tumour location and size

Indicators of difficult laryngoscopy:

- Size and site of tumour
- Friable tumour
- Previous radiotherapy ± surgery
- Prominent teeth
- Poor mouth opening <3 cm interdental distance
- Stridor
- Change in voice
- Micrognathia/retrognathia
- Calder C
- Mallampati score
- Poor neck extension

Indicators of difficulty passing the endotracheal tube:

- Tight glottic stenosis (e.g. obstructive laryngeal tumours)
- Subglottic stenosis
- Anterior larynx
- Poor view
- Blood, secretions or phlegm
- Large tumour above the glottic opening (hypopharyngeal lesions or tongue base tumours)

INVESTIGATIONS

- FBC, clotting, blood group and cross-match
- U&Es, LFTs, bone profile, blood glucose
- Auscultation, ECG

ADDITIONAL INVESTIGATIONS AS INDICATED

- CXR, pulmonary function tests, echocardiogram, arterial blood gas
- CPET, dynamic cardiac investigations

ASSESSMENT OF RISK

Major head and neck surgery is intermediate risk surgery. POSSUM has been shown useful at identifying morbidity but not mortality in these patients. The Revised Lee Cardiac Risk Index is effective at identifying the risk of a major cardiac event. The usefulness of CPET is still being evaluated.

GENERIC FACTORS

Patients should be encouraged to stop smoking. Smokers have an increased risk of intra and postoperative complications. Smoking reduces oxygen delivery to flaps and causes hypercoaguability. As part of an Enhanced Recovery Program appropriate patients should be advised about breathing exercises and given incentive spirometry. A comprehensive assessment of alcohol consumption should elicit those at risk of withdrawal. These patients should be admitted before their surgery for a period of detoxification. Dietician involvement will help optimise the patient's nutritional status and anaemia should be highlighted and treated appropriately.

PERIOPERATIVE MANAGEMENT

Premedication is not routine. It should be avoided if the airway is compromised. Normal medications should be continued, particularly cardiac medications (with the exception of ACEi and ARBs) inhalers, nebulisers and analgesia. An anti-sialogogue may be appropriate if performing a fibre-optic intubation.

AIRWAY MANAGEMENT

Appropriately skilled personnel should manage these cases. Excellent communication with the surgeon and all staff in the theatre suite is a necessity. A comprehensive plan for airway management should be in place and discussed prior to the patient's arrival. This should involve the surgical requirement for type of endotracheal tube. This can vary from oral to nasal to submental or tracheostomy. A decision should be taken about the most appropriate place to anaesthetise the patient – in the anaesthetic room or theatre. Tracheal intubation can be secured by:

- Induction with IV agent and relaxant.
- Gaseous induction (mainstay in paediatric anaesthesia, not adult airway cases).
- Awake intubation using local anaesthesia and/ or remifentanil. Intubation is performed using a fibre-optic scope or videolaryngoscope.
- Tracheostomy under LA.

Preoxygenation with nasal cannula is popular or use THRIVE. The aim is to increase the apnoeic time in patients that may be awkward to intubate. This helps to reduce the time pressure in securing the airway and therefore alters the human factors in theatre that can affect the smooth running of challenging situations. This should not be used instead of a thorough airway assessment or in a patient that would otherwise require an awake intubation or tracheostomy.

For any anaesthetic plan, alternatives should have been considered. In these high-risk surgical patients the whole team should be aware. Should Plan A fail, then pre-preparation of the subsequent plans will lead to a calm situation.

MONITORING

- AAGBI minimal standards.
- Invasive arterial monitoring – be aware of the location of free flap donor site.
- CVP (less frequently used these days) – discuss with the surgeon but femoral or subclavian veins may be the only suitable ones. Alternatively long lines can be used.
- Urinary catheter – often with temperature probe incorporated.
- Core and peripheral temperature for free flap surgery. Aim for <1.5°C difference.
- BIS.
- Arterial blood gases.
- Blood loss and fluid balance.
- Cardiac output monitor for goal directed fluid therapy.

INTRAOPERATIVE

The aim is to maintain a normothermic, normo-volaemic patient with an adequate perfusion pressure. Anaesthesia is commonly maintained with remifentanil and a volatile agent. TIVA can be used but the time to extubation may be slower after a prolonged infusion of propofol. In young healthy patients, a period of hypotension during the part of the operation when blood loss can be profound will help reduce this. This is usually achieved with anaesthetic agents; vasodilators are not required.

Local antibiotic guidelines should be adhered to. Dexamethasone helps to reduce tissue swelling and acts as an antiemetic. Exceptional care needs to be taken positioning the patient, padding susceptible points to pressure and damage. Ensure that wires, catheters, connectors and ruffled sheets are not causing marks and compression on the skin. Intermittent passive movement of joints and areas prone to pressure will help reduce painful joints and pressures sores. The eyes need lubrication and protection and nasal endotracheal tubes should not put pressure on the nostrils.

Venousthromboembolic assessment and guidelines should be followed. Patients will require lower limb pneumatic inflation devices. Compression stockings should not be used in patients with peripheral arterial disease.

Patient positioning should be 10–15° head-up tilt to minimise venous congestion. Flexion of the thighs at the hip improves venous return.

Fluid replacement should aim for euvolaemia. Blood loss is often concealed and the full extent only apparent when the drapes are removed. Depending on the surgery, blood loss can either be slow and steady or rapid at specific points, e.g. during maxillary down fracture.

Appropriate analgesia will prevent an agitated patient on extubation and catecholamine release, which is detrimental to a free flap. Analgesia will usually consist of an opiate, paracetamol ± NSAID.

FREE FLAP SURGERY

Good surgical technique remains a leading factor for graft survival. Optimum anaesthetic management should prevent vasoconstriction and enhance blood flow to the flap. Free flap surgery can be split into three sections:

1. Tumour resection and flap harvest. Main time for blood loss.

2. Disconnected ischaemic flap. This time should be minimised.

3. Flap reperfused at recipient site. Risk of secondary ischaemia.

The free flap is denervated after it is harvested. It loses intrinsic sympathetic tone but the feeding artery and draining vein at the recipient site will still respond appropriately to physical, humoral and chemical stimuli. The flap has no lymphatic drainage and is at increased risk of oedema.

Aim for a warm, normovolaemic patient with a low systemic vascular resistance. Blood haematocrit should be in the range of 0.25–0.3 to provide adequate oxygen delivery whilst reducing viscosity.

Aggressive fluid management may trigger anastomosis failure leading to increased flap complications. Most units in the UK performing free flap reconstructive surgery aim for a fluid balance between neutral and 2 L positive at the end of surgery.

Intraoperative vasopressors have no significant effect on flap failure.

EXTUBATION

Extubation can be as challenging as intubation but is often less well thought about. At the WHO checklist a plan should be prepared for extubation and postoperative care. This may change during the course of the operation. The Difficult Airway Society has extubation guidelines to help formalise a plan.

The aims are to provide a safe airway with minimal coughing and straining and the back up to secure the airway should extubation fail.

A decision needs to be made whether a tracheostomy is required or if the patient should stay ventilated overnight and extubated at a later time. These are difficult decisions that need to be made between surgeon and anaesthetist. Factors that play a role include the surgery itself, the likelihood of oedema, bleeding and swelling, comorbidities and the personnel available to care for the patient.

If extubation is planned, then this should be carried out in theatre with appropriate theatre staff readily available. To smooth the extubation, low dose remifentanil can be continued to provide an awake, cooperative patient with intact airway reflexes. Conversion to a supraglottic airway device, to prevent coughing,

is not usually appropriate after major reconstructive surgery. If there are any concerns about the ability to reintubate the patient, then extubation over an airway exchange catheter is a useful tool.

POSTOPERATIVE CARE

An HDU or a specialist head and neck ward with suitable staffing levels and skills are recommended. Analgesia is maintained with opiates (PCA or oral), paracetamol ± NSAIDs. Location of surgery or donor flap site may be amenable to nerve block or local infiltration. Dexamethasone will help minimise swelling. Patients should be nursed in a head up position to improve respiratory dynamics and venous drainage. VTE prophylaxis needs to be continued and early mobilisation encouraged.

Nutrition is important. Most patients will have a nasogastric tube or PEG in-situ to commence enteral feeding on the first postoperative day.

Continue warming the patient. Avoid shivering, particularly in the patient with free flap reconstruction. Shivering increases oxygen demand which in turn increases catecholamines release, peripheral vasoconstriction leading to a profound decrease in blood flow to the transplanted tissue. Despite appropriate treatment, blood flow may not return to normal to the free flap for over an hour. Avoid any straps, ties or compressive dressings over the neck when a free flap is in-situ as these can reduce the blood flow through the new flap.

Patients sometimes have to return to theatre due to airway difficulties, bleeding, compromised blood supply to flaps and haematoma formation.

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MIDDLE EAR SURGERY

Surgery to the external and middle ear structures tends to be elective for improvement of patient quality of life whether through restoration of hearing, decrement of infection or improvement of cosmetic defects. Patients can be of any age.

PROCEDURES

Careful dissection of small structures is involved such as ossicles, using an operating microscope. The surgical field must be as free of blood as possible. A small amount of blood can obscure the surgeon's view through the microscope. Injury to the facial nerve is possible with an incidence of 0.5%–3.5%.

PERIOPERATIVE MANAGEMENT

- Detailed assessment and investigations specific for the individual patient.
- Premedication if required.

MONITORING

- Standard minimum monitoring.
- Neuromuscular monitoring.
- Invasive blood pressure monitoring if hypotensive anaesthesia is requested in certain patients.

POSITIONING

- Head and neck will be rotated to the opposite side from the operative field. Avoid hyperextension of the neck to minimise chance of brachial plexus injury.
- Extreme lateral head movement can be avoided by using lateral tilt of the operating table.
- The dependent ear and eye should be free of excessive pressure especially in long cases, with use of a head ring.
- Head-up tilt of 10–15° helps to minimize bleeding.

AIRWAY

- Endotracheal tube is commonly used.
- Laryngeal mask may suffice but careful seating and seal must be confirmed with the head turned to the operative site.
- Long anesthesia circuit tubing is required.
- Recheck all connections and that gas exchange and ventilation are optimal in the final surgical position prior to draping.

MAINTENANCE

- TIVA using propofol.
- Inhalational agent.
- Remifentanil.
- Protect eyes.

SURGICAL FIELD

- Bleeding must be minimised. The severity of bleeding is related as much to venous as to arterial blood pressure.
- A clear airway is essential. A partially obstructed airway impedes expiration, increases CO_2 levels and raises venous pressure. An armoured endotracheal tube avoids kinking.
- Anaesthesia should be smooth. Avoid straining and coughing as they increase venous pressure and bleeding.
- Avoid tachycardia. Beta-blockers are useful – small doses of labetalol, esmolol or metoprolol can be titrated intravenously.
- Induced hypotension may be requested. Profound hypotension is unnecessary and may be harmful.

FACIAL NERVE MONITORING

- Intubate with small amount of relaxant and allow to wear off or reverse.
- Use nerve stimulator to monitor block.
- Alternatively intubate with opiates and LA spray to cords.
- Use a laryngeal mask.

NITROUS OXIDE

The use of N_2O is controversial. Its accumulation in the closed middle ear space is problematic especially in the presence of eustachian tube blockage. This results in an increase in middle ear pressure. During tympanoplasty, the tympanic membrane graft may become dislodged. It also increases PONV. If tympanoplasty, stapedotomy or stapedectomy is planned, N_2O should be avoided.

RECOVERY

The ear is bandaged at the end of the procedure. To prevent displacement of the tracheal tube and trauma to the eyes, the anaesthetist should supervise this.

Nausea, vomiting and dizziness can be a particular problem following these procedures. Antiemetics should be given using a multimodal approach and not a single agent. Pain is not usually severe.

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CROSS-REFERENCES

OESOPHAGOSCOPY

ADEL HUTCHINSON

Two types of oesophagoscopy are possible: rigid and flexible fibre-optic. Rigid oesophagoscopy necessitates general anaesthesia, whereas flexible is well tolerated with topical anaesthesia ± sedation in adults.

INDICATIONS

- Removal of foreign body/food bolus
- Investigation of carcinoma, e.g. as part of panendoscopy
- Dilatation of strictures
- Assessment and treatment of oesophageal/pharyngeal lesion
- Endoscopic treatment of pharyngeal pouch

CONSIDERATIONS

- Obstruction poses an aspiration risk with food and saliva present above the level of obstruction.

- Chronic obstruction may result in weight loss, dehydration and silent aspiration.
- Pre-existing comorbidities, e.g. cardiovascular disease, GORD, neurological conditions (dysphagia may be a presenting symptom).

PREOPERATIVE PREPARATION

- Investigate according to underlying cause.
- Treat dehydration or chest infection.
- Avoid sedating premedication.
- Measures to neutralise gastric acid take time to be effective; there will still be a danger from blood, food and secretions above the level of obstruction.

CONDUCT OF ANAESTHESIA

- Shared airway with ENT surgeons so ensure good communication.
- Rapid sequence induction and endotracheal intubation is mandatory (with a tube smaller than usual) due to risk of regurgitation. Avoid hand ventilation if possible to prevent further impaction of foreign bodies.
- Secure tube to the left to allow for the oesophagoscope – the tube can become kinked; consider a reinforced tube and pay close attention to airway pressures.
- IV induction and short-acting muscle relaxant are required. Desflurane allows prompt return of airway reflexes. Use neuromuscular monitoring and reverse if necessary.
- Cardiovascular disturbance should be expected. In the presence of dehydration or cachexia, hypotension may occur. Hypertension and tachycardia are common and should be dealt with promptly, particularly in the elderly or those with cardiovascular disease. Fentanyl or alfentanyl can attenuate this response.

POSTOPERATIVE CONSIDERATIONS

- Patients remain an aspiration risk; therefore, extubate awake and fully reversed.
- Odynophagia may indicate oesophageal perforation and can lead to pneumomediastinum, mediastinitis, pneumothorax and surgical emphysema. If there is suspicion of this, a chest X-ray and prompt discussion with the surgeons are necessary.
- Ensure IV fluids are prescribed if the patient is to remain nil by mouth postoperatively.

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CROSS-REFERENCE

ENT surgery – general principles, Chapter 19

OPERATIONS ON THE NOSE

These can be simple and straightforward, e.g. manipulation of nasal bones; or they may be more complex and prolonged, e.g. transnasal skull base surgery. Septoplasty is performed to relieve symptoms of nasal obstruction or as a component of rhinoplasty. It can be combined with turbinate reduction surgery, or to facilitated CPAP in OSA patients. Rhinoplasty is performed for cosmetic or reconstructive surgery, post-trauma, reconstruction after tumor resection or to improve nasal breathing. A bloodless field is helpful and the aim is for a still patient with no or minimal coughing and straining followed by a smooth emergence. Some operations such as septoplasty may be performed under local anaesthesia with sedation in cooperative patients although most nasal operations require a general anaesthetic.

COMMON PROCEDURES

- Submucus resection of septum, septoplasty, turbinectomy, polypectomy, antral washout, rhinectomy.

PREOPERATIVE PREPARATION

- Detailed history and examination with appropriate investigations.
- Patients with polyps often have a history of atopy or the triad of asthma, polyps and aspirin sensitivity.
- Postnasal drip and recurrent chest infections are common.
- Treat chest infections and optimise chronic medical conditions.
- Patient with nasal fractures may have sustained other injuries or swallowed a significant amount of blood.
- Correct any hypovolaemia in patients who have bled significantly.
- There is an increasing incidence of patients with obstructive sleep apnoea.
- Anxiolytic premedication if required.

Vasoconstrictors will reduce bleeding from nasal surgery and can be administered as a spray, paste, infiltration or on soaked swabs:

- Moffatt's solution (2 mL 8% cocaine, 2 mL 1% sodium bicarbonate and 1 mL 1:1000 epinephrine).
- Modified Moffatt's solution (10% cocaine and 8.4% bicarbonate).
- Local anaesthetic with 1:100,000–1:200,000 epinephrine.
- Lidocaine 5% and phenylephrine 0.5% spray.
- Xylometazoline spray.
- Cocaine 4%–10% (recommended maximum dose 1.5 mg/kg).

INTRAOPERATIVE MANAGEMENT

- Routine monitoring.
- A rapid sequence induction is required as patients may have swallowed a significant amount of blood.
- Induction agent of anaesthetist's choice.
- Oral preformed or reinforced tracheal tube. Some use a laryngeal mask.
- A throat pack is inserted, if required.
- Protect eyes with ointment but not covered in order that the surgeon can check for orbital perforation or damage to the optic nerve.

MAINTENANCE

- Inhalational agent and opiates (e.g. Remifentanil infusion)
- TIVA
- Muscle relaxant as necessary.
- Head-up position with the thighs flexed at the hip in order to improve venous return.

EXTUBATION

- When the procedure is complete, perform pharyngoscopy to ensure that the pack has been removed completely and any remaining blood clots or debris are aspirated.
- Extubate awake sitting up.

POSTOPERATIVE MANAGEMENT

- Encourage the patient to breathe through his or her mouth because nasal packs are often in place.
- Plasters and bolsters applied to the nose may make the application of a facemask difficult.
- Administer oxygen in recovery in a routine fashion but CPAP is required as soon as possible in OSA patients.
- Leave the intravenous cannula in situ in case of bleeding.
- Repacking may be necessary should bleeding from the nose continue.
- Regular paracetamol and NSAIDs are usually adequate. Opioids are needed following more extensive surgery but be aware of respiratory depression in OSA patients. Prescribe an antiemetic.

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CROSS-REFERENCE

ENT surgery general principles, Chapter 19

TONSILLECTOMY AND ADENOIDECTOMY

Recurrent acute sore throat is a very common condition presenting in primary care and tonsillectomy is one of the most common operations. It presents a significant burden of disease. For tonsillectomy there is good evidence addressing effectiveness in children but limited evidence in adults.

PROCEDURE

Adenotonsillar hypertrophy can present with nasal obstruction, recurrent infections, secretory otitis media, deafness (secondary to Eustachian tube dysfunction), and obstructive sleep apnoea (OSA).

LOCATION OF SURGERY

- *Children* – Ideally within a paediatric surgical facility as a day case, although day-case care may be contraindicated in the presence of significant sleep apnoea.
- *Adults* – Ideally as a day case.

Adenoidectomy and/or tonsillectomy procedures are performed through the mouth. A Boyle–Davis gag is used for tonsillectomy. Difficulties may be encountered because of poorly placed gag, obstructing the tracheal tube or laryngeal mask airway.

PATIENT CHARACTERISTICS

- Chronic/recurrent throat infections.
- Comorbidities
  - Obstructive sleep apnoea
  - Congenital abnormalities, e.g. Down syndrome.
  - Older adults for tonsillectomy
  - May have malignancy.
  - Other incidental medical conditions.
  - Cor pulmonale due to long-term hypoxia.

PREOPERATIVELY

- Detailed assessment and appropriate investigations
- Assess for OSA
  - STOP-BANG questionnaire
  - Epworth sleep score
  - Bleeding history is important.
  - Detailed airway assessment.
  - Possible difficult management due to large tonsils.

PREMEDICATION

- Anxiolytic if essential but avoid if a history of airway obstruction or OSA.

PERIOPERATIVE MANAGEMENT

- Standard monitoring.
- Intravenous or inhalational induction.
- Airway:
  - Intubate with a preformed oral or reinforced tube.
  - Reinforced laryngeal mask
- Oral tubes must be carefully secured in the midline in order to lie correctly in the Boyle–Davis gag.
- Patients are positioned with the neck extended.
- Instrumentation of the postnasal space during adenoidectomy may induce a bradycardia requiring treatment with atropine or glycopyrrolate.
- Analgesia
  - Opioid analgesia is usually required.
  - IV paracetamol.
  - NSAIDs unless contraindicated.
  - Infiltration of local anaesthetic into the tonsillar bed.
- Careful suctioning of the pharynx under direct vision.
- Extubation either deep or awake depending on preference.

POSTOPERATIVE MANAGEMENT

- NSAIDs have not been found to significantly increase bleeding in tonsillectomy patients.

- Maintain IV access in case of early postoperative bleeding.
- Bleeding may not be detected in children until vomiting occurs.
- Severe OSA patients have a higher incidence of perioperative complications and may need postoperative HDU/ICU care.
- Routine use of antiemetic drugs to prevent PONV is recommended.

MANAGEMENT OF THE BLEEDING TONSIL

This serious complication can present in recovery or occur hours later. Persistent swallowing can be an early indicator of bleeding from the tonsillar bed. The volume of blood loss cannot be measured and so is difficult to assess. The patient may be hypovolaemic and require fluid resuscitation prior to induction, so careful assessment of patient's fluid status and cardiovascular parameters is required prior to induction. In addition to cardiovascular instability, there is risk of aspiration due to potential full stomach with blood, in combination with a potential difficult intubation due to blood and oedema in the airway.

INTRAOPERATIVE MANAGEMENT

- Experienced anaesthetist available.
- Patients should be resuscitated and have full monitoring applied.
- Assess previous anaesthetic record.
- Suction must be immediately available and head-down tilt helps to drain blood away from the larynx.
- Rapid sequence induction. Some anaesthetists prefer an inhalation induction performed on the left side, in the head-down position with cricoid pressure. Ultimately, the technique chosen will depend on the skills and preference of the anaesthetist.
- Intubation may require a smaller sized endotracheal tube than originally inserted.
- Volume resuscitation should continue throughout surgery.
- A wide-bore naso/oro-gastric tube to empty the stomach, ensure stomach empty prior to extubation.

- Patients should be extubated when fully awake – traditionally in the head down left lateral position.

POSTOPERATIVE MANAGEMENT

- Patient must remain in recovery for an extended period to ensure the bleeding has stopped.
- Check the haemoglobin and coagulation, transfuse blood if necessary.
- Patients should be closely monitored for evidence of further bleeding.

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CROSS-REFERENCES

Infants and children, Chapter 24
ENT surgery general principles, Chapter 19
Difficult airway – management, Chapter 26
Difficult airway – aspiration risk, Chapter 26

TRACHEOSTOMY

This is an elective or emergency procedure usually performed under general anaesthesia. Rarely is it performed under local anaesthesia in an emergency setting. Critical incidents can occur during the initial placement of a tracheostomy, during the care of patients with a pre-existing tracheostomy or as a consequence of having a tracheostomy. There were 14 cases of displaced tracheostomies reported to NAP 4 with half of these resulting in death.

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INDICATIONS

- Acute or chronic upper airway obstruction.
- Planned during head and neck surgery, e.g. for cancer of tongue, oral cavity or upper airway.
- Chronic respiratory failure.
- Retention of bronchial secretions (weak cough, pneumonia, cystic fibrosis).
- Risk or presence of pulmonary aspiration (e.g. stroke).

RELATIVE CONTRAINDICATION

- Uncorrected coagulopathy and thrombocytopaenia.
- Haemodynamic instability.
- Soft tissue neck infection.
- In laryngeal cancer prior to laryngectomy, to minimise chance of stomal recurrence.

PREOPERATIVE ASSESSMENT AND INVESTIGATION

- Coexisting cardiopulmonary, neurological or muscular disease.
- Signs of aspiration or airway obstruction (e.g. stridor).
- Difficult laryngoscopy and/or difficult facemask ventilation.
- Routine investigations plus chest and cervical spine radiographs, CT scans, pulmonary function tests and baseline arterial blood gases.

PREPARATION AND PREMEDICATION

- Assume a difficult intubation: check equipment and have a plan with contingencies.
- Various sizes of tracheostomy tubes, sterile catheter mount and suction catheters should be available and checked for correct connectors.
- Anxiolytic if required: avoid if signs of obstruction.
- Antisialogogue intravenously before induction.
- Continue routine medications.

INDUCTION AND MAINTENANCE

PREOXYGENATE

- Start only when surgeon and staff scrubbed and instruments are ready.
- Use intravenous induction only if confident; otherwise, consider inhalational induction or awake fibre-optic intubation.
- Secure endotracheal tube so that it can be withdrawn cautiously when required.
- TIVA for maintenance.
- Position: careful neck extension, head-ring and head-up tilt 10–15 degrees.
- Expose the neck and increase the distance from the cricoid cartilage to the sternal notch, thereby maximizing surgical exposure.
- Clear oropharyngeal secretions.
- Tracheal incision is usually at third tracheal ring, above ETT cuff.
- Ventilate manually with 100% oxygen during tracheostomy tube insertion.
- Retract ETT until tip is just above incision enabling tracheostomy tube insertion.
- Check position of tube and $ETCO_2$ trace before reattaching ventilator. If there is no end tidal CO_2 seen or an inability to ventilate, the endotracheal tube must be reinserted into the trachea. It is likely that the tracheotomy tube is in a false passage, either anterior to the tracheal wall or posterior to the trachea into the oesophagus.
- Once correct position of tracheostomy tube is confirmed, withdraw ETT completely.

POSTOPERATIVE MANAGEMENT

- Check chest radiograph (tube position, pneumothorax, surgical emphysema, lobar collapse).
- Nurse patient sitting-up in an appropriate area (humidified oxygen, suction, monitoring, skilled staff).
- Emergency equipment available
  - Basic airway equipment: oxygen masks, self inflating bags, oral and nasal airways
  - Advanced airway equipment: laryngeal masks and laryngoscopes with appropriate tubes

- Capnography
- Fibreoptic scope
- Tracheal dilators
- Bougies
- The National Tracheostomy Safety Project has developed algorithms that bring together the knowledge, skills and actions required to manage a tracheostomy emergency. The algorithms describe a universal approach to managing such emergencies and are designed to be followed by first responders. The project aims to improve the management of tracheostomy and laryngectomy critical incidents.
- Tube change is usually only performed after a week in order to allow time for formation of a tract.

COMPLICATIONS

- *Immediate* – Bleeding, pneumothorax, false passage and venous embolism.
- *Early* – Malposition, displacement, false passage, bleeding, surgical emphysema, pneumothorax/peumomediastinum, stomal erosion and obstruction (blood, sputum, tracheal wall).
- *Late* – Displacement, obstruction, infection (pneumonia or stomal), erosion, bleeding (minor or major), stenosis (tracheal or stoma site), fistula formation, tracheomalacia and voice changes.

EMERGENCY TRACHEOSTOMY

- Usually performed under local anaesthesia when the airway is obstructed.
- Cricothyroidotomy set should be readily available where anaesthesia is undertaken.

PERCUTANEOUS

- Elective procedure usually performed by a trained Intensivist.
- At bedside: quicker, less traumatic, cost-effective.
- Possibly higher incidence of late complications.

- Performed more readily in the ICU, thus eliminating the logistic problems that may occur when transferring a ventilated patient to the operating theatre.

TECHNIQUES

- Serial dilatation (Ciaglia)
- Single tapered dilatation (Blue Rhino™)
- Guidewire dilating (Griggs') forceps
- Screw (PercuTwist™)
- Balloon dilatation
- Translaryngeal tracheostomy

INDICATIONS

- Prolonged endotracheal intubation
- To facilitate weaning from ventilator and aid nursing

PREOPERATIVE ASSESSMENT

- Coexisting disease, ventilation.
- Consider deferring if patient is PEEP dependent or coagulopathy uncorrected.

PREPARATION

- Two operators and a bronchoscope are needed.
- Give general anaesthesia with 100% O_2.
- Some advocate change to smaller ETT before starting.
- Withdraw ETT to above site of tracheostomy.
- Adjust ventilation or insert a throat pack to compensate for cuff deflation.

PROCEDURE

- Use a head-ring, sandbag under the shoulders and head-up position.
- Infiltrate incision site with lidocaine and epinephrine.
- Site is usually between first and second tracheal rings.
- Dissect down to trachea.
- Check position of introducer and guide-wire bronchoscopically from above.
- Use a high-volume low-pressure cuffed tracheostomy tube.

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POSTOPERATIVE MANAGEMENT

- Adjust ventilator settings
- Chest X-ray

COMPLICATIONS

- *Immediate* – Loss of airway, major bleeding, pneumothorax, surgical emphysema, posterior tracheal wall damage. Tracheal ring fracture and false passage formation.
- *Early* – Malposition, displacement, false passage, bleeding, surgical emphysema, pneumothorax/ peumomediastinum, stomal erosion and obstruction (blood, sputum, tracheal wall).
- *Late* – Displacement, obstruction, infection (pneumonia or stomal), erosion, bleeding (minor or major), stenosis (tracheal or stoma site), fistula formation, tracheomalacia and voice changes.

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CROSS-REFERENCES

ENT surgery general principles, Chapter 19
Difficult airway – management, Chapter 26

20 Head and neck surgery

ROSS MACNAB AND KATHERINE BEXON

DENTAL ABSCESS

Dental abscesses usually result from infected teeth and cause localized pain and swelling. They may restrict mouth opening and can be associated with systemic illness, pain, pyrexia and malaise. Their size and possible clinical consequences of fascial or cervical space infection can be underestimated. If infection is unchecked spread within fascial planes can cause orbital cellulitis, cavernous sinus thrombosis and significant airway compromise. Some cases can be done under local anaesthesia or will respond to antibiotics; general anaesthesia is often required.

PROCEDURES

- Periradicular surgery (e.g. apicectomy)
- Exodontia (including surgical removal)
- Skin incision and external drainage
- They can often be managed as day cases

PATIENT CHARACTERISTICS

- History of dental decay
- Children
- Neglected adults
- Patients with special needs

PREOPERATIVELY

- Full general assessment, including appropriate investigations
- Assessment of comorbidities
- Detailed airway assessment
  - Site of abscess
  - Mouth opening
  - Level of airway compromise
  - Identify difficulties in airway management

PERIOPERATIVELY

- Skilled assistance is essential
- Standard monitoring

- Difficult airway equipment
- Preoxygenation
- Induction plan depends on findings and the anaesthetist's preference
  - Intravenous induction
  - Inhalational induction
  - Awake fibreoptic intubation
  - Tracheostomy under local anaesthesia
- Airway
  - Laryngeal mask
  - Tracheal intubation, nasal or oral
- Use absorbent throat pack to reduce airway contamination
- Maintenance: TIVA or inhalational technique
- Antibiotics and steroids as required

POSTOPERATIVE MANAGEMENT

- Ensure any throat pack is removed.
- Oropharyngeal suction under direct vision.
- Use DAS extubation guidelines.
- Extubate when awake.
- Paracetamol, NSAIDs (unless contra-indicated) and opioids as required.

LUDWIG'S ANGINA

This is a multispace infection of the floor of mouth. Infection and inflammation spreads in the submental, sublingual, buccal and submandibular spaces. Rarely a spreading cellulitis with extensive induration and swelling of the anterior neck may occur. Fever, pain, dysphagia, trismus, acute confusional state, swelling of the floor of the mouth and significant compromise of the airway may be present. This is a potentially life-threatening condition, which requires urgent and expert management.

PREOPERATIVE MANAGEMENT

History of altered speech, odynophagia, rapidly worsening swelling, and severe trismus can signify partial airway obstruction. Stridor and dysphagia are often late signs and stridor may be absent when at rest.

- Lack of tongue protrusion is a sensitive indicator of sublingual involvement and a good indicator of impending airway compromise.

- Airway problems can be more serious and include:
  - Critical airway patency
  - Secretions
  - Trismus
  - Airway distortion and tissue immobility
- If preoperative imaging is available:
  - Facilitates surgical diagnosis and operative strategy.
  - Review for further evaluation of the airway.
- Be aware that all primary techniques may fail, so have clear rescue plans in place before commencing anaesthesia. Communicate these to all team members. Use the DAS difficult intubation guidelines.

INTRAOPERATIVE MANAGEMENT

- Decide – theatre or anaesthetic room.
- Senior anaesthetist and surgeon involvement.
- The potential for a 'can't intubate, can't ventilate' scenario is very real.
- Plan for front of neck access.
  - Facilities for immediate emergency tracheotomy with surgeon scrubbed.
  - Surgical tracheostomy is often difficult and life-threatening due to the involvement of the neck and pretracheal tissues.
  - Incising through the pretracheal fascia and exposing the prevertebral tissues to pathogens risks the spread of infection into the mediastinum.
  - Mediastinitis carries significant mortality.
- Preoxygenation
  - High flow nasal oxygen
- Careful consideration for induction strategy
  - DAS difficult intubation guidelines (Plan A, Plan B, etc.)
  - Awake fibre-optic intubation.
    - NAP 4 recommends if fibre-optic intubation is preferred option with head and neck pathology, consideration should first be made to performing it awake. The airway strategy should accept it may fail, particularly when performed in an unconscious patient.

- Caution with muscle relaxants.
  - If necessary use rocuronium, which can be rapidly reversed by sugammadex.
  - Muscle relaxants may not lead to increased mouth-opening due to trismus.

POSTOPERATIVELY

- DAS extubation guidelines.
- Ventilate electively if any concerns, until airway is safe.
- Some patients will need HDU or ITU for ongoing monitoring and support.

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CROSS-REFERENCES

DENTAL SURGERY

All community dental work requiring general anaesthesia is now carried out in a hospital setting in the presence of an anaesthetist. There are estimated to be 65,000 children and young people with severe learning disabilities in the UK, and a significant proportion of those needing dental treatment will be referred for general anaesthesia. This vulnerable group requires access, communication and perioperative care appropriate for their individual needs.

PROCEDURES

- Restorative dentistry (fillings, crowns, etc.)
- Periradicular surgery (e.g. apicectomy)
- Exodontia
- Surgical exposure or removal of teeth
- Dental implants

PATIENT CHARACTERISTICS

- Children.
- Young adults.
- Day cases.
- Dental phobia.
- Patients with learning difficulties. These patients face many physical, psychological and social challenges that affect their ability to cope with the routines of hospital-based care. A flexible, holistic approach to treatment with good communication and individualized planning of care can create a positive experience for patient, family and carers.
- A rapid smooth recovery and early discharge should be the aim when possible.

PREOPERATIVE ASSESSMENT

Assessment before the day of surgery is useful.

- Full general assessment, including appropriate investigations
- Full airway assessment
- Day-case suitability

PREMEDICATION

- Topical anaesthetic cream
- Paracetamol and NSAIDs (unless contraindicated)
- Routine medications

PERIOPERATIVELY

- Skilled assistance
- AAGBI monitoring standards

INTRAOPERATIVE MANAGEMENT

- Inhalational or intravenous induction – caution if difficult airway.
- Flexible laryngeal mask or tracheal intubation.
- Sevoflurane, desflurane or propofol (TIVA) for maintenance of anaesthesia.
- Monitor airway patency continuously and lift chin or thrust jaw if needed.
- Clear communication with surgeon regarding airway requirement.
- Throat pack can be used to reduce airway contamination.
  - If used, all safety precautions *must* be followed at end of surgery.
- Local anaesthesia for postoperative analgesia.
- Consider antibiotics and antiemetics.
- Opioids are seldom required.

POSTOPERATIVE MANAGEMENT

- Oropharyngeal suction under direct vision.
- Awake extubation.

PATIENTS WITH SPECIAL NEEDS

PREOPERATIVELY

- Best interest meeting, resolve any consent issues.
- Work together with regular care-provider, MDT approach.
- Degree of understanding should be evaluated.
- Requires careful assessment and detailed discussion with regular care-providers.
- May be impossible to assess fully.

- Comorbidity may be present; neurological diseases (epilepsy, spasticity, etc.), craniofacial malformation, congenital cardiac disease, metabolic disorders, pulmonary disease, uncommon syndromes.
- May present with a difficult airway, e.g. macroglossia.
- May not understand or cooperate with treatment and can present difficult consent issues.
- Reference to previous anaesthetic notes.
- Careful planning for dental treatment options and goals (short and long term).
- Individual personalised anaesthetic management plan.
- Opportunity to carry out other examinations, investigations and treatments by other specialties whilst anaesthetised, e.g. ophthalmology, urology, gynaecology.

PERIOPERATIVELY

- Should attend with regular care providers.
- Key role of regular care providers during perioperative period should be emphasised.
- Regular care providers present whilst awake.
- Should normally continue their usual medication.
- May benefit from sedative drugs (e.g. temazepam or midazolam)
  - Administer by best tolerated route (e.g. oral, nasal, rectal).
- Consider slower gastric emptying because of disease or medical side effects.
- Maintain familiar environment.
- May prefer either inhalational or intravenous induction of anaesthesia.
- Consider personal safety issues.
- Identify lifting and handling or positioning issues.
- Aspirate nasogastric tube and PEG-tubes if present.
- Flexible approach to induction of anaesthesia.
- Try to avoid making the next time more difficult.

- Avoid nasal intubation because patients cannot handle epistaxis and this may delay or prevent discharge in day-case surgery.
- Use short-acting agents.
- Accept goal reduction if intended treatment not possible.
- Surgical change of treatment/intervention because of new intraoperative diagnoses makes planning of exact management difficult.

POSTOPERATIVELY

- Write detailed notes on what worked and what didn't.
- May require repeat visits.
- Often appreciate being treated by staff and surroundings they recognise.
- Discharge when recovered to preoperative status.

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Preoperative assessment overview, Chapter 25
Preoperative risk and its communication, Chapter 25
Day-case anaesthesia, Chapter 25
Infants and children, Chapter 24
Difficult intubation (difficult airway), Chapter 26

FACE AND JAW FRACTURES

The face is divided into three equal parts: the lower third involving mandible, middle third or mid-face made up of the maxilla, zygoma, and lower half of the naso–orbito–ethmoidal complex and upper third involving frontal bone, sphenoid and upper half of the naso–orbito–ethmoidal complex. Maxillary fractures can also be classified according to the traditional Le Fort system: Le Fort I (horizontal) the lower maxilla, Le Fort II (pyramidal) the maxilla and nasal complex and Le Fort III (transverse) the whole mid-face dissociates from the base of the skull and facial bone. A quarter of Le Fort II/III fractures are accompanied by a nasal csf leak, and up to 6% of significant maxillofacial trauma is associated with cervical spine injury. Up to 50% of patients may have multisystem trauma.

PROCEDURES

- Simple manipulation of nasal fracture or elevation of a fractured zygoma.
- Application of intra-maxillary fixation with arch bars.
- More complex internal fixation with screws and titanium plates.

PATIENT CHARACTERISTICS

Any age group is possible but most are young with fractures due to trauma. Alcohol or drug intoxication may be implicated. There may be a coexisting head or cervical spine injury, raised ICP or diseases such as epilepsy, cerebrovascular or cardiovascular disease. There may be other significant associated injuries.

Seriously injured patients with airway difficulties need early tracheal intubation. The airway may

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be obstructed by blood, blood clots, dislodged teeth, displaced bone fragments, swollen tongue, subcutaneous emphysema, trismus or generalised oedema. A full stomach must be assumed.

Patients without a compromised airway are suitable for semi-elective repair of facial fractures when the facial swelling has reduced. Definitive surgical treatment may also be delayed if there is a need to treat other major injuries first.

PREOPERATIVE ASSESSMENT

- History and mechanism of injury
- Detailed airway assessment
- Presence of other associated injuries
- Coexisting medical conditions

PREMEDICATION

- Avoid sedative premedication in the emergency situation.
- Antisialogogue may be given if awake intubation is planned.

PERIOPERATIVE MANAGEMENT

In the acute situation with airway compromise, the priority is to secure a definitive airway. The technique will depend on patient factors and the experience of the anaesthetist. Thoroughly preoxygenate. Bag mask ventilation may be impossible because of panfacial fractures/instability of the face; beware of nasal csf leak.

Options include:

- Inhalational induction (airway obstruction or laryngospasm may occur).
- Intravenous induction.
- Awake fibre-optic intubation (cervical spine injury, skull fractures, trismus).
- Tracheostomy under local anaesthesia.
- In some rare indications a retromolar or submental intubation can be an alternative option.
- Assemble all difficult airway equipment prior to starting.

Any technique may fail in these complex cases and a series of preformulated airway management plans must be in place. The anaesthetist, surgeon and theatre team should agree upon strategies prior to starting. All the necessary equipment and personnel must be assembled.

Isolated mandibular fractures do not usually cause airway problems or intubation difficulty. Trismus associated with a fractured mandible is usually relieved on induction of anaesthesia.

Caution is required if basal skull fracture is present or it has not been excluded, as a nasal tube can pass through this fracture site and enter the cranium. There must be a risk assessment and either use an alternative route or pass tube with care with a good anatomical knowledge that the nasal floor is horizontal when upright.

MAINTENANCE OF ANAESTHESIA

- Standard monitoring, including capnography.
- Invasive monitoring indicated depending on other injuries or comorbidities.
- TIVA or inhalational agent.
- Remifentanil infusion is useful.
- If throat pack required, appropriate care taken to ensure removal at end.
- Head up position will help surgical field and reduce blood loss. Maintain cerebral perfusion pressure if significant head injury suspected with concomitant increase of intracranial pressure.
- Dexamethasone to decrease tissue swelling.
- Prophylactic antiemetics and antibiotics.

IMMEDIATE POSTOPERATIVE PERIOD

Extubation and immediate recovery period is a time of risk. If using the DAS extubation guidelines and stratifying to high risk due to significant facial swelling, the patient should remain intubated and be transferred to ICU until the swelling has subsided.

Otherwise, extubate patient once good laryngeal reflexes have returned and patient is fully awake.

POSTOPERATIVELY

Provide care in the appropriate environment with staff that have the appropriate skills. This will depend on the patient, the injury and the surgery done.

- Simple analgesia and NSAIDs with cautious use of opioids as required.
- Intravenous fluids continued until oral intake resumes.

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CROSS-REFERENCES

Difficult airway management, Chapter 26
Trauma, Chapter 30

HEAD AND NECK SURGERY – GENERAL PRINCIPLES

PATIENT CHARACTERISTICS

Patients range from the very young to the elderly. Young fit males present with trauma; the elderly ASA 3 patient presents for resection of a malignancy. The common themes concern the shared airway. There is no consensus as to the best primary plan of anaesthesia. There are advocates of awake fibre-optic intubation, tracheostomy under local anaesthesia, inhalational induction, 'standard' intravenous induction with muscle relaxants and use of a transtracheal catheter. The type of airway best suited to carry out the surgical procedure is the primary consideration.

PREOPERATIVE

- Carry out a detailed history and examination with particular reference to airway assessment and pathology.
- Assess comorbidities; previous anaesthetic history.
- Perform relevant and targeted investigations.
- Review any airway imaging: CT, MRI scans
- Flexible nasendoscopy can be used to aid anaesthetic planning.

PREMEDICATION

- Avoid sedative medication if there is any suggestion of airway compromise.
- Antisialagogue premedication is helpful when awake fibre-optic intubation is considered.
- Consider gastric acid prophylaxis.

PERIOPERATIVE MANAGEMENT

- Skilled multidisciplinary team.
- Team brief and checklist.
- Establish IV access.
- Airway equipment
  - Laryngoscopes, video laryngoscopes, fibre-optic scopes, variety of ET tubes and supraglottic airways, bougies, exchange catheters and other airway adjuncts.
  - Cricothryroidotomy surgical equipment.
- Preoxygenation
  - Face mask
  - Avoid desaturation (nasal oxygenation during efforts to secure a tube)
  - High flow nasal techniques
- Monitoring:
  - Standard monitoring
  - Neuromuscular monitoring

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- Core temperature
- Invasive monitoring required depending on comorbities, injuries, extent, complexity and duration of surgery
- Induction:
  - Location of induction; anaesthetic room or theatre.
  - IV, inhalation or awake procedure to secure the airway.
  - Induction agent titrated to patient response.
  - Rapid sequence induction if warranted.
  - Relaxant: rocuronium is appropriate with suggamadex available to reverse if required.
  - Local anaesthetic to airway.
  - Vasoconstrictor to nasal passage if nasal route for intubation.
  - Inhalational agent; sevoflurane or desflurane.
  - Opiates; remifentanil is particularly helpful in this setting.
  - Steroids for swelling.
  - Antibiotics.

MAINTENANCE

- Maintain anaesthesia with oxygen, air and inhalational agent, or TIVA.
- Remifentanil infusion.
- Head up position and relative hypotension can reduce bleeding and aid surgical field, if required.

EXTUBATION

- Any throat pack *must* be removed and confirmed by team members.
- Airway clear of secretions and blood.
- Plan for extubation using the DAS guidelines
  - Stratify to low or high risk
  - Awake or deep, advanced extubation techniques or delay extubation/surgical airway

POSTOPERATIVELY

- Return patient to the appropriate area depending on patient and surgery: day-case facility, head and neck ward, HDU, ICU.

- Postoperative analgesia: use a multimodal approach: local block, paracetamol, NSAIDs and opiates.
- Antiemetics.

COMPLICATIONS

Complications to be aware of are related to airway compromise and complications, a level of high vigilance must be maintained by all staff.

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LARYNGECTOMY AND RADICAL NECK DISSECTION

KATHERINE BEXON

Laryngectomy and its variants, e.g. vertical partial hemilaryngectomy, supraglottic partial laryngectomy and supracricoid partial laryngectomy, are performed for malignant disease of the larynx. Radical neck

dissection for cervical metastases sacrifices vital structures in the neck and significantly impacts quality of life. Modified radical neck dissection preserves one or more non-lymphatic structures. Pharyngolaryngectomy and tissue flap may be required if there is evidence of pharyngeal involvement. Reconstruction can be challenging due to the lack of local tissue and reduced tissue perfusion resulting from radiation therapy or previous surgery and scar formation.

PATIENT CHARACTERISTICS

Frequently elderly with comorbidities associated with tobacco and alcohol ingestion.

PREOPERATIVE ASSESSMENT

- A multidisciplinary team approach is essential.
- Assessment by specialist anaesthetic preoperative service.
- Comorbidities and the potential for cardiovascular instability should be considered and detailed assessment of cardiac and respiratory system carried out.
- Patients have usually had recent anaesthesia for panendoscopy or flexible fibre-optic laryngoscopy to assess the primary tumour.
- CT imaging is used to determine the extent of the disease.
- Positron emission tomography can be used to assess metastases.
- Patients may present with airway narrowing due to tumour progression or oedema secondary to radiation therapy.
- Nutritional assessment and supplementary enteral feeding may be required.

PERIOPERATIVE MANAGEMENT

- Avoid premedication especially in patients with airway narrowing.
- Difficult intubation and emergency tracheostomy equipment ready.
- Clear plan with contingencies and communicated to all team members.
- AAGBI standard monitoring with arterial line, urinary catheter, nerve stimulator and core temperature monitoring.

- If central venous access is required, use a site remote from the surgical site.
- Consider noninvasive cardiac output monitoring.

There is a risk of total airway obstruction on induction of anaesthesia for patients with partial airway obstruction. Tumours can be friable and bleeding can obscure the view.

ANAESTHETIC TECHNIQUE

- Airway management planning should consider history, examination and the findings from scans, panendoscopy and flexible nasendoscopy.
- Maintenance can be achieved with TIVA or an inhalational agent supplemented with an opioid.
- Bradycardia and hypotension can occur during dissection around the carotid vessels. Vagolytics or local anaesthesia applied by the surgeon can decrease this.
- Asymptomatic plaques at the carotid bifurcation can be dislodged during surgical manipulation.
- Inadvertent carotid compression can occur during en-bloc tumour dissection.
- Excision of the internal jugular vein impairs venous drainage.
- Damage to major vessels can be problematic when access for control is difficult.

AIRWAY MAINTENANCE

- Increased FiO_2 prior to incision of the trachea requires clear communication between surgeon, anaesthetist and all of the team.
- The tracheal tube is identified by the surgeon and can be withdrawn slowly under direct vision to just above the incision. It can be reintroduced to the lower trachea in the event of any problem.
- Insertion of a new tube by surgeon. The tube position must facilitate fashioning of the stomal site with connection beyond the immediate surgical site and good

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manoeuvrability. Reinforced or 'laryngectomy' tubes are suitable.

- At the end of the surgery the tube is usually replaced with a conventional tracheostomy tube.
- A nasogastric tube for postoperative enteral nutrition should be placed under direct vision by the surgeon.

POSTOPERATIVE CARE

These patients need care in a high dependency area or ICU. Designated clinical areas caring for these patients centralise knowledge, clinical experience and skills and facilitate the appropriate education of staff. The National Tracheostomy Safety Project (NTSP) recommends that patients should have NTSP laryngectomy bedhead signs summarizing essential information about the airway of the patient. Availability of appropriate bedside equipment including waveform capnography is vital.

Early complications are

- Problematic airway integrity and poor cough (especially after hemilaryngectomy).
- Various nerve palsies (phrenic nerve may be injured during neck dissection).
- Chyle leak.
- Hypertensive crisis (altered baroreflex mechanism due to surgical manipulation around the carotid sheath).
- Haemorrhage.
- Increased ICP (excision of the internal jugular vein).
- Facial and laryngeal oedema after radical neck dissection (tends to improve over a few days but may not return to normal).
- Treatment of tobacco and alcohol dependence.
- Pain management – patient-controlled analgesia is recommended.

Enhanced recovery protocols have been developed for patients undergoing major head and neck oncological resection, neck dissection and reconstruction with tissue flap. This should enhance the perioperative patient experience and improve outcomes.

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CROSS-REFERENCES

Laryngoscopy and microsurgery of the larynx, Chapter 19
Difficult airway – difficult tracheal intubation, Chapter 26
Hypoxaemia under anaesthesia, Chapter 26
Postoperative pain management, Chapter 30
Raised ICP and CBF control, Chapter 30

OPERATIONS ON THE SALIVARY GLANDS

There are two parotid, two submandibular and two main sublingual glands. Surgery on the salivary glands may be simple procedures, e.g. for a stone blocking a duct, or more complex excision, e.g. glandular malignancy. Most salivary gland tumours occur in the parotid gland (80%) and represent <3% of head and neck neoplasms. The majority are benign, whereas only 50%–60% of submandibular tumours are benign. Most calculi (80%) occur in the submandibular gland and its duct. The operative

approach may be either through the skin or intra-orally through the mucous membrane.

Parotidectomy is a complex procedure because the facial nerve divides it into a large supra-neural and a small infra-neural component. Accurate, safe localization of the facial nerve and avoiding injury is crucial. A nerve stimulator is often used so muscle relaxants should be avoided.

PREOPERATIVE EVALUATION

- History, examination, and appropriate investigations with evaluation of any coexisting disease.
- Detailed airway assessment including any issues related to gland enlargement.

PERIOPERATIVE MANAGEMENT

- Key issue: Facial nerve integrity monitoring and use of muscle relaxants.
- Standard monitoring.
- Invasive monitoring, only if significant comorbidities.
- Peripheral nerve stimulator, if relaxants have been used.
- Induction with propofol and opioids (e.g. remifentanil, alfentanil, fentanyl).
- Relaxant:
  - Rocuronium (rapidly reversed by sugammadex if required).
  - An alternative technique can be to intubate without relaxant using opiates to obtund the airway reflexes.
- Airway
  - Reinforced or oral RAE tube.
  - Reinforced laryngeal mask can be used as an alternative to relaxant and intubation (enabling facial nerve monitoring) or for other operations, e.g. submandibular gland excision, intra- or extra-oral short procedures. Remain vigilant as the laryngeal mask can be displaced during surgery.
- Inhalational agent and remifentanil or TIVA should enable controlled ventilation without muscle relaxants.

- Maintain deep anaesthesia without further doses of relaxant in parotid surgery.
- Facial nerve integrity is monitored intraoperatively by nerve stimulator. The excitability of indirectly evoked muscle responses by stimulation of the corresponding motor nerve is substantially more sensitive than that of direct electrical muscle stimulation. The nerve monitor indicates the muscle action potential on a monitor as well as with auditory cues.

LOCAL ANAESTHESIA

Not commonly used but if required for submandibular gland surgery, blocking of the inferior alveolar, auriculotemporal, lingual, buccal and superficial cervical plexus are required.

OTHER PROCEDURES

Transposition of parotid duct into the tonsillar fossa for treating drooling, excessive salivation due to trauma or spastic disorder. These patients may have learning difficulties. General anaesthesia with nasal intubation is usual for better visualization of duct openings.

Diagnostic and interventional sialendoscopy for ductal disorders can be carried out under local analgesia with or without sedation or general anaesthesia particularly when there are multiple and bigger stones. Avoid use of anticholinergic agents.

POSTOPERATIVE MANAGEMENT

- Simple oral analgesics including NSAIDs.
- Occasionally opiates required.
- IV fluid may be required until adequate oral intake.

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CROSS-REFERENCE

Head and neck surgery general principles, Chapter 20

PARATHYROID SURGERY

Parathyroid surgery is usually undertaken for removal of a parathyroid adenoma. Local anaesthesia, regional anaesthesia involving superficial cervical plexus blocks (with or without sedation) or more commonly general anaesthesia (GA) have been used. Local and regional anaesthesia are increasingly being employed for minimally invasive parathyroid surgery and also used for patients with marked cardiovascular disease. Surgery in patients with renal hyperparathyroidism is only indicated in cases of refractory medical management and the presence of complications of hyperparathyroidism.

Surgical approaches: full cervical exploration, limited neck dissection, minimally invasive parathyroid surgery.

PATIENT CHARACTERISTICS

Primary hyperparathyroidism is caused by excessive secretion of PTH. The most common cause is parathyroid adenoma, which accounts for 80%–90% of cases, the others being multiple gland hyperplasia or carcinoma. A family history must be taken to identify familial hyperparathyroidism, multiple endocrine neoplasia (MEN) type I and II.

Secondary hyperparathyroidism is where PTH is elevated to compensate for chronically low calcium with no intrinsic parathyroid abnormality. Vitamin D deficiency and chronic renal failure are the two most common causes.

Tertiary hyperparathyroidism is where parathyroid hyperplasia progresses and excessive PTH secretion continues despite the presence of high concentration of calcium.

Ectopic hyperparathyroidism (pseudo hyperparathyroidism) is due to secretion of PTH by tissues other than the parathyroid glands. Carcinomas of the lung, breast, pancreas, oesophagus or the kidney are common causes.

Signs and complications include:

- Skeletal muscle weakness, myopathy
- Nephrolithiasis, polyuria and polydipsia, renal failure
- Anaemia
- Peptic ulcer disease, vomiting, pancreatitis
- Hypertension, prolonged PR interval, short QT interval
- Generalized osteopaenia, bone pain, pathological fractures
- Decline in mental function, personality changes, lethargy and mood disturbances

PREOPERATIVE PREPARATION

- Full detailed preoperative assessment.
- Cardiovascular review including ECG.
- Up to date blood results.
- An adenoma may have been localised by 99 Technetium Sestamibi scanning with high-resolution ultrasonography, CT or MRI.
- Medical management of hypercalcaemia: keep well hydrated, loop diuretics, bisphosphonates, cinacalcet.
- Renal replacement therapy as required.

PERIOPERATIVE MANAGEMENT

- Principles similar to anaesthesia for thyroidectomy.
- Reinforced endotracheal tube or laryngeal mask.
- Positioning
  - Carefully to avoid pathological fractures
  - Neck extended (better access to the operating area)
  - Head up position (decreases venous congestion, reduces intraoperative bleeding)
- Standard monitoring
- Neuromuscular monitoring
- Core temperature
- Invasive monitoring depending on co-morbities
- Warming blanket and IV fluids

POSTOPERATIVE MANAGEMENT

- Simple oral analgesics.
- Avoid NSAIDs in renal patients.
- Check serum calcium at 6 and 24 hours.
- Check magnesium and phosphate levels.

COMPLICATIONS

- Hypocalcaemia
- Recurrent laryngeal nerve damage
- Haematoma

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CROSS-REFERENCES

Hyperparathyroidism, Chapter 6
Multiple endocrine neoplasia, Chapter 6
Thyroidectomy, Chapter 20

THYROIDECTOMY

Thyroidectomy may be unilateral lobectomy, isthectomy, subtotal, near total or total. Surgery is performed through a skin crease incision approximately 4 cm above the sternum.

- The recurrent laryngeal nerves must be preserved.
- Parathyroid glands should be preserved.
- Large retrosternal goitres may require a sternal split.
- Up to 5% of the population has a goitre.
- Most patients are female. Approximately 10% of nodules will be malignant.

INDICATIONS

THYROTOXICOSIS

- In the general population: 2% of females, 0.02% of males.
- Graves disease in younger women; most common cause up to 80%.
- Multinodular goitre (older patients).
- Toxic solitary nodule.
- Other causes – thyroiditis, pregnancy, drug-induced (amiodarone).

THYROID MALIGNANCY

- Papillary (80%)
  - 30–40 years old
  - 95% 10-year survival rate
- Follicular (10%)
  - May be hormone producing
  - Older population
  - 85% 10-year survival rate
- Medullary (8%)
  - Older patients
  - May be associated with multiple endocrine neoplasia
  - May produce calcitonin
  - 65% 10-year survival rate
- Anaplastic (1%)
  - Mean survival 6 months from diagnosis

COMPRESSIVE OR COSMETICALLY UNACCEPTABLE NONTOXIC GOITRE

PREOPERATIVE ASSESSMENT

- Detailed history and examination

AIRWAY

- Tracheal deviation may be marked.
- Stridor or respiratory distress, especially when supine.
- Vocal cords movement assessed by an otolaryngologist to ensure that pre-existing laryngeal nerve palsy is recognised.

CARDIOVASCULAR SYSTEM

- Hyperthyroidism can cause tachycardia, atrial fibrillation, or heart failure.
- Large goitres may obstruct venous drainage and SVC obstruction can occur with retrosternal spread.

EYES

- Lid retraction and exophthalmos mean that care is needed to protect the eyes from intraoperative drying or trauma.

OTHER CONDITIONS

- May be part of multiple endocrine neoplasia syndromes and conditions such as diabetes mellitus, hyperparathyroidism, and phaeochromocytoma must be considered.

DRUG TREATMENT IN HYPERTHYROIDISM

Patients should be clinically and biochemically euthyroid prior to surgery:

- *Carbimazole* – Inhibits iodination of tyrosyl residues in thyroglobulin.
- *Propylthiouracil* – As carbimazole but also reduces peripheral de-iodination of T4 to T3.
- β-*blockers* – Used to control cardiovascular effects. Propranolol also decreases the extra thyroidal conversion of T4 to T3.
- *Iodine* – Potassium iodide is given for 7–10 days preoperatively. Although iodine is required for normal thyroid function, excessive doses of iodine inhibit iodide binding, reduce hormone synthesis and reduce the effect of TSH. A beneficial preoperative side effect is the reduction in vascularity of the thyroid gland. In severe or recurrent disease, radioactive iodine is used to ablate the thyroid.

PREOPERATIVE INVESTIGATION

- Thyroid function tests, FBC, U&Es, calcium, magnesium and phosphate.
- ECG and echo if appropriate.
- Imaging.
- CXR and thoracic inlet views to assess airway compression or deviation.
- CT and MRI scans: with retrosternal disease or severe stridor will delineate the degree and extent of airway narrowing.

PERIOPERATIVE CARE

- Anxiolytic premedication as required.
- Usual medications.
- Standard monitoring.
- Arterial line if significant pre-existing cardiovascular disease.
- Temperature monitoring
  - Increasing temperature can be an indicator for thyrotoxic crises.
  - Hypothermia in hypothyroidism as a result of decreased basal metabolism.
- Securing airway
  - Intravenous induction and controlled ventilation is usually used.
  - If difficult airway expected, options include: inhalational induction, videolaryngoscope, fibreoptic scope, ventilating rigid bronchoscope, awake tracheostomy and even cardiopulmonary bypass if significant retrosternal spread.
  - Reinforced endotracheal tube.
  - Careful neck extension provides surgical access. Recheck tube position when patient repositioned.
- Head-up tilt reduces venous engorgement.
- Protect the eyes as drapes will be placed over the head; extra care if exophthalmos present.
- Surgeons may wish to use a nerve stimulator to locate the laryngeal nerves. In such cases, muscle relaxation should not be used once intubation is completed. 'EMG endotracheal tube' or invasive techniques using needle electrodes for monitoring the recurrent laryngeal nerve are available.
- Supplementation of β-blockade may be needed as manipulation of the thyroid may release more thyroid hormone.
- Prior to wound closure – normotension, head-down tilt, a valsalva manoeuvre will assist in assessing haemostasis.

Some surgeons request direct laryngoscopy to assess vocal cord movement at the end of surgery. In some centres, the endotracheal tube is replaced by a laryngeal mask at the end of surgery.
- Smooth emergence.
- A fibre-optic nasoendoscope can be used to assess vocal cord movement.

POSTOPERATIVE CARE AND COMPLICATIONS

- Postop analgesia: LA, paracetamol, weak opiates, NSAIDs if no contraindications.
- Antiemetics as retching may increase risk of haematoma.
- Removal of thyroid does not result in resolution of thyroxicosis as half-life of T4 is 7 days.

COMPLICATIONS

- Hypocalcaemia
  - Check serum calcium to ensure normal parathyroid function. Tetany or low serum calcium requires calcium supplementation by the intravenous or oral route.
- Recurrent laryngeal nerve palsy
  - Temporary 3%–4%, permanent <1%. Will cause the affected vocal cord to lie in adduction. Symptoms include hoarse voice, poor cough, dyspnoea, stridor.
  - Bilateral palsies can result in complete airway obstruction, requiring urgent reintubation.
- Haematoma
  - Early detection of signs of postoperative haemorrhage.
  - Incidence 0%–2%, usually early within 4 hours.
  - Can be life-threatening.
  - Neck haematoma and surgical tracheal retraction may cause laryngeal oedema.
  - Immediate removal of wound clips and sutures will decompress the neck and trachea prior to urgent evacuation and haemostasis.

- Inhalational or intravenous induction are suitable techniques. A smaller endotracheal tube may be required.
- Surgeons should be present in case of the need for an emergency tracheostomy.
- Induction in theatre.

RARE COMPLICATIONS

- *Tracheomalacia* following resection of long-standing large compressive retrosternal thyroid masses. Dynamic tracheal collapse may necessitate prolonged intubation.
- *Pneumothorax* occasionally following extensive and difficult retrosternal resection.
- *Thyroid storm* is rare due to use of antithyroid medication. It occurs due to uncontrolled release of thyroxine in a thyrotoxic patient and may be triggered by acute illness, surgery or trauma. Signs include severe hyperpyrexia, tachycardia, hypertension, arrhythmias, vomiting, diarrhoea and altered mental state. Intraoperatively this may mimic malignant hyperpyrexia. It may occur postoperatively. It can prove fatal with reported mortality rates of 10%–75%. Patients should be managed supportively in ICU.

  Treatment includes:

- Supplemental oxygen.
- Temperature monitoring, antipyretics and active cooling.
- IV fluids.
- β-blockade
  - Propranolol (inhibit peripheral conversion T4 to T3).
  - Esmolol for acute management (short acting, β1 selective).
- Anti-thyroid drugs
  - Carbimazole or propylthiouracil orally or via a nasogastric tube.
- Dexamethasone.
- Magnesium if hypertension and/or arrhythmia.
- Dantrolene use has been reported in these patients.

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Hyperthyroidism, Chapter 6
Hypothyroidism, Chapter 6
Head and neck surgery – general considerations, Chapter 20

Plastic surgery

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BRIAN J POLLARD AND GARETH KITCHEN

ANAESTHESIA FOR BURNS SURGERY

PATIENT CHARACTERISTICS

- All age groups are affected but extremes of age are common. Children up to 4 years of age form 20% of patients.
- Males are predominant other than the elderly.
- Teenagers are often injured as a result of illicit activity involving, e.g. electrocution/petrol.
- There may be other associated injuries.
- There may be associated comorbidities, e.g. epilepsy, psychiatric disturbances, history of substance abuse.
- Consider non-accidental injury in every child with thermal injury.
- Adult burns are usually caused by flame and paediatric burns by scalds.

PROCEDURES

- Management of major burns and burns surgery should occur only in specialized regional units.
- Anaesthesia may be required for tracheostomy, escharotomy, skin grafts, plastic procedures, dressing changes or surgery for associated injuries.
- Early surgery improves cosmetic result and removes necrotic tissue.
- Burns are classified by area (% of total body surface area [TBSA] burned) and depth (superficial; shallow; deep dermal; full thickness; full thickness with deep tissue involvement). A major burn in an adult is defined as greater than 20% TBSA and in children greater than 10% TBSA.
- Procedures may be lengthy, e.g. 1–2 hours for routine burns dressing change.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Has there been a history of smoke inhalation?
- Is further resuscitation required?
- Full blood count, urea and electrolytes, chest X-ray, arterial blood gases, clotting screen and urine output as a minimum.

- Blood glucose particularly important in children.
- Check adequate availability of blood and blood products.
- Check for comorbidities; assess, investigate and optimize as indicated.

THEATRE PREPARATION

- Burns theatre may be a relatively isolated site.
- If transferring patients from a Burns Unit or ICU to theatre full mobile facilities are required for transfer.
- Large burns require IPPV. Ventilated burns patients may be highly PEEP dependent. Ensure the theatre ventilator can provide the necessary requirements.
- Anticipate significant blood and fluid loss.
- Warmed rapid infusion systems and forced warmed air blankets.
- Theatre prewarmed to a thermo neutral temperature (approximately 30°C).
- Ideally involve two surgical teams as well as two anaesthetists.

PREMEDICATION

- Pre-emptive analgesia.
- Anxiolysis may be required particularly in children.

PERIOPERATIVE MANAGEMENT

- Monitoring
  - ECG
  - Oximetry
  - Blood pressure
  - Capnography
  - CVP
  - Respiratory gas analysis
  - Central temperature
  - Urine output
- Particular considerations
  - Placement of ECG electrodes may be potentially difficult.
  - Vasoconstriction/burned peripheries may limit available sites for oximeter probe.

- Pulse oximetry cannot distinguish between carboxyhaemoglobin (COHb) and oxyhaemoglobin. High readings will be seen if COHb is present.
- There is increased dead-space in inhalation injury so $ETCO_2$ may not reflect $PaCO_2$.
- Regular blood gas analysis may be needed so an arterial line is useful.
- Central venous access may be difficult and pose infection risk.

ANAESTHETIC TECHNIQUE

- If there is any possibility of airway oedema consider awake fibre-optic intubation or inhalational induction.
- Repeated fasting required for repeated surgical procedures may interfere with nutritional goals. Patients are hypermetabolic. Consider modification of fasting guidelines or parenteral nutrition.
- Anticipate major blood loss. Measure Hb intraoperatively.
- Anticipate coagulation problems. Fluids dilute clotting factors.
- Regional techniques are generally not useful in major burns due to infection risk, coagulopathy and difficulty in blocking sufficient area.
- Avoid succinylcholine after 24 hours and arguably up to 1 year postoperatively due to exaggerated hyperkalaemic response.
- Induction with propofol is popular and TIVA may be used.
- Ketamine maintains blood pressure and has analgesic properties.
- Choice of volatile does not influence outcome.
- Resistance to nondepolarising relaxants may develop after several days and persist for up to 8 weeks.
- Epinephrine containing solutions may be applied topically or subcutaneously to decrease bleeding at excision and donor sites.
- EMLA (or similar) may be applied under dressings.

- Dose requirements of aminoglycosides, cephalosporins and beta-lactams are altered due to increased clearance.
- Meticulous attention to positioning is essential.
- Postoperative analgesia – opiate infusion or PCA is recommended.

DRESSING CHANGES

- General anaesthesia may be required.
- Entonox is useful for short procedures if oxygen requirements are less than 50%.
- Ketamine is popular in some centres.
- Boluses or infusion of short-acting opioids, e.g. alfentanil, fentanyl, remifentanil.
- Sedation using propofol (possibly in combination with remifentanil) by target-controlled infusion.

POSTOPERATIVE MANAGEMENT

- Analgesia:
  - Opioids are usually required.
  - Intramuscular or subcutaneous doses are unpredictable in absorption.
  - Continuous infusion – caution with potential accumulation.
  - PCA requires use of hand and adequate conscious level.
- Beware of altered conscious state, e.g. alcohol, drugs.
- Tolerance may develop.
- Caution with non-steroidal analgesics – problems with renal dysfunction and peptic ulceration.
- Procedural pain associated with dressing changes may be severe.
- Early nutritional supplementation essential, e.g. 3000–5000 calories per day in adults. Seek advice from dietician.

OUTCOME

- Survival of major burns is age related and decreases over the age of 30.
- Worse in the presence of respiratory injury and increasing burn size.

- There may be long-term psychological disturbance and chronic disability.
- Subsequent hospital admissions may be needed for reconstructive surgery to aid functional recovery.

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CROSS-REFERENCES

Prolonged anaesthesia, Chapter 28
Blood transfusion, Chapter 30
Fluid and electrolyte balance, Chapter 30
Postoperative pain management, Chapter 30

COSMETIC SURGERY

PROCEDURES

- Operations which revise or change the appearance, colour, texture, structure or position of bodily features to achieve what patients perceive to be more desirable.
- The four most common procedures relate to surgery on the breast, eye, and nose and liposuction.

- Usually body surface surgery.
- Frequently multiple procedures undertaken.
- Often ambulatory with short hospital stay.
- Increasing popularity with awareness of availability.
- Most cosmetic surgery is not available in NHS practice.
- Increasing presentation of post-bariatric surgery patients.

PATIENT CHARACTERISTICS

- Patients have high expectations.
- A well-informed patient group via media-driven culture.
- Often seeking improved self-confidence and self-esteem.
- Generally young or middle-aged.
- Increasing numbers of patients post-bariatric surgery.

PREOPERATIVE ASSESSMENT

- May require preoperative psychological assessment by trained professional.
- Routine preoperative assessment with investigations as indicated.
- Treatment usually sought on the basis of want rather than need; therefore, ethical considerations may apply in patients with comorbidities.
- Venous thrombo-embolism prophylaxis in prolonged cases.
- A two-stage consent procedure allowing a 'cooling off' period may be performed.
- Premedication generally not required.
- Often performed as a day-case procedure so appropriate advice is necessary.

THEATRE PREPARATION

- Avoid hypothermia using active warming devices and blood warmer.
- Different positioning of equipment may be needed, e.g. for facial surgery.

PERIOPERATIVE MANAGEMENT

Monitored anaesthetic care or conscious sedation may be appropriate for patients undergoing compatible procedures. Usually the surgeon will infiltrate the surgical field with local anaesthetic. Patients should have the same preoperative screening, receive full monitoring and meet the same discharge criteria as those undergoing general anaesthesia. Formal training in sedation is mandatory for those undertaking this area of practice.

SURGERY ON THE FACE AND NECK

- Examples include facelift, brow lift, blepharoplasty, neck lift, rhinoplasty.
- A laryngeal mask may provide adequate airway control. If head movement and turning is anticipated or blood/fluid may enter the mouth, a tracheal tube is advised.
- Maintaining stable blood pressure in the low to normal range minimizes perioperative bleeding and reduces bruising postoperatively.
- Throat packs are needed for rhinoplasty and procedures where an intra-oral incision has been made. Theatre care pathways need to include a check for removal.
- A smooth extubation without coughing or bucking is essential to minimize increased bruising and/or development of haematoma.

SURGERY FOR ABDOMINOPLASTY

- Patients may have increased BMI or be post-bariatric surgery. Both groups have a significant risk of perioperative aspiration of gastric contents.
- Preoperative assessment requires detailed assessment of pulmonary function.

LIPOSUCTION

- Removes fat and improves contours (usually buttocks, thighs and abdomen).
- Large volume liposuction of more than 1500 mL of aspirate is associated with a

high level of morbidity and mortality and is generally avoided.

- Prolonged procedures involve significant fluid shifts. Up to 25%–30% of aspirate may be blood.
- Surgeons generally utilize a 'tumescent' technique where a volume of fluid containing local anaesthetic and adrenaline in excess of the volume of fat to be aspirated is injected as a 'wetting' agent prior to liposuction. Up to 50% of this fluid may remain at the end of the procedure.
- Good communication between surgeon and anesthetist is essential to ensure acceptable formulation of injectate.
- Generally considered a safe procedure with rare complications including pulmonary fat embolism.

POSTOPERATIVE MANAGEMENT

- Postoperative pain is usually easy to manage initially as for most procedures the surgeon will have infiltrated local anaesthetic during the procedure.
- Postoperative nausea and vomiting needs to be anticipated and managed particularly in young, anxious patients undergoing breast surgery.

OUTCOME

- Patients usually mobilise early and leave hospital within 48 hours and there should be a clear emergency readmission policy.
- There are no prospective studies defining predictors of poor outcome.
- Putative factors associated with poor patient satisfaction with results of surgery are known to be youth, male gender, depression, and psychological disorders.

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CROSS-REFERENCES

Obesity, Chapter 4
Operations on the nose, Chapter 19
Free-flap surgery, Chapter 21
Day-case surgery, Chapter 25
Local anaesthetic toxicity, Chapter 30
Thrombotic embolism, Chapter 30

FREE-FLAP SURGERY AND RELATED PROCEDURES

PATIENT CHARACTERISTICS

- Sections of skin, with or without underlying vascular supply (and possibly nerve supply) are moved from one place to another in the same patient.
- Flaps may vary greatly in size, shape and site.
- Transplantation of whole digits or limbs may be undertaken.
- Almost any age group and ASA status may be encountered.
- Patients with malignant disease may require free-flap surgery to cover a defect from a resected area.
- Trauma may be the cause of amputated digits and the procedure may have to be managed as an emergency.

PREOPERATIVE ASSESSMENT

- Assessment of cardiac and respiratory reserves.
- Ability to safely cope with an induced hyperdynamic circulation.
- Assessment of the airway and the likelihood of difficult intubation if surgery is anticipated around the head and neck area or that area is involved in trauma.
- Possible need for elective tracheostomy.
- DVT prophylaxis.
- Blood transfusion availability.
- Antibiotic prophylaxis.
- Prepare for a prolonged procedure (several hours is not unusual).
- Discussion with the surgeon regarding accessibility of a particular part of the body as the donor site.
- Anxiolytic premedication may be required.

THEATRE PREPARATION

- Adequate skilled manpower for positioning patients.
- Patient supports and padding to pressure areas (potential long operation).
- Anaesthetic room and theatre temperature and humidity carefully controlled.
- Warming blanket, warm intravenous fluids, warm air overblanket.
- Equipment for difficult intubation, including a tracheostomy set if anticipated.

MONITORING

- ECG
- BP (invasive advisable if a prolonged procedure)
- SpO_2
- Central venous pressure (or transoesophageal Doppler)
- $ETCO_2$
- Inspired oxygen and inhalational agent
- Core and peripheral temperature
- Neuromuscular blockade
- Urinary catheter if prolonged operation
- Blood loss

ANAESTHETIC TECHNIQUE

- General anaesthesia is preferred.
- The patient must be maintained absolutely still for microsurgery.
- Adjuvant local anaesthetic techniques may promote flap blood flow.
- Induced hypotension may be requested for part of the procedure.
- Following arterial and venous anastomosis of the flap in its new location blood flow through the flap should be promoted. This can often be achieved by using a suitable inhalational agent with vasodilatory effects.
- Adequate hydration with crystalloid infusion is essential.
- Replace blood loss as it occurs.
- Maintain optimum fluid balance (CVP or TOE).
- Maintain haematocrit at around 30%.
- Meticulous attention to maintaining body temperature.
- Provide adequate analgesia.
- Maintain normocapnia and good oxygenation.
- Vasodilation may be requested – sodium nitroprusside, glyceryl trinitrate, phenoxybenzamine, phentolamine and calcium channel blockers have all been used with success. Whichever drug is used, it is desirable for its vasodilatory effect to last into the postoperative period.
- An operating microscope is required for anastomosis of blood vessels and nerves.
- Two teams may be needed as the procedure can be very prolonged and last 6–12 hours or even longer.

POSTOPERATIVE MANAGEMENT

- HDU care is recommended for continued monitoring of circulatory control and adequate analgesia.
- Colloid solution is often infused at up to 7 mL kg^{-1} day^{-1} for 3 days.
- The flap should be observed regularly for viability and may be monitored with

impedance or photoplethysmography or laser Doppler flowmetry.

- Common complications include flap arterial thrombosis and haematoma requiring re-exploration.

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CROSS-REFERENCES

Difficult airway – management, Chapter 26
Prolonged anaesthesia, Chapter 28
Blood transfusion, Chapter 30
Complications of position, Chapter 30
Fluid and electrolyte balance, Chapter 30

PAEDIATRIC PLASTIC SURGERY

GENERAL FEATURES

- Children of all ages may present for corrective surgery, from the neonate to the adolescent.
- Operations range from minor day-case procedures to major reconstructive surgery.
- Most operations are elective. Children with features of a recent upper respiratory tract infection should be delayed for a minimum of two weeks.
- Some children may present for surgery with syndromes associated with features of anaesthetic significance (e.g. difficult intubation).
- Many children require multiple surgical procedures. It is important to establish a rapport with the child and parents. Preoperative preparation is essential and oral premedication may be required.
- Heat loss is a major problem, especially for smaller children undergoing major surgery. Maintain theatre temperature at 22°C–24°C and avoid unnecessary exposure of the child. Use a warming mattress and/or convective warm air heating blanket and/or overhead radiant heater. Warm and humidify anaesthetic gases. Warm intravenous fluids. Temperature measurement is essential.
- Operations on the skin and superficial tissues are painful. Regional techniques including local infiltration, peripheral nerve blocks and caudal and epidural blockade facilitate a lighter plane of anaesthesia and provide a smoother recovery.
- Local infiltration with adrenaline is used routinely by many plastic surgeons; up to 10 µg kg^{-1} can be used safely. Avoid inhalational agents that are known to predispose to cardiac rhythm abnormalities in these cases.

CLEFT LIP AND PALATE

- A relatively common condition with an incidence of 1 in 600 live births. It is an isolated defect in 90% of cases.
- Isolated cleft palate (1 in 2000 live births) is often associated with other congenital abnormalities that may be of anaesthetic significance (Table 21.1).
- A range of deformities is possible.
- Average age of correction
  - Cleft lip: 8 to 12 weeks (neonatal repair is decreasing in popularity)
  - Cleft palate: 6 to 12 months (dependent upon the size of the cleft)
- These children usually have multiple problems and their clinical care requires a combined team approach.

Table 21.1 Syndromes of anaesthetic significance associated with cleft palate

Syndrome	Significant features
Klippel–Feil	15% associated with cleft palate
	Short, webbed neck
	Fused cervical vertebrae
	Congenital cardiac disease
Pierre Robin	80% associated with cleft palate
	Severe micrognathia
	Glossoptosis
	Congenital cardiac disease
	Intubation easier with age
Shprintzen	90% associated with cleft palate
(Velocardiofacial)	Facial dysmorphism
	Learning difficulties
	Congenital cardiac disease
Treacher Collins	28% associated with cleft palate
	Micrognathia
	Malar and maxillary hypoplasia
	Ear malformations
	Intubation more difficult with age

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Assess the airway. Upper respiratory tract infection will increase the risk of airway complications and compromise wound healing.
- Exclude other associated conditions (e.g. cardiac disease).
- Blood should be available.

PREMEDICATION

- Usually unnecessary.
- Avoid opioid or sedative premedication in children under 6 months or if airway is compromised.
- If difficult airway is anticipated, give atropine 20 µg kg^{-1} L M.

MONITORING

- ECG
- Noninvasive BP
- SpO$_2$
- ETCO$_2$ and inhalational agent
- Nerve stimulator
- Core temperature
- Fluid balance
- Blood loss

INDUCTION OF ANAESTHESIA

- Inhalational or IV induction. If there is a suspected airway problem, use sevoflurane in 100% oxygen. Use of CPAP will assist maintenance of the airway.
- Intubate deep with child breathing spontaneously or after a muscle relaxant once a safe airway has been established.
- Care should be taken to prevent the laryngoscope blade lodging in the cleft. A roll of gauze may be used to fill the defect or a lateral approach with the laryngoscope can be used.
- A preformed RAE tube may become kinked by the gag. Using a reinforced tube will resist compression but it is more difficult to secure at the correct length.
- Throat pack.

MAINTENANCE OF ANAESTHESIA

- Controlled ventilation is appropriate. Intravenous fentanyl (1–2 µg kg^{-1}) can be used to supplement anaesthesia but should be given cautiously in neonates or in the presence of upper airways problems.
- Local infiltration produces good analgesia, an improved operating field and reduced blood loss.
- Infraorbital nerve block for cleft lip repair or nasopalatine and palatine block for cleft palate surgery is effective.

POSTOPERATIVE MANAGEMENT

- The patient should be extubated only when fully awake, breathing adequately and in the tonsillar position. Smooth pain-free emergence reduces crying and the risk of postoperative bleeding.

- Postoperative airway obstruction can be a particular problem in cleft palate repair and a nasopharyngeal airway or stent may be required in certain patients.
- The insertion of a tongue stitch to allow the tongue to be pulled forward is a useful, although uncommonly used, technique.
- Postoperative analgesia includes regular paracetamol and codeine phosphate with feeding commonly possible within a few hours of surgery. Ibuprofen may be added. Ketamine may be useful as a non-respiratory depressant analgesic in the early stages of postoperative management.

CRANIOFACIAL SURGERY

- Major reconstructive surgery involving the facial skeleton, facial soft tissues and cranial shape. Children with craniosynostosis often have associated syndromes and other congenital abnormalities of importance to anaesthesia. These should be assessed preoperatively.
- Problems include difficult intubation, major blood loss and hypothermia.
- Direct arterial and central venous pressure measurement (via the femoral vein) are required.

HAEMANGIOMA

- May be a simple peripheral procedure.
- If involving the face or oral cavity this may be a complicated procedure with associated airway problems, major blood loss and potential for air embolism.
- A cutaneous haemangioma around the face and neck may be associated with a subglottic haemangioma. There is a risk of bleeding into mouth and airway if this is subject to trauma.

CYSTIC HYGROMA

- These are multiloculated cystic swellings. If present in the neck, it may invade the oropharynx and tongue. It can present

as an upper airway obstruction in the neonate.
- Intubation can be hazardous; spontaneous ventilation must be maintained until intubation has been achieved. Severe cases will require tracheostomy.
- Postoperative problems include bleeding and respiratory obstruction.

OTOPLASTY

- Correction of prominent ears is often associated with a high incidence of PONV, which can last up to 48 hours and an antiemetic will be required.
- A procedure including local infiltration or nerve block is helpful.

HYPOSPADIAS REPAIR

- Anatomical correction of congenital abnormality of male urethra.
- Usually undertaken in an infant.
- Spontaneous or controlled ventilation can be used according to length of procedure.
- Caudal block with 0.5–1 mL kg^{-1} of 0.25% bupivacaine provides excellent analgesia that can be extended by adding clonidine, diamorphine or ketamine.

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CROSS-REFERENCES

Infants and children, Chapter 25
Paediatric airway, Chapter 26
Paediatrics – overview, Chapter 24

PERIPHERAL LIMB SURGERY

PROCEDURES

- May be elective or emergency as a result of trauma.
- May include surgery for correction of acquired deformities, e.g. Dupuytren's contracture, carpal tunnel syndrome and excision of tumours or skin lesions.
- Reimplantation of digits or limbs following traumatic amputation may be lengthy procedures.

PATIENT CHARACTERISTICS

- Almost any age or ASA status.
- In children, early correction of congenital malformations may be associated with other anomalies.
- May have associated trauma or alcohol/substance abuse injuries.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- If trauma, exclude or assess any coexisting injuries.
- Consider implication of other comorbidities, e.g. rheumatoid arthritis.
- Investigations as indicated.
- ECG, chest radiology or other investigations as indicated.
- Sickle screen in at-risk populations if tourniquet is to be used.
- Premedication if indicated.
- Discuss procedure and details/implications in nerve block to be used.
- Preoperative analgesia may be needed in recent trauma.

MONITORING

- ECG
- BP (invasive advisable if a prolonged procedure)
- SpO_2
- $ETCO_2$
- Inspired oxygen and inhalational agent
- Core and peripheral temperature
- Neuromuscular blockade
- Urinary catheter if prolonged operation
- Blood loss

ANAESTHETIC TECHNIQUE

- A regional block with or without sedation is usually the most appropriate technique.
- Continuous peripheral nerve blockade is popular for postoperative analgesia.
- Ultrasound-guided regional nerve block facilitates should be available.
- Establish venous access prior to insertion of a block.
- In reimplantation surgery associated sympathetic block improves perfusion.
- A regional block should be inserted before induction of general anaesthesia if that is also to be used.
- General anaesthesia alone or supplemented by a regional block is useful in the following situations:
    - Paediatric patients
    - Prolonged procedures
    - Uncooperative patients, e.g. learning difficulties, dementia

TOURNIQUET

- Use of a tourniquet is common in peripheral limb surgery.
- Maximum pressure in the leg should be 300 mm Hg.
- Maximum pressure in the arm should be 200 mm Hg.
- Maximum tourniquet time should be 1 to 2 h.
- Its use should be avoided in sickle disease: if use is essential, the advice of a haematologist should be sought.

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CROSS-REFERENCES

Sickle cell syndrome, Chapter 7
Day-case surgery, Chapter 25
Prolonged anaesthesia, Chapter 28
Local anaesthetic toxicity, Chapter 30
Local anaesthetic blocks (chapter), Chapter 29

Orthopaedics

22

ROBERT PETER LOVERIDGE

ANAESTHESIA FOR ORTHOPAEDIC SURGERY: GENERAL PRINCIPLES

Patients tend to be young (trauma and fit sport participants) or elderly (trauma and elective arthroplasty). Prevalent problems in this latter group include osteoarthritis, rheumatoid arthritis and the associated immunosuppressive medications, comorbidity and polypharmacy.

PREOPERATIVE CONSIDERATIONS

- Liaise with surgeon and review anticoagulation if planning on undertaking regional technique.
- Exercise tolerance often limited by musculoskeletal pathology so assessment of cardiorespiratory fitness can be challenging.
- Patients having arthroplasty will often be on enhanced recovery after surgery (ERAS) programs.

INTRAOPERATIVE MANAGEMENT

- Antibiotic prophylaxis often required.
- Blood loss can be significant.
- Bone cement implantation syndrome (BCIS) can cause significant cardiovascular compromise.

POSTOPERATIVE MANAGEMENT

- High risk of thromboembolic disease so prophylactic measure need to be considered. This can influence decisions relating to regional anaesthesia.
- Excellent analgesia can aide patient mobility and functional recovery.
- NSAIDs may affect bone healing and their use should be discussed with surgeon.

SPECIFIC PROBLEMS

HAEMORRHAGE

- Haemorrhage can be significant and may be increased in certain operations (revision arthroplasty, pelvic surgery) or in certain pathologies (Paget disease, metastatic disease).
- Tranexamic acid at doses over 1000 mg during hip and knee arthroplasty has been shown to

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reduce the need for blood transfusion while not increasing the risks of thromboembolic disease or acute kidney injury.

INFECTION

- Antibiotic prophylaxis is standard in orthopaedic implant surgeries to avoid surgical site infections. Each hospital will have specific antimicrobial guidelines.

VENOUS THROMBOEMBOLISM

- Orthopaedic patients are at specific risk due to comorbidity, tissue trauma, immobility and major surgery causing dehydration and hypercoagulability.
- Fatal pulmonary embolism may occur in up to 1.7% of patients after knee arthroplasty and 2% patients after hip arthroplasty.
- Delicate balance exists between the risk of venous thromboembolism and the risk of bleeding.
- Multimodal approach of early mobilisation, pharmacological and mechanical prophylaxis should be used.
- Incidence can be reduced by using central neuraxial blockade.

TOURNIQUETS

- Commonly used to reduce blood loss and provide a bloodless surgical field.
- Pneumatic tourniquets are used which are set to an exact pressure above systolic blood pressure.
- Minimal tourniquet time should be the aim. The surgeon should be notified at 60 minutes; 120 minutes is the maximum.
- After 30–60 minutes a rise in respiratory rate, heart rate and blood pressure may be noted, even if using a peripheral regional anaesthetic technique. This is ischaemic mediated 'tourniquet pain'. These physiological derangements are often resistant to opioids and will subside with tourniquet release.
- Can be associated with soft tissue damage, neural injury, VTE, pain and reperfusion injury.

THERMOREGULATION

Heat loss is common in orthopaedic procedures due to the following:

- Patients at extremes of age
- Laminar airflow present in theatre
- Use of regional anaesthesia
- Use of cold irrigation fluid during arthroscopy
- Administration of cold IV fluids/blood
- Cooler theatre atmosphere for surgeon comfort if wearing 'space suit'
- Haemorrhage and blood transfusion

Inadvertent perioperative hypothermia can increase the risk of postoperative wound infection and the need for blood transfusion. Therefore, active warming using warming mattresses and forced air warmers is important as well as warmed IV fluids and joint irrigation.

REVISION SURGERY

Although essentially the same as primary arthroplastic surgery, revision surgery can be associated with:

- Increased duration of surgery
- Increased blood loss
- Increased postoperative pain

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CROSS-REFERENCES

Regional anaesthetic techniques, Chapter 29
The elderly, Chapter 25

HIP AND KNEE ARTHROPLASTY

Total hip arthroplasty (THA) involves replacement both of the acetabulum and head/neck of the femur. Hemiarthroplasty involves only replacing the femoral head. Both procedures are to treat the pain and loss of function usually due to chronic osteoarthritis.

Total knee arthroplasty (TKA) involves the removal and replacement of the articular surfaces of the tibia and femur, with metal prostheses. Unicompartmental knee replacement (hemiarthroplasty or Oxford knee replacement) can be performed in patients with single compartment arthritis. This has been found to have a lower morbidity and mortality rate but at the expense of a threefold revision risk.

Revision procedures are becoming more prevalent, as patients approach the natural lifespan of various prostheses and more prostheses are inserted.

PATIENT CHARACTERISTICS

Usually aged over 50 years, and can be frail and elderly, with or without comorbidity.

ENHANCED RECOVERY AFTER SURGERY (ERAS)

ERAS is a collection of multidisciplinary, evidence-based procedure-specific interventions that aim to improve preoperative care, reduce the physical stress of the operation, and decrease postoperative discomfort, thereby leading to improved postoperative mobility and earlier supported discharge.

PREOPERATIVE MANAGEMENT

- Full history and examination.
- Relative immobility may mask myocardial ischaemia, necessitating alternative functional assessment.
- Anaemia is common: FBC, U+Es and group and save required.

INTRAOPERATIVE MANAGEMENT

MONITORING

- Routine AAGBI recommended monitoring.
- Additional:
  - Invasive BP for patients at high risk of cardiovascular complications.
  - Oesophageal Doppler in patients under general anaesthesia, undergoing revision surgery.

ANAESTHETIC MANAGEMENT

- The relative advantages of general versus neuraxial and regional anaesthesia continue to be debated.
- With ERAS there is now a trend to prioritise postoperative mobility over analgesia, resulting in the proliferation of 'low-dose' spinal anaesthetic techniques with no or short-acting intrathecal opioids.
- Perioperative administration of non-opioid analgesia (e.g. ketamine, dexamethasone, gabapentin/pregabalin) may reduce pain and opioid requirements.
- Tranexamic acid (10–15 mg/kg) has been shown to reduce blood loss and transfusion requirement.
- Restrictive use of IV fluids without dehydration is advocated.

BONE CEMENT IMPLANTATION SYNDROME (BCIS)

BCIS is common after the insertion of cemented prostheses and occurs in up to 19% of cases with reactions involving severe hypoxia and/or hypotension, or cardiovascular collapse in 2.7% and 0.5% of operations, respectively.

POSTOPERATIVE MANAGEMENT

- Multimodal analgesia – paracetamol, NSAIDs, non-opioids and opioid analgesics.
- High-volume periarticular infiltration of low concentration local anaesthetics by the surgeon can be used as a single dose or followed by continuous infusion. The evidence supporting this technique is debatable.
- Prophylaxis and treatment of PONV.

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CROSS-REFERENCES

KNEE ARTHROSCOPY

Knee arthroscopy is a commonly performed procedure involving the insertion of a rigid, fine-bore arthroscope and surgical instruments through small, periarticular incisions into a joint cavity. It is performed as a diagnostic tool, to assist the removal of tissue or debris, or to repair ligamentous injury as a definitive intervention. Compared to open procedures, arthroscopy improves perioperative outcome (reduced pain, blood loss, infection rates) and reduces inpatient length of stay. Over 90% of knee arthroscopies are performed as day-case procedures.

PATIENT CHARACTERISTICS

- Young, fit sportsmen and women presenting for damage assessment and/or cartilage repair after injury.
- Older patients presenting for diagnosis and assessment of osteoarthritic joint damage prior to definitive arthroplasty.

PREOPERATIVE MANAGEMENT

- Older patients may have significant comorbidity that requires evaluation. They may not be eligible for day-case surgery.
- Patients can be premedicated with paracetamol and an NSAID if not contraindicated.

INTRAOPERATIVE MANAGEMENT

- General anaesthesia or spinal anaesthesia.
- Laryngeal mask and IV opioid is routine.
- Supine with operative knee flexed to around 90°.
- Benefit of regional anaesthesia (fascia iliaca block, femoral nerve block, fascia lata block or 3 in 1 block) is questionable. Femoral block may delay return to function in adolescents undergoing cruciate ligament repair.

POSTOPERATIVE MANAGEMENT

- Avoid morphine due to incidence of PONV in day cases.

INTRA-ARTICULAR LOCAL ANAESTHETIC

Some surgeons may infiltrate local anaesthetic into the joint cavity. There has been some suggestion that this practice can inhibit cartilage synthesis or cause inflammation or chondrolysis. The experimental data suggests this is worse with continuous infusions of local anaesthetic as opposed to single doses and when epinephrine is used as an adjunct.

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CROSS-REFERENCES

The elderly, Chapter 25
Regional anaesthesia, Chapter 29
Spinal anaesthesia, Chapter 29

REPAIR OF FRACTURED NECK OF FEMUR

Hip fracture is the most common reason for a frail older person to require anaesthesia, and although the prevalence of hip fracture is reducing, the increased age of the UK population is contributing to an increased incidence of hip fractures. The 30-day mortality is 8.2% and morbidity following hip fracture is high with only 46.2% of those admitted from home able to return within 30 days.

The majority of fractures are treated surgically. This is the best way to provide analgesia and reduces the complications associated with the prolonged immobility of conservative management. Surgery should occur within 48 hours of hospital admission and be undertaken by appropriately experienced surgeons and anaesthetists. Surgical treatment is by either internal fixation or arthroplasty. Fixation can be achieved by using pins alone, with a side plate or by the use of an intramedullary nail inserted into the femoral head.

PATIENTS

The mean age is 83 years and is negatively skewed with incidence peaking in the 85–89 age group. Female:male ratio is 3:1; the vast majority are ASA grade II-III. Approximately 84% have at least one comorbidity with 26% having three or more comorbidities. The most common is hypertension (55%). Polypharmacy is common.

PREOPERATIVE MANAGEMENT

Most patients are admitted via a fast-track admission pathway which should provided guidance for the provision of IV fluid administration and analgesia. Regional anaesthetic techniques such as the fascia iliaca compartment blockade (FICB) have been shown to improve pain control, decrease opioid demand and

Table 22.1 Reasons for which it may be acceptable to delay surgery for a short period in order to investigate and treat, according to the AAGBI

Haemoglobin concentration <8 g $\times$ dL^{-1}
Plasma sodium concentration <120 or >150 mmol $\times$ L^{-1}
Plasma potassium concentration <2.8 or >6.0 mmol $\times$ L^{-1}
Uncontrolled diabetes mellitus
Uncontrolled or acute onset left ventricular failure
Correctable cardiac arrhythmia with a ventricular rate >120 min^{-1}
Chest infection with sepsis
Reversible coagulopathy

prevent delirium in certain patients and are recommended in NICE guidelines. Care should also be shown towards patient warming and pressure care.

Preoperative blood loss can be significant and cause anaemia, especially if the fracture is subcapital, transcervical or basicervical. This is because the blood supply to the femoral head is interrupted. Intertrochanteric and subtrochanteric fractures tend to preserve this.

Patients may benefit from preoperative orthogeriatric input. In addition to the major systems, the patient should be assessed with regard to musculoskeletal abnormalities, skin condition, pressure areas, dentition and hearing aids.

Full blood count, urea and electrolytes should be requested routinely along with an ECG. Request coagulation studies and chest X-ray if indicated.

Historically surgery was delayed while comorbidities were investigated and optimised. AAGBI have suggested a short list of reasons for which it may be acceptable to delay surgery for a short period in order to investigate and treat (Table 22.1).

Paracetamol should be routinely prescribed as a premedicant. Opioids should be used with caution and NSAIDs are relatively contraindicated.

PERIOPERATIVE MANAGEMENT

Patients should be managed on protected trauma lists, by a consultant-delivered service that operates separately from general emergency lists. These should be provided 7 days a week.

Whichever anaesthetic technique is used, it is crucial that hypotension is avoided.

Meta-analysis has failed to show a significant difference in outcome between regional and general anaesthesia for hip fracture surgery in terms of 30-day mortality, pneumonia, myocardial infarction, cerebrovascular accident, delirium, congestive cardiac failure, acute kidney injury, pulmonary embolism, blood transfusion, length of surgery or length of hospital stay (Table 22.2). However, when anticoagulants were not used for prophylaxis of deep vein thrombosis, the incidence of this was found to be lower with regional anaesthesia.

AAGBI recommends that neuraxial anaesthesia is considered for all patients. Spinal anaesthesia should be administered using hyperbaric bupivacaine (<10 mg) with the patient positioned laterally (bad hip down). Coadministration of intrathecal opioids should be restricted to fentanyl. If sedation is required, this should be midazolam or propofol. Supplemental oxygen should always be provided. If conducting general anaesthesia, inhalational agents should be considered for the induction of general anaesthesia and spontaneous ventilation should be used in preference to IPPV.

Due to the increased risk of hypotension, neuraxial and general anaesthesia should not be combined.

Both AAGBI and NICE recommend considering the use of perioperative peripheral regional anaesthesia as a supplement to the anaesthetic and analgesic plan.

Routine monitoring to AAGBI guidelines should be instituted. Arterial line, cardiac output and near patient testing of haemoglobin are also recommended in some patients.

Bone cement implantation syndrome (BCIS) is common after the insertion of cemented prostheses and can be seen in up to 19% of cases with reactions involving severe hypoxia and/or hypotension, or cardiovascular collapse in 2.7% and 0.5% of operations, respectively.

POSTOPERATIVE MANAGEMENT

Postoperative hypoxia is common, and additional nasal oxygen should routinely be administered. Intravenous fluid therapy should be continued until the patient is eating and drinking. Simple analgesia with paracetamol is often sufficient; opioids should be used with caution given the incidence of renal disease and its constipating effects.

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Table 22.2 Advantages and disadvantages of regional vs. general anaesthesia

Advantages of general anaesthesia	Advantages of regional anaesthesia
• Airway control	• Intraoperative and postoperative analgesia
• Variable duration of anaesthesia	• Decreased PONV
• Ability for use of oesophageal Doppler probe	• Decreased incidence of thromboembolism
Disadvantages of general anaesthesia	**Disadvantages of regional anaesthesia**
• Trend for increased blood loss, MI and incidence and postoperative cognitive dysfunction	• Potential for haemodynamic instablility
	• Positioning for insertion

CROSS-REFERENCES

The elderly, Chapter 25
Regional anaesthetic techniques, Chapter 29
Monitoring, Chapter 27
Orthopaedic surgery – general principles, Chapter 22

SHOULDER SURGERY

Shoulder surgery can be performed either by open (e.g. total and hemi-arthroplasty, repairs for instability, subacromial decompression, fracture fixation) or arthroscopic (e.g. subacromial decompression, stabilisation, rotator cuff repair) techniques.

Surgery can be performed under general anaesthesia or a regional technique. If awake shoulder surgery is to be undertaken, patient selection is imperative. Interscalene brachial plexus block provides excellent postoperative analgesia but has a conversion rate to general anaesthesia in up to 8.7%–13%. The skin over the posterior port site is supplied by the supraclavicular nerve (C4 or T2) and may require supplemental local anaesthetic infiltration. Whether open or arthroscopic, the patient is often in the deck chair position although some surgeons prefer the patient lateral.

PROCEDURES

1. Shoulder replacement
   a. Total shoulder replacement (replacement of glenoid and humeral head)
   b. Hemi-arthroplasty (replacement of humeral head, often because of traumatic fracture)
   c. Reverse total shoulder replacement (position of glenoid and humeral head reversed, used for rotator cuff arthropathy)
2. Shoulder arthroscopy

PATIENTS

Vary from fit young patients with sports injuries to frail elderly patients with complex comorbidities.

PREOPERATIVE MANAGEMENT

- Full history and examination.
- ECG in the elderly.
- FBC, U+Es and group and save.
- In the frail and elderly, immobility may mask myocardial ischaemia.
- If deck chair position is to be used, the preoperative blood pressure should be noted and risk factors for cerebral ischeamia evaluated.

POSITIONING

In the deck-chair position, the trunk and head are raised to an angle of 20–30°. Patients are prone to hypotension due to venous pooling. This is magnified if an effective regional anaesthetic technique has been used.

The Bezold–Jarisch reflex has been described, resulting in profound bradycardia, hypotension and even cardiac arrest.

INTRAOPERATIVE MANAGEMENT

- Standard monitoring according to AAGBI guidelines.
- Arterial line if high risk of hypotension and/or cerebral hypoperfusion.
- The use of near-infrared spectroscopy (NIRS) to assess cerebral desaturations has been described.
- General anaesthesia alone or in combination with regional anaesthesia is commonly used. If regional anaesthesia is not used, an IV PCA should be considered in addition to multimodal postoperative analgesia.
- Regional anaesthesia can be provided via single shot or continuous interscalene brachial plexus block. These have been associated with decreased postoperative opioid requirement, faster hospital discharge and less intraoperative blood loss.
- Suprascapular nerve block can reduce pain and opioid consumption but is less effective than interscalene block.

- Intrarticular injection of local anaesthetics is little better than placebo and has been associated with chrondrolysis; it should be avoided.
- Sedation may be needed – boluses of midazolam, or propofol target-controlled infusion are effective. Supplemental oxygen should be administered.

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CROSS-REFERENCES

Day case surgery, Chapter 25
The elderly patient, Chapter 25
Complications of position, Chapter 30
Upper limb blocks, Chapter 29

TRAUMA

Any patient can present with trauma. The procedure may be urgent and therefore the patient may not be starved or optimally prepared for surgery. There may be other injuries, considerable pain or distal ischaemia complicating management.

MAJOR TRAUMA AND TRAUMA TEAMS

It is commonplace to now meet the seriously injured patient in the emergency department as part of the multidisciplinary trauma team. The team leader is generally a senior emergency physician but the team also includes anaesthetists, intensivists, radiographers, nurses, orthopaedic, and general and neurosurgeons.

TRAUMA MANAGEMENT

CARDIOVASCULAR RESUSCITATION

Patients with severe injuries should have reliable large bore IV access. If IO access is required, this should not be sought in limbs with fractures.

CERVICAL COLLARS

Although previously routine, the use of rigid cervical collars has been questioned. They may increase secondary neurological injury, intracranial pressure and certainly worsen intubation conditions.

COAGULATION

Trauma-induced coagulopathy results from tissue injury combined with hypoperfusion. There is a complex interaction between coagulation, cellular and inflammatory dysfunction. Anti-fibrinolytics have been shown to be beneficial when given to bleeding trauma patients, although if given more than 3 hours after injury it can be less effective or even harmful.

DAMAGE CONTROL RESUSCITATION

With its origins in military conflicts, damage control resuscitation describes proactive early treatment to counter the 'lethal triad' of acidosis, hypothermia and coagulopathy. It aims to limit physiological derangement and includes permissive hypotension and early treatment of anticipated coagulopathy with blood products.

INJURY SEVERITY SCORE (ISS)

Individual body regions are assigned an abbreviated injury scale (AIS) score (Table 22.3). The three most severely injured body regions then have their score squared and added together to produce the ISS score.

Table 22.3 Abbreviated injury scale

AIS score	Injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Unsurvivable

SURGICAL PROCEDURES

Manipulation under anaesthesia (MUA) is performed to relocate dislocated joints, correct fracture deformity, improve the mobility of 'fixed' joints or to effect adhesiolysis and improve mobility after arthroplasty.

Internal fixation is used to restore correct anatomy in unstable fractures, promoting bone healing and allowing earlier rehabilitation and return to function. Distal long bone fracture plating is a common procedure, usually performed on a younger, fitter patient group. The frequency of internal fixation for smaller hand and feet bones has increased with improved prosthetic technology. Intramedullary 'nailing' procedures reduce the soft tissue damage and nonunion related to tibial, femoral and humeral fractures, but are associated with an increased incidence of fat embolism syndrome compared to plating procedures.

External fixation involves the use of external rods and frames to maintain bony anatomy and minimise soft tissue injury, either as a definitive procedure or until such time as internal fixation can be attempted.

PREOPERATIVE MANAGEMENT

Emergency patients may not be fasted, and require antacid prophylaxis followed by rapid sequence induction and endotracheal intubation. Occult blood loss from long bone fractures and other injuries can be considerable and not apparent prior to the induction of anaesthesia.

INTRAOPERATIVE MANAGEMENT

- Regional or general anaesthesia may be used. General anaesthesia avoids any contention that post-traumatic neurapraxia may have resulted from regional anaesthesia, rather than the original injury, compartment syndrome or manipulation.
- Fracture reduction requires general or regional anaesthesia, particularly if K-wires are required. Proximal fracture reduction or joint relocation under general anaesthesia may require the administration of a small dose of non-depolarising neuromuscular blocker to facilitate manipulation.
- If a prosthesis is required as part of the surgery, prophylactic antibiotics will be required.

POSTOPERATIVE MANAGEMENT

MUA for fracture/relocation usually relieves pain, with simple analgesics only required postoperatively. Patients undergoing internal and external fixation may require additional analgesia because of the additional tissue damage secondary to the insertion of various pieces of metalwork. Due to prolonged immobility thromboprophylaxis will be required.

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CROSS-REFERENCE

Emergency surgery, Chapter 25

Transplantation

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RICHARD WADSWORTH, GREG COOK,
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ANAESTHESIA AND BRAINSTEM DEATH

GREG COOK

While the concept of anaesthesia for a patient with confirmed brainstem death may seem paradoxical, the role of the anaesthetist is essential in preserving organ function prior to harvesting, thus optimising the probability of subsequent successful organ transplantation.

PATHOPHYSIOLOGY

Brainstem death typically occurs after a devastating cerebral injury which causes a progressive increase in intracranial pressure (ICP) such that the brainstem and cerebellar tonsils herniate through the Foramen Magnum ('coning'), rendering the midbrain ischaemic with irreversible loss of function. The clinical manifestation is complete and irreversible loss of consciousness and ability to breathe. These patients will therefore be mechanically ventilated via an endotracheal tube, ideally with invasive blood pressure and central venous pressure monitoring in ICU.

It is important to understand the adverse physiological changes that affect the major organ systems during and after the process of brainstem herniation that results in brainstem death.

CARDIOVASCULAR

There is an initial adrenergic surge with significant hypertension. The associated Cushing's reflex of marked bradycardia may or may not be seen. In addition to hypertension, raised pulmonary artery pressure and myocardial ischaemia may be seen. There follows a complete loss of spinal cord sympathetic activity as brainstem death is established, usually accompanied by hypotension and end organ hypoperfusion.

RESPIRATORY

Neurogenic pulmonary oedema can accompany the adrenergic surge of brain stem ischaemia. Further increases in extravascular lung water can be seen as part of the inflammatory process associated with brainstem death.

ENDOCRINE

Hypothalamic and pituitary functions are lost resulting in low levels of

- Corticosteroid
- Thyroxine
- Antidiuretic hormone (ADH)

Thermoregulation is impaired.

Glucose homeostasis is impaired and hyperglycaemia appears.

HAEMATOLOGICAL

Disseminated intravascular coagulation is driven by catecholamine effect on platelet function, hypothermia and the release of plasminogen activator and thromboplastin as a consequence of damaged brain tissue.

RENAL

Renal function is largely dependent on haemodynamics. Polyuria is associated with the loss of ADH secretion (neurogenic diabetes insipidus). This can further drive end organ hypoperfusion through reduced preload.

ANAESTHETIC MANAGEMENT

CARDIOVASCULAR

Arterial and central venous access is essential but will usually be in place.

Hypertension can be managed with a variety of agents:

- Labetalol
- Esmolol
- GTN
- SNP

The transition to hypotension can be brisk.
The preferred vasopressor agent is Vasopressin.

- Start with a 0.5 IU/mL infusion; 1 IU/mL can be used.
- Second line vasopressor is norepinephrine – high dose is associated with impaired organ perfusion and decreased organ harvesting.

Intravenous fluid boluses should be goal directed as excess fluids are undesirable.

Avoid synthetic colloids. Use Human Albumin Solution only when necessary.

During organ retrieval, significant fluid shifts and blood loss may be seen and the blood pressure may be very labile.

Maintain haemoglobin above 7 g/dL and guided coagulation by TEG or equivalent. If this is unavailable, aim for INR and APTT ratios <1.6. Maintain platelets >50 and be further guided by platelet function monitoring if this is available.

The retrieving team may request a pulmonary artery catheter as part of their cardiac assessment.

RESPIRATORY

Lung protective ventilatory strategies are essential when lung harvesting is possible. This will minimise any iatrogenic injury which is exacerbated by inflammation.

- Tidal volume 6–8 mL/kg
- Peak pressure <30 cmH$_2$O
- PEEP (escalate with FiO$_2$)
- Normoxia (PaO$_2$ >8 kPa)
- Permissive hypercapnia
- 30° head elevation
- ETT cuff pressure <25 mmHg

Excessive IV fluids are detrimental to lung function and reduce the likelihood of lungs being suitable for transplantation. All patients should receive methylprednisolone 15 mg/kg IV to attenuate the inflammatory process and reduce EVLW soon after brainstem death is confirmed. Bronchoalveolar lavage may be required as part of the assessment or optimisation of lungs considered for transplantation. The retrieving team will perform this.

ENDOCRINE

A variable rate IV infusion of insulin should be used to maintain a blood sugar <10 mmol/L.

Desmopressin (DDAVP) as a 1–2 mcg IV bolus can be used for the polyuria of DI.

Triiodothyronine (4 mcg IV bolus followed by 3 mcg/h infusion) can be given intravenously for

those patients whose cardiac output is suboptimal despite fluid loading and vasopressor use.

RENAL

Match the previous hour's urine output with 5% dextrose or enteral water in cases of DI. Continue maintenance IV fluids with balanced crystalloid solutions. This can continue where enteral feed is in progress. Avoid significant hyper/hyponatraemia. Monitor and correct electrolyte disturbances.

TEMPERATURE

Maintaining normothermia can be difficult.

- Metabolic rate is significantly reduced.
- A combined midline laparotomy and median sternotomy permits significant heat loss.
- Blood and fluid loss may be significant.

Active external warming devices are advised and a fluid warmer is essential.

Continuously monitor core temperature.

CHOICE OF ANAESTHETIC AGENTS

There will be no sedation in use. For harvesting of organs, a muscle relaxant is essential as spinal cord reflexes may be preserved. There is no preferred relaxant, though the histamine release associated with Atracurium may exacerbate hypotension. The use of a nerve stimulator is advised.

Some advocate the use of a low dose inhalational agent under the premise of beneficial ischaemic preconditioning; however, there is no strong evidence base and it is not universally practiced.

Surgical time can be prolonged depending on which organs are deemed suitable for retrieval. Anaesthetic involvement ends with aortic cross-clamping.

HEART TRANSPLANTATION

ANDREW ROSCOE

- Median sternotomy with cardiopulmonary bypass (CPB), using bicaval venous cannulation.

- Orthotopic transplant requires anastomoses of left atrium with native pulmonary veins, right atrium with IVC and SVC, main pulmonary artery and ascending aorta.
- Increasing use of donation after cardiac death (DCD) donor hearts.

PATIENTS

- End-stage heart failure (NYHA 4) with life expectancy less than 1 year.
- Peak VO_2 <14 mL/kg/min (<12 mL/kg/min with β-blocker therapy).
- Majority have ischaemic or dilated cardiomyopathy.
- Typically have biventricular pacemaker/ implantable cardioverter-defibrillator (ICD).
- Decompensated, urgent in-patients usually receiving intravenous inotropes, with intra-aortic balloon pump (IABP) support. Approximately 50% on preoperative mechanical circulatory support (MCS) as 'bridge-to-transplant.'
- May have undergone sternotomy for prior cardiac surgery.
- Absolute contraindications:
  - Untreatable malignancy
  - Active infection (suspended from transplant list until treated)
  - Severe co-morbidity (not amenable to multi-organ transplant procedure)
  - Fixed elevated pulmonary vascular resistance (PVR) >3 Wood units
  - Substance abuse (including tobacco) within previous 6 months
  - Mental illness/inability to comply with postoperative medication
  - Inadequate social support
- Relative contraindications:
  - Age >70 years old
  - BMI >35 kg/m²
  - Poorly controlled diabetes mellitus with HbA_{1C} >7.5%
  - Renal dysfunction with eGFR <30 mL/min/1.73 m²
  - Significant cerebral or peripheral vascular disease

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PREOPERATIVE MANAGEMENT

- Patients often receiving high doses of anti-heart failure medications: ACE inhibitors, ARBs, β-blockers, diuretics and anticoagulants.
- Check electrolytes and INR.
- Calculate transpulmonary gradient (TPG):
  - TPG = mean PA pressure – pulmonary capillary wedge pressure
- TPG >10: may benefit from inhaled nitric oxide (iNO) therapy post-CPB.
- Renal dysfunction common secondary to low cardiac output (CO).
- Preoperative creatinine >220 μmol/L doubles postoperative mortality.
- Preoperative haemofiltration may be used to optimize renal function and fluid balance.
- Patients with intrinsic renal failure may be suitable for heart-kidney transplant.
- Hepatic congestion and dysfunction possible due to right ventricular (RV) failure.

PREMEDICATION

- Oxygen by facemask.
- Avoid sedative medication due to hypoventilation, hypercapnia and pulmonary hypertension, precipitating RV failure.

PERIOPERATIVE MANAGEMENT

- Intravenous fluid warmer. Forced air-warming device.
- Heparin drawn up in advance in case of cardiovascular collapse and emergency institution of CPB.
- Cross-matched blood readily available if resternotomy.
- Pacemaker technician to deactivate ICD: external defibrillator pads essential.

MONITORING

- Wide-bore venous access and invasive arterial monitoring prior to induction.
- 5-lead ECG, SpO_2, $ETCO_2$, nasopharyngeal temperature, urinary catheter.
- CVP and PA catheter sheath: PA catheter may be floated post-CPB.
- Transoesophageal echocardiography (TOE).
- Consider depth of anaesthesia monitor (BIS): high risk of awareness.

PHYSIOLOGICAL GOALS

- Avoid bradycardia: CO becomes rate-dependent in end-stage cardiac failure.
- Avoid increasing SVR: small increases in SVR cause large reductions in stroke volume.
- Avoid excess fluid administration: heart towards end of Starling curve.
- Avoid negative inotropy: high-dose fentanyl cardiovascularly stable.
- Avoid hypoxaemia/hypercapnia: precipitate RV failure due to increased PVR.

INDUCTION

- Preoxygenation.
- Midazolam, etomidate or propofol.
- High-dose fentanyl. Remifentanil has been used but beware bradycardia.
- Pancuronium, rocuronium or vecuronium. Atracurium if significant hepatic or renal dysfunction.
- Prophylactic antibiotics.
- Methylprednisolone.
- Antifibrinolytic (tranexamic acid).
- Vitamin K (patients on warfarin).

MAINTENANCE

- IPPV with O_2/air mixture.
- Inhalational agents have more consistent pharmacokinetics than intravenous drugs in patients with end-stage heart failure.

WEANING FROM CPB

- Target heart rate 90–100 bpm achieved with isoprenaline, dopamine or pacing.
- Epicardial pacing wires routinely inserted: risk of A-V block.
- TOE required to assess ventricular function and filling.

- Risk of RV failure increases as ischaemic time extends beyond 4 hours.
- Avoid excess fluid administration: stiff ventricles easily fluid overloaded.
- LV dysfunction: dopamine, dobutamine or adrenaline.
- RV dysfunction: milrinone, enoximone and/or levosimendan (with vasopressor).
- Low threshold for IABP insertion.
- Consider iNO/nebulised prostacyclin to reduce RV afterload.
- Severe primary graft dysfunction may necessitate MCS.

POSTOPERATIVE MANAGEMENT

- Bleeding is a common complication, especially in patients 'bridged' with MCS.
- Insert nasogastric tube for early administration of enteral immunosuppression.
- Cardiac function/haemodynamics monitored by PA catheter and TOE.
- Majority of cases extubated within 24 hours and managed similar to routine cardiac surgery patients.

OUTCOMES

- Actuarial survival figures are
  - 85% at 1 year
  - 75% at 5 years
  - 55% at 10 years
- Higher early morbidity and mortality in patients 'bridged' with MCS.
- Long-term mortality caused by infection, organ rejection, cardiac allograft vasculopathy (accelerated coronary artery disease), renal failure and malignancy (secondary to immunosuppression).

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LIVER TRANSPLANTATION

ZOKA MILAN

PROCEDURE

- Laparotomy by subcostal (right or bilateral) incision with or without an upper middle incision (Mercedes incision).
- Surgery is divided into phases:
  - Dissection phase – skeletonization of native liver
  - Anhepatic phase – removal of native and implantation of donor liver
  - Reperfusion phase – graft revascularisation with portal or less frequently hepatic artery blood
  - Post-reperfusion phase – haemostasis, completion of hepatic arterial or less commonly portal vein anastomosis and biliary drainage
- Orthotopic liver transplantation (OLT) is the replacement of a whole diseased liver with a donor liver. A successful outcome depends

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on the type and quality of donor liver. Two most common types of donor organs are: DBD (donation after brain death) or DCD (donation after cardiac death). Alternatives include living related or unrelated donor grafts, auxiliary grafts, split or reduced grafts and ABO-incompatible grafts.

- Donor risk index (DRI) is a scoring system used as a valid tool for scoring donor liver quality. Parameters used to calculate DRI are: donor age, cause of death, race, DCD, partial/split liver graft, donor's height, organ location and cold ischaemic time (CIT). CIT extends from the initiation of cold preservation of the recovered organ to restoration of warm circulation after transplantation. For liver transplantation, the CIT should be <8 hours. Warm ischaemic time (WIT) is the interval of time between extubation until the initiation of cold perfusion. WIT should not exceed 30 min for successful liver transplantation.

PATIENTS

Aged from 1 week to 70 years and they fall into two distinct patient groups.

ACUTE LIVER FAILURE (ALF)

- Jaundice and encephalopathy developing in a patient with no history of chronic liver disease.
- Hyperacute liver failure – encephalopathy within 7 days of onset of jaundice.
- Acute liver failure – encephalopathy in 8–28 days from onset of jaundice.
- Subacute liver failure – encephalopathy in 5–12 weeks from onset of jaundice.

Exclusion criteria for LT are: age >70 years (relative), malignancies outside the liver, severe cardiac, lung or multi-organ failure, severe septic shock and recipient's brain death.

CHRONIC LIVER DISEASE (CLD)

A wide variety of congenital and acquired disease in both adults and children may lead to end-stage liver disease. Transplantation is frequently required in order to prolong and/or improve quality of life.

COMMONLY ASSOCIATED PATHOLOGY (MAINLY REVERSIBLE AFTER SUCCESSFUL LT)

- Central nervous system:
  - Encephalopathy (CLD)
  - Cerebral oedema (80% of ALF)
- Respiratory:
  - Restrictive defect due to massive ascites and pleural effusion (CLD)
  - Hepato-pulmonary syndrome (hypoxaemia and intrapulmonary shunting) (CLD)
  - Pulmonary hypertension (1% of CLD) (mean PAP >50 mmHg contraindication to transplantation as postoperative mortality >50%)
  - Adult respiratory distress syndrome, noncardiogenic pulmonary oedema (ALF)
- Cardiovascular:
  - Hyperdynamic circulation (high cardiac output and low systemic vascular resistance)
  - Cirrhotic cardiomyopathy (cardiac dysfunction manifest during stress)
  - Reduced effective circulating volume
- Renal:
  - Pre-renal or renal failure (ALF)
  - Hepato-renal syndrome (CLD)
- Electrolytes/metabolic:
  - Hyponatraemia, hypomagnesaemia, hyperkalaemia (CLD), metabolic acidosis
  - Hypoglycaemia (ALF)
- Haematology:
  - Anaemia, hypofibrinogenaemia, thrombocytopaenia (hypersplenism), platelet dysfunction (CLD)
  - Reduced/defective synthesis of vitamin K dependent clotting factors (ALF, CLD)
  - Hyperfibrinolysis ± low grade DIC (ALF, CLD)

The Model for End-Stage Liver Disease (MELD), Paediatric End-Stage Liver Disease (PELD) and UK

End-Stage Liver Disease (UKELD) scores are numerical scales that are currently used for liver allocation. Scores are based on objective and verifiable medical data and represent patient risk of dying while waiting for a liver transplant.

PREOPERATIVE MANAGEMENT

A multidisciplinary approach is essential to assess risk, aimed at precisely defining the multisystem involvement. Occult cardiovascular disease is a major cause of perioperative death and complications; rigorous cardiac assessment includes echocardiography, cardiopulmonary exercise testing (CPET) or dobutamine stress echo if CPET not possible. Angiography when indicated.

- Detailed patient and relative counseling.
- When an organ becomes available, liaison with donor team is essential.
- Perioperative assessment on day of LT as patients can deteriorate while waiting for the transplant.
- Preoperative optimization on day of LT: correction of anaemia, hypocoagulability, hypofibrinogaenaemia, oxygenation, fluid balance and nutrition optimization (carbohydrate fluids up to 2 hours before surgery).
- Success depends on support services such as blood bank and laboratory services, anaesthetic technical back-up and clinical perfusionists for rapid infusion devices, cell saver and bypass equipment (<5% of LT).

PERIOPERATIVE MANAGEMENT

MONITORING

- ECG, SpO_2, $ETCO_2$
- Invasive arterial pressure, CVP
- Core temperature, urine output, blood loss
- Pulmonary artery catheter, LiDCO, PiCCO or transoesophageal echocardiography (TOE)
- BIS monitoring or cerebral oxygen saturation
- Intracranial pressure

BLOOD SAMPLES

- Arterial gases with electrolytes, glucose and lactate hourly + mixed venous gases
- Full blood count and clotting screen regularly
- Thromboelastography (TEG) or ROTEM, one for each phase of surgery

INTRAVENOUS ACCESS

Wide-bore peripheral venous access, 4–5 lumen internal jugular access, vascular catheter for rapid infusion and postoperative dialysis if required. Large bore bypass lines if veno-venous bypass is used (femoral and jugular or axillary).

ANAESTHETIC TECHNIQUE

- Rapid sequence induction if patient is not fully fasted, or in the presence of massive varices and ascites.
- Induction with narcotic and sleep dose of an intravenous agent.
- Insert a wide-bore nasogastric tube.
- Maintenance with IPPV oxygen/air/ inhalational agent, short-acting narcotic and relaxant infusion.
- TIVA for ALF to decrease ICP.
- PEEP desirable for long surgery, undesirable for increasing intra-hepatic pressure.
- Maintain renal perfusion. Renal function can deteriorate due to liver failure, bleeding, anhepatic phase and acidosis.
- If already on CVVH, continue intraoperatively.
- Maintain cardiac output and oxygen transport, especially in the anhepatic phase. Expect hyperdynamic circulation (high CO, low SVR).
- Inotropes are frequently required.
- Proactive with adequate volume replacement with crystalloids, preferably without lactate. Fluid overload can lead to delayed graft function.
- Intraoperative monitoring of coagulation and appropriate blood product replacement combined with ionized calcium supplementation.

- Prophylactic antibiotic administration, repeat every 4 hours or more frequently when extensive blood loss occurs. Antifungals when blood loss is more than circulating volume. Intraoperative doses of immunosuppression.
- Active warming.

POSTOPERATIVE MANAGEMENT

- Half of patients with CLD are extubated at the end of the procedure.
- Recipient age over 65, BMI >30 kg/m^2, female gender, MELD score >12, redo LT, pre-LT hospital stay longer than 1 day, duration of surgery >6 hours, vasopressors at the end of surgery and intraoperative blood transfusion over 1400 mL are predictors of longer ICU stay and worse outcome.
- In ALF, ICU stay may be prolonged and continuous invasive monitoring is recommended.
- Early liver function is monitored by resolution of metabolic and lactic acidosis and of coagulopathy (falling INR).
- Early complications include bleeding (<5%) and hepatic artery or portal vein thrombosis (<1%), which require early re-exploration.
- Poor initial graft function is treated with all-organ support.
- Primary non-function is rare in UK centres (1–2%), but will entail emergency retransplantation. Initial poor function is relatively common with the increasing use of 'marginal' (DCD, split livers, moderately fatty, older donors, etc.) donors.
- Acute rejection, suggested by changes in AST and INR and confirmed by biopsy if coagulation permits. Treat with additional steroids and/or modification of immunosuppressive regime.
- Cyclosporin or tacrolimus may be introduced early if renal function is satisfactory.

OUTCOME

- Patients with acute liver failure are selected for OLT if their chances of survival with medical treatment are <10%. One-year survival following emergency OLT is currently 60%–80%.

- Patients with chronic liver disease currently have a 1-year survival of 90% and 5-year survival of 76% following OLT.
- Quality of life is improved; the majority of patients are severely incapacitated prior to transplantation.
- The majority of deaths in the early postoperative period are due to sepsis. Later deaths are due to complications of immunosuppression, chronic rejection or recurrence of the primary liver disease.
- Retransplantation will be required in 5%–10% of patients.

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CROSS-REFERENCES

Chronic liver disease, Chapter 4
Previous liver transplant, Chapter 4

LUNG AND HEART–LUNG TRANSPLANTATION

ANDREW ROSCOE

PROCEDURE

- Heart–lung transplant (HLT) is performed through median sternotomy or clam-shell (transverse sterno-bithoracic) incision, with CPB.

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- HLT anastomoses: right atrium (RA) to SVC and IVC, ascending aorta and trachea.
- HLT is becoming a very uncommon procedure (<50 worldwide per year).
- Lung transplant (LT): single (SLT) and bilateral sequential (BSLT).
- SLT: via thoracotomy.
- BSLT: via clam-shell incision or bilateral thoracotomies.
- Anastomoses: main bronchus, pulmonary artery and pulmonary vein cuff to left atrium.
- SLT usually performed off-CPB.
- BSLT may be performed on or off-CPB, or with ECMO support.
- Some evidence that elective use of CPB may be detrimental to outcomes.
- Living donor lobe transplant involves two donors (usually one lower lobe from each) providing organs for BSLT for recipient (usually child or small adult): common in Japan.

DONOR ORGANS

- Significant shortage of donor lungs (less than 20% of donors of other organs have lungs suitable for donation). Approximately 30% on waiting list die before receiving transplant.
- Use of marginal donors, non-heart-beating donors and *ex-vivo* lung perfusion reconditioning designed to increase donor pool.

PATIENT CHARACTERISTICS

- HLT: majority for congenital heart disease.
- LT: end-stage pulmonary disease where transplant provides survival benefit.
- Indications:
  - Emphysema (COPD or alpha-1-antitrypsin deficiency): 38%
  - Idiopathic pulmonary fibrosis (IPF): 24%
  - Cystic fibrosis (CF): 16%
  - Primary pulmonary hypertension (PPH): 3%
  - Sarcoidosis: 3%
  - Retransplantation: 3%
  - Others: 13%

- SLT: 47% for emphysema; 34% for IPF.
- BSLT: 32% for emphysema; 24% for CF; 19% for IPF.
- Emphysema: FEV_1 <20% predicted.
- IPF: diffusing capacity (DL_{CO}) <39% predicted.
- CF: O_2-dependent with hypercapnia and pulmonary hypertension.
- PPH: cardiac index (CI) <2 L/min/m² and RA pressure >15 mmHg.
- Patient may have had previous thoracic surgery, e.g. lung volume reduction surgery.
- Absolute contraindications:
  - Recent malignancy (except locally resectable cutaneous BCC or SCC)
  - Severe comorbidity (not amenable to multi-organ transplant procedure)
  - Evidence of active *Mycobacterium tuberculosis* infection
  - Severe chest wall deformity
  - BMI >35 mg/kg/m²
  - Substance abuse (including tobacco) within previous 6 months
  - Mental illness/inability to comply with postoperative medication
  - Inadequate social support
- Relative contraindications:
  - Age >65 years old
  - BMI >30 mg/kg/m²
  - Hepatitis B/C or HIV infection
  - Colonized with resistant organisms (*Burkholderia cepacia* in CF patients)
  - Severe, symptomatic osteoporosis

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Airway: determine ease of intubation and size of double-lumen tube (DLT) required.
- Respiratory:
  - Chest X-ray/CT scan may provide anatomical information
  - Arterial blood gases (ABGs) on air provide baseline measurement
  - PFTs differentiate between restrictive and obstructive defect
  - Ventilation–perfusion (V/Q) scan displays differential blood flow to each lung

- Cardiac:
  - Right heart catheter measures PA pressures
  - Echocardiogram displays biventricular function
- Renal: patients should have normal kidney function.
- Liver:
  - Mild dysfunction may be present due to congestion from RV failure
  - Hepatic failure (in some CF patients) amenable to lung and liver transplantation
- Consent patients for postoperative insertion of thoracic epidural/paravertebral catheter for analgesia.
- Increased incidence of decompensated patients 'bridged' to transplant with veno-venous ECMO support.

PREMEDICATION

- Oxygen by facemask.
- Bronchodilator therapy.
- Sedative medication is avoided as it can exacerbate hypoxaemia and hypercapnia, precipitating acute pulmonary hypertension and RV failure.

PERIOPERATIVE MANAGEMENT

THEATRE PREPARATION

- Intravenous fluid warmer. Forced air-warming device.
- Left-sided DLT: patients with IPF usually have contracted thoracic cavity and require smaller DLT.
- Fibre-optic bronchoscope (FOB) to confirm position of DLT.
- Heparin drawn up in advance in case of cardiovascular collapse and emergency institution of CPB.

MONITORING

- Wide-bore venous access and invasive arterial monitoring prior to induction.
- ECG, SpO_2, $ETCO_2$, CVP, PA catheter, naso-pharyngeal temperature and urinary catheter.

- Transoesophageal echocardiography (TOE).
- Consider depth of anaesthesia monitor (BIS): high risk of awareness.

PHYSIOLOGICAL GOALS

- Avoid severe hypoxaemia and hypercapnia: tolerated limits are extended based on baseline arterial blood gas measurement.
- Cardiovascular stability.
- Obstructive defects:
  - Low inspiratory pressure, no PEEP, long expiratory phase
  - Risk of dynamic hyperinflation and gas-trapping
- Restrictive defects:
  - High inspiratory pressure, high PEEP, low tidal volume
  - Ventilation strategy similar to ARDS patients
- Infective disease: may require repeated suctioning to remove sputum plugs.
- Pulmonary hypertension:
  - Maintain positive chronotropy/inotropy (adrenaline)
  - Maintain right coronary perfusion pressure (noradrenaline)
  - Avoid hypercapnia/high intrathoracic pressures
  - May require iNO or urgent institution of CPB

INDUCTION

- Preoxygenation.
- Midazolam, etomidate or propofol.
- Fentanyl. Remifentanil has been described.
- Pancuronium, rocuronium or vecuronium. Atracurium if significant hepatic dysfunction.
- Other medications:
  - Prophylactic antibiotics
  - Methylprednisolone
  - Antifibrinolytic (tranexamic acid) if use of CPB
- Single-lumen endotracheal tube for HLT.
- Left-sided DLT for SLT or BSLT.
- Advantage of DLT in BSLT with CPB: allows ventilation and oxygenation of first transplanted lung, whilst second lung being implanted.

MAINTENANCE

- IPPV with O_2/air mixture. Avoid N_2O due to increases in PVR.
- Intravenous drugs (e.g. propofol TIVA) may provide more consistent pharmacokinetics than inhalational agents in patients with profound V/Q mismatch and air leaks.
- Indications for CPB:
  - Severe pulmonary hypertension
  - Unmanageable haemodynamic instability
  - Unable to tolerate one-lung ventilation (OLV)
- If CPB used, heart remains warm and beating throughout.
- Without CPB, the lung with the least perfusion (from V/Q scan) is explanted first, with OLV of the contralateral native lung. After completion of the first side, the transplanted lung undergoes OLV and the second native lung is explanted. Once implanted, both lungs are ventilated with protective lung strategy.
- Advantages of CPB:
  - Avoidance of OLV and hypoxaemia/hypercapnia)
  - Limited duration of haemodynamic instability
- Disadvantages of CPB:
  - Heparinization and increased bleeding
  - Crystalloid fluid loading causing pulmonary oedema
  - Systemic inflammatory response to CPB
  - Reduced allograft function
- Increased use of intraoperative veno-arterial (VA) ECMO support instead of full CPB to reduce degree of heparinization and bleeding complications.

FLUID MANAGEMENT

- Keep crystalloid administration to a minimum: lung allograft prone to low pressure pulmonary oedema due to ischaemia-reperfusion microvascular leak, re-expansion injury and absence of lymphatic drainage.
- Blood and blood products transfused as indicated.

WEANING FROM CPB

- HLT:
  - Similar to heart transplant
  - TOE essential to monitor ventricular function & filling
- BSLT:
  - Protective lung ventilation strategy to avoid barotrauma
  - Use minimal tolerated FiO_2 to reduce free radical injury
  - TOE to assess RV function and pulmonary vein anastomotic sites
- Severe primary graft dysfunction: consider iNO/temporary ECMO support.

POSTOPERATIVE MANAGEMENT

- Change DLT for single-lumen tube: surgeon will pass large bronchoscope to assess bronchial anastomoses.
- Insert nasogastric tube for early administration of enteral immunosuppression.
- Consider epidural catheter once coagulopathy excluded.
- Early extubation is beneficial.
- Maintain restrictive fluid administration regimen.
- HLT: note loss of carinal reflex and lack of cough on suctioning.

OUTCOMES

- HLT:
  - 70% 1-year survival
  - 50% 5-year survival
  - 40% 10-year survival
- LT: BSLT patients have better survival than SLT.
- Actuarial survival figures are
  - 1-year: 80% overall 78% (SLT) 82% (BSLT)
  - 3-year: 65% overall 61% (SLT) 68% (BSLT)
  - 5-year: 54% overall 47% (SLT) 58% (BSLT)
  - 10-year: 31% overall 26% (SLT) 43% (BSLT)
- CF patients have better outcomes – 46% 10-year survival.

- Long-term mortality caused by infection, chronic rejection (bronchiolitis obliterans), renal failure and malignancy (secondary to immunosuppression).

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PANCREAS TRANSPLANTATION

ROSS MACNAB

Pancreas transplantation improves quality of life, stabilises or improves secondary complications of diabetes mellitus and has long-term survival advantage in patients with type 1 diabetes. There are three main categories of pancreas transplantation: simultaneous pancreas and kidney transplant (SPK), pancreas transplant alone (PTA) and pancreas after kidney (PAK). SPK is by far the most common procedure. Other procedures exist, e.g. segmental transplant of the tail from live donors and islet cell transplant.

Surgery takes 5–7 hours via a laparotomy and midline incision and involves placing the graft in the peritoneum. Venous drainage is obtained by anastomosing donor portal vein to recipient iliac vein or vena cava; arterial revascularization is accomplished using the recipient's right common or external iliac artery. Drainage of exocrine secretions is achieved by anastomosing donor duodenum to jejunum (more common) or bladder. In an SPK the pancreas is implanted first. The native pancreas is not removed. The benchwork to prepare the pancreas takes about 2 h. The goal should be to implant the graft as soon as possible keeping the cold ischaemic times to ideally less than 12 h.

The most common indication for islet transplantation is severe hypoglycaemic unawareness. It reliably cures the patients of their hypoglycaemic unawareness; however, most patients continue to require insulin although control of diabetes is much easier. The majority of islet transplant recipients are likely to require more than one graft to complete their treatment. Future research into stem cells and genetic engineering will undoubtedly help improve the success rate of islet transplantation and it will ultimately replace pancreas transplantation as a simpler and safer procedure.

PATIENTS

There is a national protocol for assessment of pancreas transplant patients. All have type 1 insulin-dependent diabetes mellitus. Patients for SPK have diabetes and end-stage chronic renal failure or a predictive date for dialysis within 6 months. For PTA, patients have diabetes with significant diabetic complications. For PAK, they have diabetes, stable function of previous renal allograft and fulfil the criteria for PTA. Patients with poor cardiac reserve and untreatable CAD are excluded. Average waiting time for a pancreas transplant is 2–3 years once listed.

PREOPERATIVE MANAGEMENT

Extensive preoperative assessment and investigations will have been undertaken including

- Echocardiography.
- Assessment of cardiovascular reserve (CPET, 6-minute walk, myocardial perfusion scan or stress echocardiography).

On admission patients require a full history and examination with particular reference to cardiac, renal and diabetic pathology. They require the following up to date investigations on the day of surgery:

- Full blood count
- Urea, electrolytes and calcium
- Coagulation
- ECG
- Four units of blood cross-matched and four bottles of 4.5% albumin available

PREMEDICATION

Cardiac medications should be given (but usually omitting ACE-I and AR2 blockers) and gastric acid prophylaxis considered. Start a standard insulin and glucose sliding scale preoperatively. Anxiolysis can be prescribed if required.

PERIOPERATIVE MANAGEMENT

- Establish IV access, 16 G or above. Do not use any arm with a fistula.
- Thoracic epidural.
- TAP or rectus sheath blocks/catheters if epidural not used.
- Immunosupression and broad spectrum antibiotics.
- Warming equipment.
- Monitoring:
  - Routine monitoring ideally with 5 lead ECG and ST analysis.
  - Arterial line. Inserted awake if significant autonomic dysfunction.
  - Central venous line is essential.
  - Noninvasive cardiac output monitoring.
  - Neuromuscular monitoring.
  - Core temperature.
  - Urinary catheter.
  - Nasogastric/nasojejunal tube.
- Induction:
  - Titrate to patient response. Rapid sequence if warranted.
  - Relaxant: atracurium or cisatracurium is the usual choice.

- Inhalational agent: isoflurane or desflurane as they are metabolised least.
- Opiates: fentanyl or remifentanil.
- Care to obtund the hypertensive response with appropriate agents.

Take baseline bloods for arterial gases, blood sugar and central venous saturation. Near-patient tests of coagulation can be helpful during the operation. Monitor blood gases and near-patient parameters regularly throughout the procedure.

MAINTENANCE

The aim is to keep the patient's fluid optimised, maintain an appropriate perfusion pressure, normothermia and normal biochemical parameters. The goal is to optimise graft perfusion and function. Maintenance with oxygen, air and inhalational agent is usual, but TIVA can be used.

FLUID MANAGEMENT

- Aim to keep well-filled: optimise stroke volume. Parameters to target:
  - HB >10
  - MAP >70 or higher to match patients baseline
  - $ScVO_2$ >70%
  - Lactate <2
- Crystalloid: balanced salt solution should be used to avoid hyperchloraemic acidosis caused by large volumes of 0.9% saline.
- Colloids: 4 bottles of 4.5% albumin. Some prefer to avoid gelofusine due to concerns of graft oedema.
- Blood. Aim for a haemoglobin >10 g/dL.
- 10% mannitol (0.5 g/kg) prior to reperfusion of graft.

REPERFUSION

Aim for a well-filled circulation prior to unclamping of vessels. Stop insulin sliding scale on reperfusion of pancreas. Monitor blood glucose every 15 min for first hour and then every 30 min until stable. Surgeons should be made aware of any significant rise in glucose >10.

POSTOPERATIVELY

- Patients will go to ICU.
- Postop analgesia: epidural or PCA ± LA infusions.
- Aggressive fluid management to aid graft perfusion. Aim to keep well filled.
- Maintenance with balanced salt solutions (e.g. rate = last hour's urine output + 60 mL), with fluid challenges of crystalloid or colloid to maintain CVP or maximise stoke volume. Transfuse when necessary (e.g. if Hb <10 g/dL).

COMPLICATIONS

Complications include early and late rejection, graft thrombosis, pancreatitis, haemorrhage, anastomotic leak, local and systemic infection, systemic inflammatory response syndrome and side effects of immunosuppression.

OUTCOME

The survival rates at 1 year after transplantation are 86% (SPK) and 65% (pancreas only). Overall 1-year survival is 93%–100%.

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CROSS-REFERENCES

Diabetes mellitus, Chapter 6
Pancreatic surgery, Chapter 10
Chronic renal failure, Chapter 5

RENAL TRANSPLANTATION

KAILASH BHATIA

Renal transplantation (RT) improves both length and quality of life and is the most cost-effective treatment available for patients with end-stage renal disease (ESRD). ESRD is defined as chronic kidney disease (CKD) with a glomerular filtration rate (GFR) <15 mL/min/1.73 m^2 or where renal replacement therapy is needed. Diabetes is the most common cause of ESRD in the UK followed by glomerulonephritis, polycystic kidney disease, pyelonephritis, hypertension and auto-immune disorders. The demand for kidney transplant currently far outstrips supply.

Patients being considered for renal transplantation and with no known contraindications are referred to a transplantation program when the estimated glomerular filtration rate (eGFR) is <20–30 mL/min/1.73 m^2. Although no form of renal replacement is indicated at this level of kidney function, early referral allows time for complete evaluation as the majority of patients will have coronary artery disease, diabetes and peripheral vascular disease.

RECIPIENT EVALUATION

A thorough medical, surgical, and psychosocial history and a detailed physical examination are essential. The main contra-indications are

1. Active infection
2. Active malignancy

3. Active substance abuse
4. Reversible renal failure
5. Uncontrolled psychiatric issues
6. Nonadherence to current treatment

Relative contraindications include:

1. Primary oxalosis
2. ANCA (anti-neutrophil cytoplasmic antibody) vasculitis
3. Severe malnutrition

Age is not a contraindication; 18–20% of recipients are ≥65 years of age. Patients with systemic amyloidosis and cardiac involvement might not be suitable as it is associated with high mortality. Individual risk assessment is necessary.

PREOPERATIVE ASSESSMENT AND PREPARATION

- Full blood count, coagulation profile.
- Urea and electrolytes, liver function tests, parathyroid hormone, HbA1c (diabetics), pregnancy test.
- Chest X-ray, ECG, echocardiography.
- HIV, Hepatitis B, C (not a contraindication if well controlled).
- Urine analysis, cultures.
- Screening for measles, mumps, rubella, malaria, CMV, EBV, syphilis, viral infection, sepsis, tuberculosis, infections of unknown aetiology.
- Screening for malignancy.
- Dobutamine stress echocardiography or thallium dipyrimadole stress tests are considered especially in patients with diabetes.
- CPET testing can be undertaken. Patients with anaerobic threshold <11 mL/min/kg combined with inducible ischaemia are at higher risk of postoperative adverse cardiovascular events. If reversible ischaemia is demonstrated, then coronary angioplasty, stenting and revascularization are considered before being placed on the transplant list.

- Cardiovascular diseases, especially IHD and left ventricular dysfunction, are important causes of morbidity and mortality.

THE MATCHING PROCESS

Donor and recipient matching is divided into three areas:

- Blood group and type
- Tissue type
- Final cross-match

Six antigens (major histocompatibility complexes), at three loci (A, B and DR), are considered during tissue matching. In cases of ABO incompatibility, novel immunosuppression techniques such as rituximab, plasma exchange, double filtration plasmapheresis and immune-adsorption techniques can be employed to decrease antibody titres within acceptable limits. A lympho-cytotoxicity cross-match between donor lymphocytes and recipient serum is the final test performed. If the cross-match is positive, then the risk of hyper-acute rejection necessitates consideration of novel immunosuppression techniques or paired donation.

PREOPERATIVE MANAGEMENT

- Full history focussing on cause of renal failure, cardiopulmonary comorbidities especially hypertension, ischaemic heart disease and assessment of functional exercise tolerance.
- Anaemia is common and blood should be cross-matched.

Patients with ESRF will be on dialysis – note the route of dialysis, frequency, amount of fluid removed, vascular access in use. An important decision is whether the patient needs to be dialysed before surgery. Severe acidosis, hyperkalaemia, uraemia-affecting neurology and fluid overload are the most common indications. Dialysis before surgery can lead to intravascular fluid depletion and can exacerbate hypotension from anaesthesia.

Most ESRD patients are taking antihypertensives, antidiabetics, diuretics, statins, erythropoietin and bicarbonate. Beta-blockers, calcium channel blockers,

alpha-blockers, diuretics, aspirin and statins should be continued. Angiotensin converting enzyme inhibitors and angiotensin II receptor antagonists are usually omitted on the day of surgery. Full blood count, coagulation, urea and electrolytes, ECG and echocardiography need to be reviewed before surgery

SURGICAL CONSIDERATIONS

- A curvilinear incision (Gibson's incision) is made from above the symphysis pubis to the anterior superior iliac spine.
- The donor kidney is placed in the iliac fossa, below the native kidney, which is typically left *in situ*.
- The iliac vessels are exposed and end to side anastomoses made to the renal vessels. There is a period of vascular cross-clamping of the iliac artery and vein.
- The ureter of the transplanted kidney is anastomosed to the bladder. Stents are often used to minimize the risk of ureteric obstruction but need to be removed before discharge. A critical point in the operation is when the vascular clamps are removed and the transplanted kidney is reperfused.
- The first warm ischaemia time (WIT) is between clamping the donor vessels and placing the organ in ice in live and heart-beating donors. In DCD donors, this time commences from the onset of asystole to cold perfusion. The second WIT is between removing the organ from ice and completion of anastomosis with the recipient's vessels. Prolonged WIT (>45 min) is associated with worse outcomes and delayed graft function.

The CIT was defined as the period from the start of cold perfusion until the start of the first vascular anastomosis at implantation.

- Cold ischaemia times (CIT) are from the time of cold kidney perfusion to the start of the first vascular anastomosis. The CIT is always lower for live donor transplantation compared to DBD or DCD. CIT of <20 h for DBD and <16 h DCD are associated with better outcomes and lower risk of delayed graft function and primary nonfunction.

ANAESTHETIC TECHNIQUE

General anaesthesia is preferred. Combined spinal-epidural has been described and is acceptable in those with increased risk of respiratory complications. The risks of neuraxial haematoma and infection (epidural abscess) are marginally higher in view of altered coagulation and immune-suppressive strategies.

INDUCTION

An IV agent, opioid and muscle relaxant are most commonly used. Propofol and thiopentone are suitable but the dose should be titrated to minimise the risk of hypotension. Fentanyl and remifentanil are the preferred opioids. The liver metabolizes morphine but its metabolites are excreted in the urine. Cis-atracurium and atracurium are the muscle relaxants of choice.

Suxamethonium can be used if a rapid sequence induction (symptomatic reflux or autonomic dysfunction) is required provided the serum potassium is less than 5.5 mmol/L. Rocuronium 0.9 mg/kg is a suitable alternative but 30% is excreted by the kidneys. Sugammadex can be safely used but the sugammadex–rocuronium complex is eliminated by the kidney. Total intravenous anaesthesia (TIVA) with propofol and remifentanil is satisfactory.

MONITORING

- Routine AAGBI minimal monitoring (take care not to place NIBP cuff or IV lines on the same side as an arteriovenous fistula).
- Neuromuscular blockade.
- Temperature (maintaining normothermia prevents infection, promotes drug clearance and is essential for good graft function).
- Central venous catheter – to monitor central venous pressure (CVP) and allow use of potent vasopressors if necessary. The internal jugular vein is preferred; femoral sites are contraindicated as they potentially interfere with surgery and carry a higher risk of infection.
- Arterial lines are not routine unless there is LV dysfunction or valvular heart disease. Select a

site that will not impede future AVF formation, or undermine existing fistula function.

- Cardiac output monitoring is frequently used to guide fluid therapy although the evidence base is limited. Trans-oesophageal echocardiography can be useful when patients have significant cardiovascular comorbidities.

MAINTENANCE

- Isoflurane, desflurane and sevoflurane are all appropriate choices.
- Analgesia: IV paracetamol, incremental fentanyl. Supplement with regional techniques (TAP block or wound infiltration catheter). Central neuraxial blocks using single shot spinal with a small dose of intra-thecal local anaesthetic and an opioid are increasingly popular as part of enhanced recovery programmes but maintain mean arterial blood pressure >80 mmHg. Epidural catheters are usually avoided for risk of infection and haematoma. Avoid NSAIDS.

FLUID THERAPY

A significant number of recipients are on fluid restriction of 1–1.5 L especially those on dialysis. The use of 0.9% saline is associated with hyperchloraemic acidosis and hyperkalemia so a balanced salt solution (e.g. Hartmann's) is usually used. Hydroxyethyl starch is avoided. Albumin and gelatins are alternative colloids.

Maintain CVP low (5 mmHg) until vessels are clamped followed by high CVP (15 mmHg) when clamps are released to promote graft function and decrease vasopressors and diuretics as required. Cardiac output monitoring can provide useful information on hypovolaemia and response to fluid challenge.

Mannitol up to 0.5 g/kg or furosemide or a combination are frequently used for diuresis during surgery though benefit on graft function has not been shown. There is no evidence to support the use of dopamine. Blood transfusion is avoided unless HB <7 g/dL due to inherent risks including hyperkalemia, allo-sensitisation and transmission of infection.

Intraoperative hypotension and prolonged operating times are the key risk factors for slow recipient graft function. A mean arterial pressure of 80–90 mmHg is recommended especially at the time of graft arterial clamp removal.

Hyperkalemia is treated with sodium bicarbonate, calcium chloride or insulin dextrose solution.

IMMUNOSUPPRESSION

Methylprednisolone is administered at the time of venous anastomosis. Basiliximab 20 mg is given by slow IV injection at induction. It reduces the risk of acute rejection.

In patients with ABO/HLA incompatibility, anti-thymocyte globulin (ATG) and alemtuzumab and rituximab are used. These cause profound lymphocyte depletion and there is a potential for anaphylaxis.

POSTOPERATIVE CARE

Most recipients return to a renal ward unless there are significant cardiovascular comorbidities. HDU care is not normally necessary. Postoperative analgesia is provided with fentayl PCA. Morphine and oxycodone are occasionally used but both are renally eliminated. CVP monitoring guides fluid administration but evidence suggests that suboptimal MAP rather than suboptimal CVP increases the incidence of graft failure.

OUTCOME

Kidney transplantation is a life-changing procedure. Approximately 94% of living donor kidneys and 88% of deceased donor kidneys are functioning at 1 year. Complications include:

- Transplant rejection (hyperacute, acute or chronic)
- Vascular thrombosis and stenosis
- Ureteral obstruction
- Urinary leakage
- Infections and sepsis due to immunosuppression
- Post-transplant lymphoproliferative disorder
- Electrolytes imbalance (particularly calcium and phosphate)

LIVE DONOR NEPHRECTOMY (LDN) FOR RENAL TRANSPLANT

Advantages include:

1. An increase in the donor pool
2. Increased graft survival
3. Totally elective procedure at a time suitable to donor and recipient
4. Avoidance of prolonged dialysis for recipient

Living donor renal transplant is the procedure of choice and improved public awareness leads to an increased number of altruistic donors prepared to undergo this surgery. The welfare of the potential donor should always take precedence over the needs of the potential transplant recipient.

TYPES OF DONORS

Living donors can be:

1. Related: blood relatives of the recipient.
2. Unrelated: with emotional connection (e.g. partners/spouses). Outcomes are equal to genetically related donations.
3. Unrelated without emotional connection:
   a. Altruistic donation: motivated individuals who donate a kidney to a stranger via the national matching and allocation system.
   b. Paired donation: a relative, friend or partner is fit and able to donate an organ but is incompatible with the potential recipient. They are matched with another donor and recipient in a similar situation, so that both patients in need of a transplant receive a compatible organ.
   c. Pooled donation: a form of paired donation whereby the pair are matched with other donors and recipients from a pool of pairs in similar situations, and more than two donors and two recipients are involved in the swap, so that more than two patients in need of a transplant receive a compatible organ.

SELECTION OF DONOR

All living donations for kidney transplantation have to be approved by the Human Tissue Authority (HTA) before donation can take place. The Authority must be satisfied that:

1. No reward has been, or is to be, given.
2. Consent to remove for the purpose of transplantation has been given (or removal for that purpose is otherwise lawful).
3. An Independent Assessor (IA) has conducted separate interviews with the donor (and if different from the donor, the person giving consent) and the recipient (or the person acting on behalf of the recipient) and submitted a report of their assessment to the HTA.

Most of the donors are ASA I or II patients.

Individuals 18 or under are usually not considered as potential living kidney donors and if they are being considered in exceptional circumstances then a parental consent, approval from HTA and from the Court is necessary. Biological age is considered more important than chronological age (donors as old as 70–80 years have been successfully recruited).

Hypertension is not a contraindication as long as kidney function and urine protein are normal. Obesity is a relative contraindication as operating time is longer, blood loss higher, and wound complications and length of stay higher compared to non-obese patients.

Absolute contraindications for donation include diabetes mellitus, malignancy and hypertension with evidence of end-stage organ damage. Albumin creatinine ratio >30 mg/mmol, protein creatinine ratio >50 mg/mmol or 24-hour total protein >300 mg/day usually contraindicates donation.

All living donors must make a voluntary and informed decision and the donor is given the option to withdraw at any time. An IA also assesses the donor to ensure a totally altruistic donation.

ASSESSMENT OF DONOR

Medical assessment of the potential donor is extensive and is performed by a clinician who is not part of the transplant team. A systematic history, battery of blood tests, infection, malignancy screening and a detailed cardiorespiratory assessment are carried out. A psychological assessment is performed to ensure the donor can give fully informed

Table 23.1 Acceptable GFR by donor age prior to donation

Donor age (years)	Acceptable corrected GFR prior to donation (mL/min/1.73 m^2)
Up to 46	80
50	77
60	68
70	59
80	50

consent and is not being coerced. Hypertension will develop in at least 30% of patients following unilateral nephrectomy and donors need to be informed about this. Renal anatomy is assessed in detail using ultrasound and CT/MRI scans and usually the left kidney is chosen because it has a longer renal vein or the kidney with significantly lower function in case of split function is selected for nephrectomy, irrespective of vascular anatomy. It is also essential that the donor in his or her lifetime will not develop clinically significant renal impairment as a result of unilateral nephrectomy. Hence, the donor must have sufficient kidney function prior to donation to have an effective GFR (37.5 mL/min/1.73 m^2) at the age of 80 years, independent of the age at which he or she donated. The acceptable corrected GFR by donor age as per British transplantation Society (BTS) guidelines are highlighted in Table 23.1.

SURGICAL APPROACHES TO LDN

Open LDN using a subcostal incision has been superseded by laparoscopic techniques as they lead to reduced blood loss, decreased tissue trauma, lower analgesic requirements, faster resumption of food intake, shorter hospitalization, quicker return to work and better postoperative cosmetic appearance. The variety of approaches include:

- Laparoscopic transperitoneal LDN
- Laparoscopic hand-assisted transperitoneal LDN
- Robotic assisted laparoscopic transperitoneal LDN
- Laparoscopic retroperitoneal LDN

Surgery for donation and recipient is either carried out synchronously or sequentially to minimize CIT and improve outcome.

ANAESTHETIC CONSIDERATIONS

- Good preoperative hydration is important and some transplant units routinely give IV fluids overnight.
- A good IV access is essential as there is potential for blood loss.
- General anaesthesia with endotracheal intubation is most commonly used.
- Regional techniques such as TAP blocks (laparoscopic) and rectus sheath blocks (hand-assisted laparoscopic) are commonly employed with a GA technique. Epidural or paravertebral blocks are employed in cases of open LDN.
- Arterial line is used in patients with pre-existing hypertension and in cases of left LDN (as potential to damage the spleen) and it allows beat-to-beat blood pressure monitoring, blood gas sampling and cardiac output monitoring.
- Appropriate positioning to prevent brachial plexus damage, pressure sores, airway and venous access compromise.
- Graduated compression stockings and pneumatic compression devices to prevent thromboembolism.
- There is no evidence for the use of prophylactic antibiotics; some centres use a single dose at induction.
- Normoxia, normocarbia, normothermia and normotension along with good perioperative hydration using Hartmann's solution are the gold standard. 10–15 mL/kg/h of fluid is used to maintain a urine output of 1.5–2 mL/kg/h. Oesophageal Doppler may be used to guide fluid therapy.
- Mannitol 0.5 g/kg is given as a free radical oxygen scavenger and diuretic prior to kidney retrieval though the evidence base is limited.
- Dopamine is no longer used.
- Postoperative analgesia is provided via a fentanyl PCA. Early mobilization is recommended along with breathing exercises and incentive spirometry.

POSTOPERATIVE COMPLICATIONS

The mortality from LDN is 1:3000 with pulmonary embolism, hepatitis, myocardial infarction and arrhythmias being the most common. Pneumonia, atelectasis, urinary tract infection and wound infections are the most common complications encountered. Chronic pain incidence is around 5%. Splenic lacerations and splenectomy risk is 1:300–1:500.

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CROSS-REFERENCES

Chronic renal failure, Chapter 5
Nephrectomy, Chapter 13
Fluid and electrolyte balance, Chapter 30

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Paediatrics

BERNADETTE LOMAS

ANAESTHESIA FOR PAEDIATRIC SURGERY: GENERAL PRINCIPLES

Children should receive specialised care coordinated around their individual age-appropriate needs and that of their families. This includes staff that are trained and experienced in providing perioperative care for children.

PREOPERATIVE PREPARATION

BEFORE ADMISSION

Both child and parents should be given a clear explanation of the proposed surgery and admission procedures at the initial outpatient clinic visit. They should be supplied with suitable written information regarding surgery, anaesthesia and instructions on preoperative fasting requirements. Children have the same fasting requirements as adults (6 hours for food or opaque liquids, 2 hours for clear liquids). A baby is considered starved four hours after a breast milk feed.

Preadmission care may include attendance at a clinician or nurse-led preoperative assessment clinic. Preadmission services, which include age-appropriate psychological preparation using books, pictures, videos, play therapy and/or a tour of the hospital, may be useful in reducing children's anxiety.

Full birth history including the duration of pregnancy, delivery or perinatal problems, presence of any congenital and/or acquired diseases should be taken.

Details of previous anaesthetics and any family history of anaesthetic problems should be detailed.

Perform blood tests and other investigations when indicated. Sickle-cell screening should be performed in susceptible ethnic groups.

A physical examination and airway assessment including noting the presence of any loose or vulnerable teeth is necessary.

Allergies and vaccinations. The routine UK vaccination schedule should be followed for all children. For children undergoing major surgery, risk assessment should incorporate the risks of side effects from vaccinations occurring if there is expected to be a prolonged postoperative recovery period.

Surgery following immunization with inactivated vaccines should be delayed for 48 hours to avoid post-vaccination symptoms causing diagnostic confusion. Surgery following immunization with live attenuated vaccines does not need to be delayed, provided the child is otherwise well. There is no

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contraindication to vaccination immediately after surgery, once the child is well and recovered from the procedure.

Telephone contact on the day before surgery provides an opportunity to confirm attendance, re-enforce preoperative instructions and detect reasons for late cancellation, such as family problems or an acute infection.

ON ADMISSION

The child should be admitted to a dedicated children's day-case unit or ward staffed by medical and nursing staff trained in dealing with children and their families. The facility should be suitably decorated and toys, books, videos and a play therapist should be available. All children should be weighed, BMI calculated and have their pulse rate, blood pressure and temperature recorded.

The anaesthetist should see the child and the parents before surgery to confirm the adequacy of (or perform) the preoperative assessment, confirm compliance with fasting guidelines, discuss anaesthetic techniques and postoperative pain management and obtain verbal consent for invasive procedures such as suppositories and nerve blocks. A major aim of the discussion should be to establish rapport with the child and parents, allay anxiety and provide reassurance to the family.

- All communication should be comprehensible to the child and the parents and all questions should be answered truthfully, but tactfully.
- The possible modes of induction (IV/inhalation) should be discussed and the wishes of the child and parents complied with where possible.
- If IV induction is planned, a topical local analgesic preparation should be applied over possible venepuncture sites.
- Selected children may benefit from oral sedative premedication (e.g. oral midazolam, 0.5 mg kg^{-1}, maximum 20 mg, 30–45 min preoperatively).
- The child should be allowed to wear suitable clothing of their own to theatre.
- A parent should be invited to accompany the child at induction of anaesthesia and their role in the anaesthetic room discussed at the preoperative visit.

Children with a history of upper respiratory tract infection within 4 weeks of operation are at increased risk of respiratory complications during or after anaesthesia. Ideally, elective surgery should be postponed for 4–6 weeks, but this is not always practical as symptoms tend to recur. If a decision is made to proceed, the increased risk of airway reactivity and complications must be discussed with the child's parents. It may be prudent to intubate and ventilate the patient during anaesthesia to minimize the risk of coughing or laryngospasm. Careful monitoring and supplemental oxygen will be required during recovery. When signs of lower respiratory tract infection are present, elective surgery must be postponed for 4–6 weeks to allow hyperactive airways to return to normal.

MANAGEMENT OF ANAESTHESIA

INDUCTION

Inhalation induction is often more convenient for infants and toddlers who have poor venous access and difficulty cooperating with an intravenous induction. Inhalation induction is also rapid in these very young patients owing to their relatively large minute volume ventilation in relation to the functional residual capacity and a relatively high cardiac output. Sevoflurane is useful due to its lack of pungency, rapid uptake and elimination and reduced incidence of cardiovascular effects. For older children with visible veins, intravenous induction of anaesthesia with thiopental or propofol is quicker and creates less operating room pollution.

AIRWAY MANAGEMENT

A laryngeal mask (LM) is the most common method of managing the airway in children over 1 year of age undergoing relatively short procedures with spontaneous ventilation. It may also be used for short procedures in some infants aged 6–12 months depending on the experience and preference of the anaesthetist. The appropriate sizes of LM for paediatric patients together with their maximum inflation volumes are given in Table 24.1. Over-inflation of the cuff must be avoided as it may cause trauma to the pharynx and

Table 24.1 Laryngeal mask sizes for paediatric patients

Size	Weight (kg)	Maximum inflation Volume (mL)
1.5	5–10	7
2	10–20	10
2.5	20–30	15
3.0	30–50	20
4.0	> 50	30

larynx or herniation of the cuff. The LM is usually inserted during moderately deep anaesthesia without the aid of a muscle relaxant. It can be left in situ at the end of the case and removed by the recovery nurse when the child is fully awake.

Despite the popularity of the LM, tracheal intubation remains the 'gold standard' for paediatric airway management, especially when controlled ventilation is required.

In contrast to adults, infants and children are intubated with the head in a neutral position as raising the head on a pillow does not improve the view of the larynx. The most effective manoeuvre to optimize laryngosopy is the application of external pressure at the level of the cricoid cartilage to push the larynx into view.

In infants a flat-blade laryngoscope such as the infant Magill which passes posterior to the epiglottis may be more suitable than a curved one, since it flattens out the U-shaped curvature of the epiglottis and can be used to lift it forwards to expose the larynx. In children over 1 year of age, laryngoscopy can usually be accomplished using a medium-sized Macintosh blade with the tip placed in the vallecula.

Traditionally cuffed endotracheal tubes (ETT) have not been used in prepubertal children. The correct sized ETT is one that passes easily through the cricoid ring giving a minimal leak with lung inflation pressures up to 20 cm H_2O. Complete absence of a leak in an uncuffed ETT implies close contact between the tube and larynx. This should be avoided due to the risk of pressure necrosis. There is now increasing use of microcuffed ETT in children who have not reached puberty. These are high volume, low pressure cuffed ETTs which minimise the pressure effect on the tracheal mucosa. Even after calculation of the appropriate ETT size, there is still variation in size

between individuals which can necessitate changing the ETT. The main advantage of cuffed ETT is to minimize instrumentation of the larynx with reintubation episodes required to resize the ETT. Whenever a cuffed ETT is used, cuff pressures must be checked frequently.

The following formulas for estimating ETT size may be used as a guide in children 2 years of age and over:

Uncuffed Tube Size (Internal Diameter in mm)

$$= \frac{Age\ (Years)}{4} + 4$$

Cuffed Tube Size (Internal Diameter in mm)

$$= \frac{Age\ (Years)}{4} + 3$$

ETT sizes in infants and children less than 2 years of age have to be memorized. A normal neonate weighing 3 kg usually requires a 3 mm ETT; premature and low-weight babies may require a 2.5 mm ETT. Other sizes can be interpolated.

Some anaesthetists cut ETTs to a length that allows the tip of the tube to be placed in the midtrachea, while 2–3 cm protrudes from the mouth for fixation. The following formula may be used to estimate orotracheal ETT length in children over 2 years of age:

$$Orotracheal\ Tube\ Length\ (cm) = \frac{Age\ (Years)}{2} + 12$$

Orotracheal tube lengths for patients less than 2 years of age have to be memorized. The length for neonates is 10 cm, and for a 1-year-old is 12 cm; other tube lengths can be interpolated. The position of the tracheal tube should always be checked by auscultation of the lung fields.

MAINTENANCE OF ANAESTHESIA

In general, infants are poor candidates for anaesthesia with spontaneous ventilation because of poor pulmonary mechanics. In most of these patients, the

combination of tracheal intubation and balanced anaesthesia with full doses of muscle relaxants, controlled ventilation, minimum concentrations of inhalational anaesthetics and reduced doses of opioids will be required. This regimen provides ideal surgical conditions with minimal cardiovascular depression and rapid return of laryngeal reflexes at the conclusion of anaesthesia.

Children over 1 year of age undergoing long or complex surgery will also benefit from balanced anaesthesia. However, for many children undergoing operations lasting less than 30–40 min, simple inhalation anaesthesia with 66% nitrous oxide in oxygen and sevoflurane (2%–3%) may be adequate. This may be combined with an opioid analgesic, local infiltration or a regional block to provide analgesia in the postoperative period.

Nerve blocks in children are almost always performed once the child is anaesthetised. Consent for the block must be established preoperatively in discussion with the parents, and there should be meticulous attention to correct site block. All nerve blocks performed in adults are suitable for children, with the exception of the vertical infraclavicular block approach. Ultrasound has been shown to increase the success of nerve block siting.

ANAESTHETIC BREATHING SYSTEMS

Anaesthetic breathing systems may be classified into those that do not contain a chemical means of absorbing carbon dioxide and those that are equipped with such units. In the past, concerns about resistance to breathing and apparatus dead space with the use of absorber systems led paediatric anaesthetists to use mainly nonabsorber breathing systems. However, concerns for economy and environmental pollution have greatly increased the use of circle absorber systems in paediatric anaesthesia.

The Jackson Rees T-piece is a popular nonabsorber breathing system for paediatric anaesthesia due to its compact size, low resistance to breathing and low apparatus dead space. The low compression volume of the T-piece gives a good 'feel' for the lung compliance in infants and young children and facilitates hand ventilation even in the face of a decrease in lung compliance or partial respiratory obstruction. Notable disadvantages of the system include its high fresh gas requirements (3–8 L min^{-1}) and the inability to scavenge waste gases. However, the practical advantages of the T-piece can outweigh its disadvantages when the system is used for induction of anaesthesia and anaesthesia of short duration.

The main advantages of circle absorber systems are economy in the use of anaesthetic agents and gases, conservation of heat and moisture in the respiratory tract and reduced operating room pollution. These advantages are most evident when the circle system is used for maintenance of anaesthesia of intermediate-long duration. The main disadvantage of circle systems in paediatric anaesthesia is their high compression volume which gives a poor 'feel' for the lung compliance in infants and young children and may make it difficult to hand-ventilate these patients in the event of an unexpected decrease in lung compliance. Accordingly, a system with a low compression volume, such as the T-piece, should always be readily available when using a circle system in children.

MONITORING

Routine monitoring should include:

- ECG
- Noninvasive blood pressure
- Pulse oximetry
- Respiratory gases

A range of paediatric cuffs must be available for measurement of blood pressure; the correct sized cuff is one that covers two-thirds of the upper arm. Core temperature measurement is mandatory in infants and young children who are at especially high risk of developing hypothermia. Heating devices such as electric under-blankets and warm air blowers should be available to counter heat loss in these patients.

INTRAVENOUS THERAPY

Intravenous fluids are given during surgery to correct the preoperative fasting deficit, satisfy maintenance requirements and replace intraoperative losses (e.g. third space losses and blood). In most children

over 1 month of age, all these requirements should be managed initially by giving an isotonic fluid (e.g. normal saline or Hartmann's solution). Neonates, and some other high-risk patients, should receive a glucose-containing maintenance fluid and/or have their blood glucose monitored during surgery and postoperatively.

The fasting deficit can usually be treated by giving a bolus of 10 mL kg^{-1} of Hartmann's solution after induction. Maintenance fluid requirements can be calculated from the patient's weight using Holliday and Segar's work (Table 24.2). Third space loss may be as little as 1–2 mL kg^{-1} for neurosurgery and 6–10 mL kg^{-1} h^{-1} in major laparotomy. Clinical signs such as heart rate, blood pressure and capillary refill time can be used to guide replacement, but when large fluid shifts are anticipated CVP measurement is essential.

Blood loss should be estimated and replaced initially with isotonic fluid or colloid. Blood transfusion is indicated if the Hb decreases to 7.0 gm dL^{-1}, which corresponds to a haematocrit of 25%.

POSTOPERATIVE MANAGEMENT

At the conclusion of anaesthesia the child should be turned into the lateral position and transported, breathing oxygen, with end-tidal CO_2 monitoring, to a fully equipped recovery room. Details of the operative procedure and any special instructions should be given to the recovery nurse assuming care of the child. Recovery-room protocol should include airway maintenance, provision of oxygen therapy, end-tidal CO_2 monitoring, monitoring of oxygen saturation, pulse, respiration and blood pressure, and the completion of a postanaesthetic recovery chart. Once the child is awake, a parent should be called to the recovery room. The anaesthetist should check that the patient is pain-free

and that postoperative fluids, analgesics and antiemetics have been ordered before the child is returned to the surgical ward. Codeine is not used in children younger than 12 years, and never in children for tonsillectomy who have OSA. Some centres do not use codeine for children of any age.

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CROSS-REFERENCE

CIRCUMCISION

PROCEDURE

Circumcision is excision of the foreskin from around the penis. It is usually performed on an elective day-case basis.

Table 24.2 Holliday-Segar method for calculating maintenance fluid requirements in children

First 10 kg	100 mL/kg/day	4 mL/kg/h
Second 10 kg	50mL/kg/day	2mL/kg/h
Every 1 kg thereafter	20mL/kg/day	1mL/kg/h

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PATIENT CHARACTERISTICS

Approximately 12,200 boys each year in England are circumcised in the hospital setting (3.8% of boys <15 years old). Pathological phimosis is the only absolute indication for circumcision. This affects 0.6% of boys, with a peak incidence at 11 years of age, and is rarely encountered before the age of 5. Most circumcisions are scheduled for recurrent balanitis. In addition, many procedures are performed by nonmedical practitioners for religious and cultural reasons. These procedures are usually performed in the neonatal period.

PREOPERATIVE ASSESSMENT

- Exclude active respiratory tract infection with productive cough or pyrexia.
- Exclude those with a recent exposure to childhood infections.
- Ensure no history of bleeding diathesis or anaesthetic problems.

PREOPERATIVE INVESTIGATIONS

- Not necessary unless clinically indicated.

PREOPERATIVE PREPARATION

- Confirmation of fasting status.
- Obtain informed consent and explain to parents if a local anaesthetic procedure and/or rectal analgesics are to be used during surgery.
- A parent should be invited to accompany the child at induction of anaesthesia.

PREMEDICATION

- Topical local anaesthetic preparation over possible venepuncture sites.
- Oral paracetamol 20 mg kg^{-1} and/or ibuprofen 5 mg kg^{-1}, 30–45 min before operation to enhance intraoperative and postoperative analgesia.
- If anxiolytic required, oral midazolam (0.5 mg kg^{-1}) may be given 30–45 min before surgery.

PERIOPERATIVE MANAGEMENT

- Routine noninvasive monitoring.

GENERAL ANAESTHESIA

- Intravenous or inhalation induction.
- Spontaneous ventilation via laryngeal mask for children over 1 year of age.
- IPPV via tracheal tube for children under 1 year of age.
- IV paracetamol 15 mg kg^{-1} if not given as premedication.

LOCAL ANAESTHESIA

- The local anaesthetic block should be performed after induction to provide intra- and postoperative analgesia.
- Penile nerve block using plain 0.25% levobupivacaine (1–3 mL) for babies less than 1 year of age and plain 0.5% levobupivacaine (3–5 mL) for those over 1 year of age (Box 24.1).
- Alternatively, a single shot caudal extradural block, using 0.25% levobupivacaine (0.5 mL kg^{-1}) is suitable (Box 24.2).
- Clonidine (1 μg kg^{-1}) or preservative free ketamine (0.5 mg kg^{-1}) additives may be used to prolong the caudal block.

BOX 24.1: Penile nerve block

- The nerves lie deep and superficial to Buck's fascia and may be separated by a midline septum.
- Supine position.
- Aseptic technique.
- A 21-SWG regional block needle is used.
- Palpate the lower border of the symphysis pubis with the index finger and retract the penis.
- Insert the needle between finger and arch of pubis until there is a slight 'give' or bone is struck; if bone is struck, 'walk' the needle inferiorly until it is free.
- Local anaesthetic solution should be injected into either side of the midline by directing the needle from a single puncture.
- Aspirate before injecting deep and superficial to Buck's fascia.
- Analgesia should last about 4–6 h.

- The sacral hiatus is located by placing the child on his left side (for a right-handed operator) with legs flexed at the hips. The posterior superior iliac spines are located with the thumb and middle finger and an equilateral triangle formed with the index finger will reliably locate the sacral hiatus at the lower end of the vertebral column.
- Strict aseptic technique.
- A 22-SWG cannula or a regional block needle is used.
- The cannula or needle is advanced through the sacrococcygeal membrane at the apex of the hiatus until a 'give' is felt. The cannula or needle should only be advanced a few millimetres to avoid dural puncture.
- Check for blood/CSF free flowing, and on aspiration.
- Local anaesthetic should inject easily, with no swelling around the sacrum.
- Analgesia should last about 4–6 h and up to 12 h with caudal additives.

POSTOPERATIVE MANAGEMENT

- Good postoperative analgesia is essential for smooth recovery; be prepared to supplement with oral or intravenous opioids.
- Provide an information leaflet, contact details and oral analgesia on discharge.

COMPLICATIONS

- Failure to micturate (more likely with inadequate analgesia; penile block has a higher failure rate than caudal).
- Unsteady when walking (occasionally with caudal epidural, but should not delay discharge in prewalking children).
- Nausea and vomiting.
- Bleeding.

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CROSS-REFERENCES

Infants and children, Chapter 25
Local anaesthetic toxicity, Chapter 30

CONGENITAL DIAPHRAGMATIC HERNIA

The incidence of congenital disphragmatic hernia is 1 in 2–4000 live births. It is probably a primary defect of lung growth – the affected lung is intrinsically abnormal. Right-sided hernia (10%) is associated with higher mortality. Aetiology is unclear; 15% of those affected have chromosomal abnormalities. Approximately 40% have associated major congenital abnormalities.

Congenital disphragmatic hernia presents with respiratory failure at birth or now more often at antenatal ultrasonography – around 60% are detected antenatally at an average gestational age of 24 weeks. Mode of birth should be decided solely by obstetric considerations and delivery should be as close to

term as possible. Patients have bilateral pulmonary hypoplasia (normal side affected by mediastinal shift during growth) and tend to revert to fetal circulation with severe right–left shunting. The degree of pulmonary hypoplasia and pulmonary hypertension largely determines outcome. Long-term follow-up is essential.

Death occurs due to:

- Inadequate gas exchange surface.
- Fixed high pulmonary vascular resistance (decreased vascular cross-sectional area, normal cardiac output).
- Reversible pulmonary hypertension (abnormal muscularity of vessels).
- Pneumothorax.
- Additional anomalies (5%) and complications of intensive therapy.

RESUSCITATION

Mask inflation distends herniated viscera, worsening mediastinal shift and risking pneumothorax, and barotrauma further damages the hypoplastic lung. Use immediate tracheal intubation with muscle relaxants to facilitate IPPV. Nasal intubation aids secure fixation and ventilator compliance. Pass a nasogastric tube to deflate the gut and keep on free drainage.

PREOPERATIVE PREPARATION

Surgery often worsens lung mechanics and is not an emergency. Time (days) should be taken to stabilize and improve gas exchange by meticulous medical management aiming to avoid trigger factors for pulmonary vasoconstriction (hypoxia, hypercarbia, acidosis) and allow the normal physiological fall in pulmonary vascular resistance to occur. Precise indicators of optimal time for surgery have not been established.

MONITORING

Peripheral arterial cannulation allows arterial pressure and blood gas monitoring with minimal disturbance. Published predictive indices require

post-ductal oxygen values. Pulse oximeters placed pre- and post-ductally may demonstrate the variability of shunting.

VENTILATION

- Risk of pneumothorax from high inflation pressures and asynchrony.
- Muscle relaxants (e.g. atracurium or cisatracurium) by infusion give optimal control and decrease oxygen consumption. Continue until weaning after surgery.
- 'Gentle ventilation strategies' (Peak airway pressure <25 mmHg) reduce barotrauma.
- No firm evidence for benefit from individual treatment modalities (e.g. surfactant, HFOV, iNO, ECMO).
- Protocolized care may improve institutional survival.

ACID–BASE STATUS

Metabolic acidosis should be corrected with buffers. Moderate alkalosis by systemic alkalinization (to pH 7.5–7.6) or hyperventilation (to $PaCO_2$ 30–35 mmHg) may enhance pulmonary circulation.

PULMONARY CIRCULATION

Echocardiogram is essential to estimate the severity of the PHT. Refractory hypoxaemia may respond to pulmonary vasodilators. Nitric oxide (iNO) 10–20 ppm has superseded other agents.

FLUID BALANCE

Preoperative restriction (6 mL kg^{-1} per 24 h) avoids fluid retention. Initial maintenance fluid should contain 5%–10% glucose (e.g. 0.18% NaCl with 10% glucose) switching to parenteral nutrition by 48 h. Circulating volume should be maintained with plasma or blood (maintain Hb above 14 g dL^{-1}).

SEDATION

Lability in response to handling (unusual) may be helped by a narcotic (e.g. morphine) infusion.

SURGERY

The timing of surgery per se does not affect survival, so it should be undertaken after optimisation of clinical parameters. Usually undertaken via an upper abdominal incision, which permits correction of gut malrotation. In about 5% of cases, closure can only be achieved with a prosthetic patch or latissimus dorsi muscle flap. Such patients are often slower to wean. Thorascopic surgery is possible in older children and adults.

ANAESTHESIA

- The exact pattern of preoperative ventilation should be continued.
- Volatile agents should be avoided – cardiovascular depression.
- Fentanyl in large doses (up to 25 μg kg^{-1}) obtunds response to surgery (even with 100% oxygen) and has negligible cardiovascular effect. Alternative is remifentanil infusion.
- Monitoring must include ECG, direct or indirect arterial pressure, pulse oximetry, capnography and body temperature.
- A glucose-containing maintenance fluid should be used in place of parenteral nutrition intraoperatively and for 24 h postoperatively. Blood glucose should be monitored. Intraoperative losses can be replaced with an isotonic fluid (e.g. Hartmann's solution or 0.9% NaCl) or 4% human albumin. Red cell transfusion is rarely required.

POSTOPERATIVE CARE

General intensive care with attention to fluid and nutritional requirements (enteral or parenteral) must continue. Weaning from IPPV in severe cases may take months. After tracheal extubation, a period on CPAP is usual. The most severely affected survivors have little respiratory reserve, require long-term oxygen therapy and may die in infancy from respiratory infection.

OUTCOME

Current management has evolved without controlled trials. Reported mortality in large series ranges from 29% to 55% (but note case selection bias). Nutritional problems remain a source of morbidity for survivors. Common long-term problems are chronic lung disease, gastroesophageal reflux, poor weight gain and developmental delays including hearing problems. Around half of those repaired with a patch will require surgery for herniation repair within 3 years.

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CROSS-REFERENCE

Infants and children, Chapter 25

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS

Repair of congenital hypertrophic pyloric stenosis (Ramstedt's procedure) is performed:

- Via either a transverse incision in the right upper quadrant or a hemicircumferential supraumbilical incision (both in a skin crease).

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Some centres perform the operation laparoscopically.

- By a longitudinal serosal and muscular incision down to the mucosa of the pylorus.
- Patency is often checked by passage of air through the pylorus injected via a nasogastric tube at operation.

PATIENT CHARACTERISTICS

- Typically first-born male children (more males affected than females), higher incidence in those with affected parents.
- Age 3–8 weeks.
- Incidence 1 in 300 live births, but considerable regional variation.

Symptoms and signs are shown in Box 24.3.

Pyloric stenosis is one of the most common gastrointestinal abnormalities presenting in the first 6 months of life. Associated abnormalities are found in 6%–20% and are predominantly gastrointestinal, congenital cardiac abnormalities and minor renal abnormalities.

BOX 24.3: Clinical features

- Bile-free vomiting after every feed, becoming projectile.
- Hungry.
- Dehydrated: may vary from mild to severe hypovolaemia.
- Visible peristalsis in left upper quadrant from left to right.
- Palpable tumour.

PREOPERATIVE ASSESSMENT, INVESTIGATIONS AND RESUSCITATION

The operation is never an emergency and full resuscitation is imperative prior to anaesthesia. This may take several days. Preoperative management involves the cessation of oral feeding, passage of a nasogastric tube and correction of the fluid and electrolyte status. Diagnosis is usually made clinically and confirmed with ultrasound scan.

There are two main preoperative problems:

- Dehydration: assessment of volume status (note that 1 L of water is approximately equal to 1 kg weight) (Table 24.3).
- Biochemical defect: hypochloraemic metabolic alkalosis; hypokalaemia.

Loss of hydrochloric acid in the vomitus results in metabolic alkalosis. Correction of this defect depends on retention of hydrogen ion by the kidney. As hydrogen and potassium ions are exchanged for sodium in the distal renal tubule, retention of hydrogen ions results in increased potassium excretion and hypokalaemia. If dehydration becomes severe, increased reabsorption of sodium ions increases excretion of both hydrogen and potassium, exacerbating hypokalaemic alkalosis.

Treatment requires replacement of the calculated fluid deficit with 0.9% saline with potassium (40 mmol L^{-1}).

In severe dehydration up to half of the calculated fluid deficit may be required in the first hour. Once the circulation is restored and urine output of at least 1 mL kg^{-1} h^{-1} is established, the remaining deficit can be replaced over 24–48 h. Maintenance fluid requirements should also be given as 0.45% saline in 5% dextrose.

Table 24.3 Assessment of hydration in the infant

	Mild	Moderate	Severe
Loss of body weight (%)	5	10	15
Clinical signs	Dry skin and mucous membranes	Mottled cold periphery; loss of skin turgor; sunken fontanelle; oliguria; low blood pressure	Shocked; moribund
Replacement (mL kg^{-1})	50	100	150

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The serum electrolytes and acid–base status must be normal before surgery takes place.

PREMEDICATION

Not usually required.

THEATRE PREPARATION

Increase ambient temperature in theatre to 24–26°C. Warming blanket, warm gamgee padding, fluid warmer and warm-air blower should be available.

PERIOPERATIVE MANAGEMENT

Monitoring:
- ECG
- Noninvasive blood pressure
- SpO_2
- $ETCO_2$
- Core temperature
- Peripheral nerve stimulator
- Fluid balance and blood loss

ANAESTHETIC TECHNIQUE

General anaesthesia should be induced in the operating theatre with all efforts made to maintain the infant's body temperature. All precautions protecting against a full stomach should be employed. The nasogastric tube should be aspirated prior to induction, manoeuvring the infant into the left and right lateral, and head-down positions, allowing maximum aspiration of stomach contents. Two techniques for induction of anaesthesia are commonly used:

- A rapid sequence induction (RSI) with preoxygenation and gentle cricoid pressure. In addition to preoxygenation, gentle ventilation is often necessary to prevent hypoxaemia during RSI in infants due to their relatively high oxygen consumption and small functional residual capacity (FRC).
- An inhalational induction with oxygen and sevoflurane followed by tracheal intubation.

Both techniques of induction should be followed by a nondepolarising relaxant and controlled ventilation.

Following the relatively short procedure and reversal of any residual neuromuscular blockade, the infant should be extubated awake and vigorous in the left lateral position.

POSTOPERATIVE MANAGEMENT

Postoperative pain relief can be provided by wound infiltration with local anaesthetics (e.g. 0.25% chirocaine 1 mL kg^{-1}) and intravenous paracetamol 10 mg kg^{-1} 8 h^{-1}. Only rarely are more potent analgesics required. Opioids increase the risk of postoperative apnoea.

There should not be any need for critical care admission postoperatively if the infant is otherwise well. The infant can be fed soon after surgery; 70% of infants tolerate this. Graduated feeding regimens are of no proven benefit.

COMPLICATIONS

Morbidity is low (<1%) and predominantly relates to surgical complications – wound dehiscence, duodenal perforation and inadequate pyloromyotomy.

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CROSS-REFERENCE

Infants and children, Chapter 25

TRACHEO-OESOPHAGEAL FISTULA AND OESOPHAGEAL ATRESIA

Tracheo-oesophageal fistula (TOF) and oesophageal atresia (OA) repair is performed as follows:

- Left lateral position (right side up).
- Axillary skin crease or axillary longitudinal incision.
- Extrapleural approach, if possible.
- Requires compression collapse of right lung.
- In the presence of OA, closure of TOF and either primary oesophageal anastomosis or gastrostomy and possibly oesophagostomy is required.

PATIENT CHARACTERISTICS

- Generally less than 1 week old
- Incidence of 1 in 3500 live births
- 30% of babies are premature

There are several different combinations of fistula and atresia. The three most common are oesophageal atresia and lower pouch fistula (80%), oesophageal atresia with no fistula (10%), and tracheo-oesophageal fistula with no oesophageal atresia (2%) (Figure 24.1). Approximately 50% of newborns with TOF/OA have associated abnormalities.

COMMON ASSOCIATIONS WITH TOF

- 20%–25% have associated major cardiac anomalies
- Polyhydramnios in the mother is common
- VACTER (L) anomalies
- Increasing antenatal diagnosis

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

Increasing antenatal diagnosis; many still be diagnosed postnatally. TOF should be suspected in newborns with difficulty clearing saliva, repeated choking episodes or transient cyanosis shortly after

| 80% | 10% | 2% |
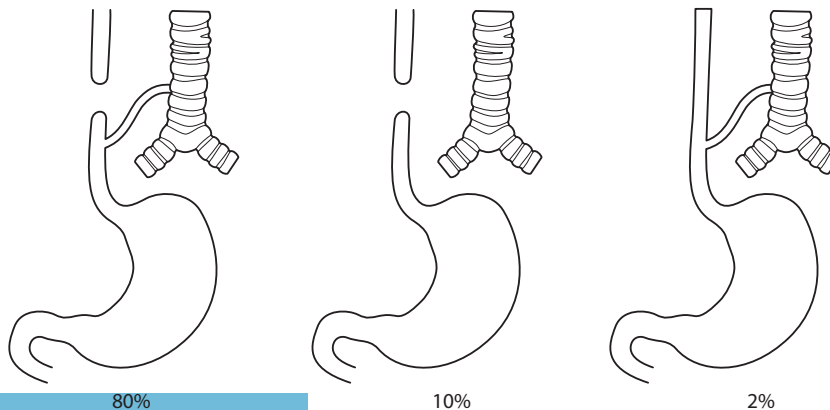
Figure 24.1 Common presentations of oesophageal atresia.

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birth. Later presentation can be sudden onset respiratory distress at times of feeding.

ROUTINE

- Haemoglobin
- Urea and electrolytes
- Cranial ultrasonography
- Single radiograph of chest and abdomen
- Cross-match blood

SPECIFIC

- Rectal examination
- Renal ultrasonography
- Echocardiography
- Blood gases if indicated, especially if premature

PREOPERATIVE MANAGEMENT

- Replogle tube to prevent aspiration of saliva.
- A small number of premature babies may require positive pressure ventilation. If lung compliance is low and the fistula large, immediate surgery may be the only method of achieving adequate ventilation.

THEATRE PREPARATION

- High theatre temperature
- Warming blanket
- Humidification of inspired gases

PERIOPERATIVE MANAGEMENT

INDUCTION

- In the operating theatre
- Thiopental and atracurium

TRACHEOSCOPY

- Requires a ventilating bronchoscope to identify the fistula position.
- A small percentage of cases will have upper and lower pouch fistulas.
- In some units, flexible fibre-optic bronchoscopy is an alternative.

MONITORING

- ECG
- SaO_2
- Noninvasive blood pressure
- $EtCO_2$
- Core temperature

Care should be taken with siting the monitors. The axillary artery in the uppermost arm may be compressed by surgical traction.

MAINTENANCE

- Oxygen and nitrous oxide with 0.5%–2% sevoflurane and fentanyl 1–2 µg/kg.
- Inspired oxygen may occasionally need to be increased during lung collapse.

SPECIAL CONSIDERATIONS

Many authors stress the importance of tracheal tube placement relative to the fistula in order to prevent excessive quantities of gas being forced into the stomach. This risk is believed to be overstated and normal-length tracheal tubes are usually sited irrespective of fistula position. Gastric distension is much more commonly the result of over-vigorous ventilation with high airway pressures; gentle hand ventilation is the best way of avoiding this. Lung collapse is produced by surgical retraction, which has the advantage of compressing both alveoli and blood supply together, making ventilation–perfusion mismatch uncommon. This method of collapsing the lung does, however, commonly result in intermittent tracheal obstruction due to overenthusiastic retraction by the assistant. Hand ventilation using a Jackson Rees T-piece allows obstruction to be detected instantly and also allows reinflation of the compressed lung periodically during periods of surgical inactivity.

IMMEDIATE POSTOPERATIVE PERIOD

Although TOF repairs can be managed without elective postoperative ventilation, it may be difficult to

predict which patients will require ventilation and which will not. In order to decrease the risk of collapse requiring emergency ventilation, often during the hours of darkness, patients with TOF repair in the author's unit are electively ventilated overnight. A small number of patients, particularly preterm babies, may require longer periods of ventilation. It is common surgical practice to request ventilation for difficult TOF repairs for periods of between 5 and 10 days postoperatively. In ventilated babies, care should be taken with both tracheal suction and physiotherapy to avoid the risk of disruption of the repair.

OUTCOME

- Good in TOF with no other abnormalities.
- Mortality is <1.5% for those without major cardiac abnormalities and birth weight >1500 g.
- Overall survival 90%
- If there are associated anomalies, mortality rate is much higher.
- Anastamotic leak occurs in 11%–21%.
- A small number have tracheal weakening at the site of the fistula and may require aortopexy, tracheopexy or, in severe cases, tracheoplasty before extubation is possible.
- 35%–60% of patients have gastroesophageal reflux disease, likely due to intrinsic oesophageal dysfunction.

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CROSS-REFERENCE

Infants and children, Chapter 25

PART

3

ANAESTHETIC FACTORS

25

Preoperative assessment

SANTOSH PATEL AND TOM WRIGHT

PREOPERATIVE ASSESSMENT – OVERVIEW

SANTOSH PATEL

Assessment differs according to general and specific medical and surgical conditions. Detailed assessment for individual medical and surgical conditions or groups of patients is discussed in other sections.

GOALS

1. To identify existing medical problems, their management and any implications.
2. To understand all surgical and procedural problems and their implications.
3. To determine current drug therapy.
4. To identify risk factors for morbidity and mortality and if possible apply measures to reduce risks, e.g. cardiac, respiratory, aspiration, renal, haematological, endocrine.
5. To prepare an appropriate anaesthesia and analgesia plan including regional technique, airway management and invasive monitoring.
6. To plan postoperative recovery, e.g. HDU or ICU.
7. To obtain consent for the plan and discuss routine and specific risk-benefits and address concerns.
8. If necessary, to seek a second opinion or advice from another specialist or refer to another specialist.
9. To prevent on-the-day cancellations.

CURRENT PROBLEMS

If admitted to the hospital for more than the expected time before surgery, explore the following:

- When were they admitted and why?
- What progress has been made since admission?
- Are there any functional limitations? If so, how severe and what are the causes? Functional limitation mIf not, when did they change?
- Review all available records.
- Discussion with colleagues as necessary.

HISTORY

MEDICAL

- Co-morbidities:
  - When was the diagnosis made and how severe is the problem?
  - Has the primary condition led to other systemic or secondary complications?
  - Is the condition optimally managed?
  - Is there appropriate regular follow-up?
  - Does the condition need any specific investigation?

SURGICAL

- Note all previous surgery, type of anaesthesia and any complications.
- Has previous management or current condition resulted in any anatomical (e.g. face, neck, spine, lung resection) or physiological (e.g. endocrine) changes?

ANAESTHETIC

- Note any previous anaesthetic or postoperative problems.
- Is there a history of difficult or failed intubation?
- Is there a history of failed regional technique?

DRUG HISTORY

- List all current and recent medications and the reason for each.
- Record any allergies.
- Remember non-prescription drugs and herbal remedies.

PERSONAL HISTORY

Smoking is associated with increased cardiopulmonary risks, impaired wound healing and in some cases prolonged hospital stay. Smoking may create a state of chronic inflammation and increase analgesic requirement by enzyme induction.

Any history of smoking must be sought in the preoperative assessment. This is an opportunity to advise and inform about perioperative risks. Many smokers are not aware of the risks of smoking during the perioperative period. Stopping smoking any time before surgery is beneficial although reductions in serious complications such as cardiorespiratory, requires abstinence for 6–8 weeks.

Patients may be referred to Quit Smoking clinics for counselling. Pharmacological replacement therapy may be considered for high risk surgery. Options include nicotine replacement therapy, nicotine partial agonists, buprprion, nortryptilline and clonidine. However, safety and efficacy of replacement therapy during the perioperative period is not well studied.

Alcohol consumption should be determined as should use or abuse of any social or psychoactive agents.

FAMILY HISTORY

Determine any family history of problems with anaesthesia, e.g. malignant hyperthermia, suxamethonium apnoea.

EXAMINATION

- Vital signs: Are they normal? If not, determine the cause and correct if possible.

SYSTEMATIC

- History may help to focus on specific areas.
- Are there any signs of organ impairment or failure?
- Are there any specific anatomic findings which may make procedures (e.g. invasive lines, regional techniques) difficult or impossible?
- Signs of hypo- or hyper-volaemia?
- What is the nutritional status?

- For regional and neuraxial blocks, inspect and palpate the relevant anatomical areas. Check appropriate sites are marked. Document any preoperative deficits, particularly neurological. These may absolutely or relatively contraindicate a specific regional anaesthetic technique. Ensure there is no local infection.
- Airway assessment

INVESTIGATIONS

In April 2016, NICE recommended that preoperative investigations should be based on type of surgery (minor, intermediate or major) and the ASA risk grading system (Table 25.1).

Additional investigations which may be indicated:

- *Chest X-ray* – Not routine. Preoperative X-ray findings usually do not alter perioperative management or predict postoperative pulmonary complications. Often new findings are already known clinically or a new finding is unmodifiable.
- Some suggested indications include acute respiratory symptoms, clinical signs suggestive of new pulmonary disease, unstable signs of previous pulmonary disease, recent history of chest trauma, likelihood of pulmonary metastases or cardiothoracic surgery.
- Many patients undergo advanced investigations, e.g. CT scan, which can provide more detailed information.
- *ECG* – A resting ECG is indicated in the presence of signs and symptoms of cardiovascular disease, high-risk elective or emergency surgery or intermediate risk surgery with at least one risk factor of a revised cardiac risk index. ECG is also useful in patients with pacemakers and with significant electrolyte abnormalities.
- *Liver function tests* – Not necessary as a routine in the absence of history of, or clinical evidence of, liver disease. Approximately 1 in 700 patients have incidental abnormal liver function which may be asymptomatic or herald underlying liver disease. Liver function tests comprise tests for cellular function (alanine aminotransferase and aspartate aminotransferase), bile excretion (bilirubin and alkaline phosphatase) and synthetic function (serum albumin and prothrombin time).
- *Blood group and save/cross-match* – If indicated it should be requested. Refer to local hospital policy.
- *Specialised investigations* – Additional cardiac, pulmonary, haematological and endocrine investigations may be necessary. An expert opinion should be sought. CPET may prove useful. A pregnancy test is needed in women of childbearing age.

FASTING

Clear fluids (water, pulp-free juice, tea or coffee without milk) are allowed up to 2 hours prior to induction. Solid foods and milk are allowed up to 6 hours before anaesthesia. There is no difference for obese, pregnant, gastrointestinal reflux and uncomplicated diabetic patients. Routine use of prophylaxis to prevent aspiration pneumonia is not recommended.

Carbohydrate-rich fluids are safe to use up to 2 hours before induction as a component of enhanced recovery.

CONSENT

In the UK, patients aged 16 and above can give consent. It is a legal and ethical requirement. Usually surgical consent is taken with preprinted consent forms. There is currently no separate written consent for anaesthesia required in the UK. Consent and related communication is a legal document. It may be used as evidence in case of legal disputes. Therefore, it is imperative that it is clear, concise and comprehensive.

Advanced decisions to refuse treatment come into effect if the individual loses mental capacity to make a decision. When the advanced decision was made, the individual must have had mental capacity to make that decision. Such decisions must be valid and applicable. For life-sustaining treatment including surgery and critical care, the decision must be written, signed by the patient in the presence of a signed witness, and must state that it applies even if life is at risk. Advice from a legal team may be necessary.

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Table 25.1 The National Institute for Health and Clinical Excellence guidance for preoperative laboratory investigations

	Minor surgery			Intermediate surgery			Major surgery		
	I	II	III or IV	I	II	III or IV	I	II	III or IV
ASA									
FBC	NR	NR	NR	NR	NR	If CVS or renal disease	Yes	Yes	Yes
Haemostatic	NR	NR	NR	NR	NR	Consider if chronic liver disease As per anticoagulant Local guidance	NR	NR	Consider if chronic liver disease As per anticoagulant Local guidance
Serum Na, K	NR	NR	NR	NR	NR	Yes	If patient at risk of AKI	Yes	Yes
Renal function	NR	NR	If patient at risk for AKI	NR	If patient at risk for AKI	Yes	If patient at risk for AKI	Yes	Yes
ECG	NR	NR	If not done in previous year	NR	If CVS or renal or diabetes	Yes	If age >65 and no ECG in last 12 months	Yes	Yes
Pulmonary function/ABG	NR	NR	NR	NR	NR	Seek anaesthetist's advice	NR	NR	Seek anaesthetist's advice

Source: National Institute for Health and Care Excellence. (2016). Adapted from *NG45 Routine preoperative tests for elective surgery.* Manchester: NICE. Available from https://www.nice .org.uk/ng45. Modified with permission.

Abbreviations: ABG = arterial blood gas analysis; AKI = acute kidney injury; ASA = American Society of Anesthesiologists; CVS = cardiovascular system; NR = not recommended.

PREOPERATIVE ASSESSMENT CLINICS

Nurse-led preoperative assessment clinics are increasingly utilized to screen patients for fitness. Such clinics have been found efficient and cost-effective. Patients can be assessed for anaesthesia several days before surgery. This allows preoperative diagnostic testing, minimises cancellations and delays for medical reasons on the day of surgery and reduces the length of hospital stay. It also provides sufficient time to discuss patients' priorities, issues and concerns which are central to patient consent, education, satisfaction and quality of care. They can be a one-stop centre for assessment and completion of other formalities, e.g. laboratory and imaging investigations.

The assessment is usually by face to face interview based on a structured questionnaire and form to complete. Such a structured approach helps to triage patients for further referrals. In some centres it can be performed via telephone or using a computerised online questionnaire.

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CROSS-REFERENCES

CARDIOPULMONARY EXERCISE TESTING (CPET)

TOM WRIGHT AND SANTOSH PATEL

Assessment of functional status is an integral part of preoperative assessment. Functional status reflects, to a degree, the overall cardiorespiratory status of the patient. Patients with poor cardiorespiratory status are less able to increase and sustain oxygen delivery in response to the increased oxygen demand as a result of the neuro-humoral stress response to surgery.

For major surgery or where there are concerns about cardiorespiratory status, objective assessment of their cardiorespiratory function is useful. Assessment tools such as the Duke activity status index and the incremental shuttle walk test are alternative techniques. Cardiopulmonary exercise testing (CPET) is safe and noninvasive and provides an accurate and objective assessment of functional capacity. Initially developed in sports medicine to establish peak V_{O2} it has become an established technique to diagnose and assess prognosis in heart failure, investigate dyspnoea of unknown aetiology and in preoperative assessment.

It should be remembered that exercise limitation could be due to several reasons:

- *Pulmonary* Impaired gas exchange, disease, respiratory muscle fatigue or chest wall problems
- *Cardiovascular* Reduced stroke volume, abnormal heart rate response, circulatory insufficiency
- Anaemia
- Malnutrition
- *Muscle* Atrophy, neuromuscular diseases, reduced oxidative capacity
- Environmental
- Lack of motivation

PHYSIOLOGICAL BASIS

Exercise tolerance is determined by pulmonary gas exchange, cardiac and vascular function and by skeletal muscle function and metabolism. CPET provides an integrated and global assessment of cardiovascular, respiratory and skeletal muscles (Figure 25.1) by measuring several physiological parameters related to oxygen delivery and consumption, and carbon dioxide production and expiration.

Older et al. (1999) have summarised indications and contraindications for preoperative CPET.

INDICATIONS FOR PREOPERATIVE CPET

Centres vary as to how they utilize preoperative assessment and this is an evolving area of practice. Current indications include:

- Lung resection surgery with moderate to high risk of postoperative dyspnoea to assess VO_{2max}.
- Patients undergoing elevated risk procedures in whom functional capacity is unknown.
- Patients >60 years undergoing major abdominal surgery (colorectal, upper GI, major HPB, urology, gynaecological–oncology).
- Patients undergoing planned AAA surgery.

CONTRAINDICATIONS TO CPET

- *Absolute* – Acute cardiac disease with unstable condition or potential to cause haemodynamic compromise (unstable angina and arrhythmia, severe symptomatic aortic stenosis), acute or uncontrolled respiratory disorder (e.g. uncontrolled asthma, acute
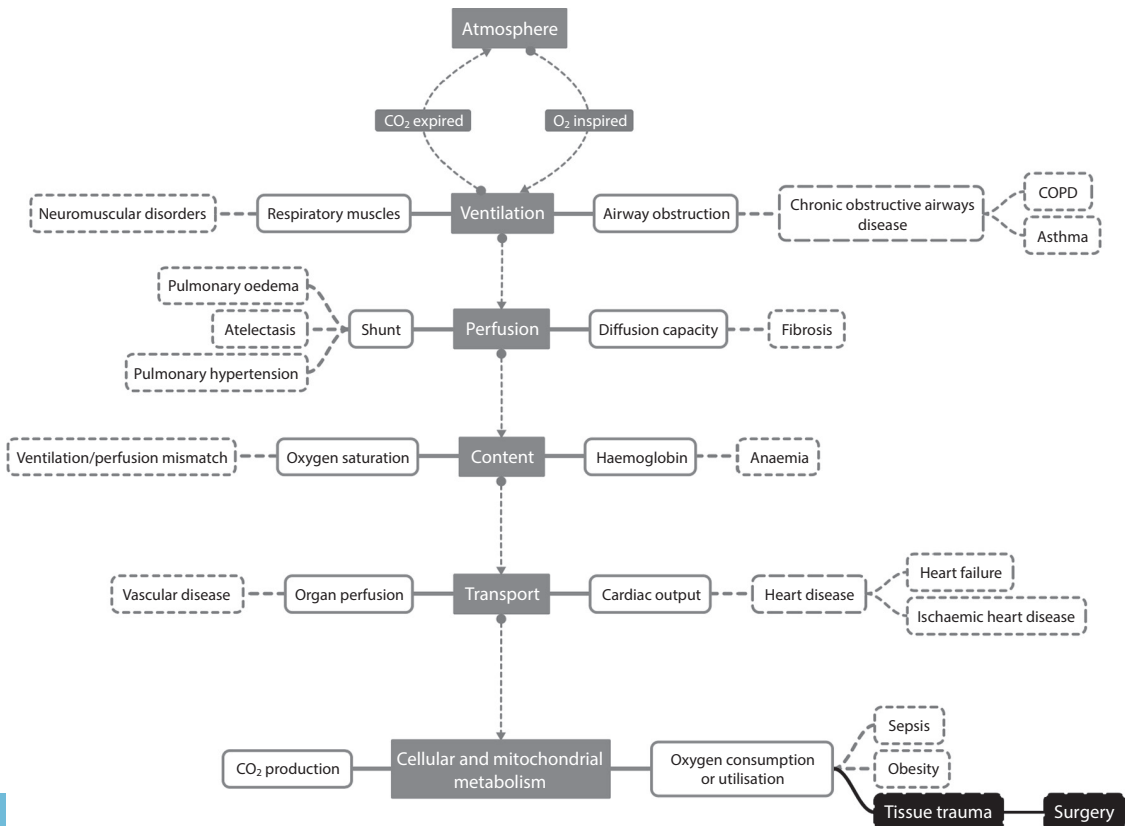
Figure 25.1 Physiopathologic factors affecting CPET variables.

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pulmonary embolism, SpO$_2$ <88% on room air), deep vein thrombosis, acute systemic illness that may limit exercise, significant psychiatric/cognitive impairment limiting cooperation.

- *Relative* – Left main or three vessel coronary artery disease, moderate stenotic valvular heart disease; severe arterial hypertension, significant pulmonary hypertension; tachy- or bradyarrhythmia; hypertrophic cardiomyopathy; electrolyte abnormality.

EQUIPMENT AND CONDUCT OF CPET

EQUIPMENT REQUIRED

- Metabolic cart capable of breath-to-breath O$_2$ and CO$_2$ analysis and gas flow analysis
- Monitoring
  - SpO$_2$
  - NIBP
  - 12 lead ECG
- Cycle ergometer or (less commonly) a treadmill
- Resuscitation equipment

CONDUCT OF CPET

Before starting, patients should have a clinical assessment, including identification of contraindications. Informed consent should be obtained. The patient should wear loose fitting clothing. Ideally two members of appropriately trained staff should be present throughout the test.

Several protocols are described. Where a cycle ergometer is used, a ramp protocol is typical; a continuous increase in the work rate required through the duration of the test.

The patient is connected to the monitoring, inserts the mouthpiece and applies the nose clip. Baseline observations are made and spirometry performed to establish FEV$_1$ and FVC.

The patient begins cycling at a cadence of 50–60 rpm against no resistance for 3 min.

Resistance is increased in a ramp-like fashion at 10–25 Watts/min maintaining a cadence of 60 rpm. The rate of increase of resistance is calculated using the Wasserman equation based on predicted V$_{O2}$

peak and V$_{O2}$ unloaded to enable the loaded exercise section of the test to last 6–10 min.

Full monitoring is maintained after termination of the test until physiological parameters have returned to normal. The test may be terminated prior to completion. Possible reasons for early termination include patient exhaustion, chest pain, myocardial ischaemia identified on ECG or hypoxia.

CPET PARAMETERS

Measurements are made of a range of variables. A single CPET test generates large amounts of data. Much of this is presented on a '9 panel plot', a chart of 9 graphs. Detailed analysis can identify causes of exercise limitation. For example, differential increase or decrease in the value of certain parameters may help to determine whether the origin of exercise limitation is cardiac, pulmonary or effort related (Table 25.2). In the context of preoperative assessment, certain parameters are of particular interest to aid preoperative decision making and these are explored next.

ANAEROBIC THRESHOLD

This describes the V$_{O2}$ (in mL/kg/min) at which cellular respiration becomes predominantly anaerobic. To supply increasing metabolic demand of muscle, aerobic respiration is insufficient and anaerobic metabolism with glycolysis becomes more predominant. The anaerobic threshold can be identified in several ways. Most commonly it is identified by finding the point at which during increasing exercise intensity the linear relationship between increasing V$_{O2}$ and V$_{CO2}$ changes. There is a break point where V$_{CO2}$ starts to climb more rapidly than V$_{O2}$. The V$_{O2}$ at which this occurs is the anaerobic threshold.

Work by Older et al. published in 1999 demonstrated that patients undergoing major surgery with an anaerobic threshold lower than 11 mL/kg/min had higher mortality than those whose anaerobic threshold was above this level. This value is therefore commonly used to distinguish high- from low-risk patients. The concept of the anaerobic threshold is not without controversy and its physiological basis is debated. However, it is a widely referenced parameter when using CPET in preoperative assessment.

Table 25.2 CPET variables in specific conditions

	Normal	Cardiac disease (e.g. CCF)	Pulmonary disease – COPD	Effort (e.g. poor effort or neuromuscular disease)
VO_{2max}	N	↓	↓	↓
Heart rate reserve	Absent or small reserve	Absent or small reserve	Large reserve (>30 beats/min)	Large reserve (>30 beats/min)
Breathing reserve	<0.8	<0.8	>0.8	<0.8
SpO_2	N	N	↓	N
$EtCO_2$	↓	↓	↑ or N	↑ or N

Note: Breathing reserve = Minute ventilation achieved/maximum voluntary ventilation; Heart rate reserve = Difference between observed maximal heart rate and predicted maximal heart rate; N = Normal; ↑ = increase; ↓ = decrease.

PEAK OXYGEN CONSUMPTION

Peak oxygen consumption is the maximum V_{O2} (mL/kg/min) achieved by the patient on that test. It differs from VO_{2max}, which is the highest V_{O2} attainable by that individual. VO_{2max} can be identified by failure to increase V_{O2} in response to increasing exercise intensity. It occurs close to exhaustion. Achieving VO_{2max} may not be possible or desirable in many patients undergoing CPET.

VENTILATORY EQUIVALENTS FOR CO_2 (V_E/V_{CO2})

This parameter describes the ratio between V_E and V_{CO2}; the minute ventilation required to eliminate a given volume of CO_2 per minute. It is an index of ventilatory efficiency that reflects ventilation/perfusion matching. In a healthy individual, the ratio is 25–35. During exercise, the ratio decreases until it reaches its lowest point at the anaerobic threshold. Thereafter it increases as metabolic acidosis ensues. The value at anaerobic threshold is normally reported and a value >34 is considered abnormal. An increase in the V_E/V_{CO2} is associated with adverse outcome from surgery (Table 25.3).

ROLE OF CPET IN PREOPERATIVE ASSESSMENT

RISK STRATIFICATION

Patients may be stratified according to CPET parameters, the presence or not of cardiac ischaemia and the surgery to be undertaken

RISK MODIFICATION

Causes of exercise limitation, such as myocardial ischaemia, may be identified. Subsequent specialty review, pharmacological therapy, smoking cessation advice or exercise programmes will aid in modifying individual patient risk.

INFORMED CONSENT

An individualised risk profile for a patient undergoing major surgery can be made and fed back to patients improving the consent process.

PLANNING SURGICAL TREATMENT

Surgical technique may be influenced by CPET data. In vascular surgery, the decision to proceed with EVAR or open repair of AAA in some centres incorporates the outcome of CPET.

PLANNING POSTOPERATIVE CARE

Accurate risk stratification can help determine postoperative destination and the appropriate allocation of critical care resources.

LIMITATIONS OF CPET

- Several technical protocols for CPET exist.
- There is variation in studies for parameter chosen to quantify exercise capacity.
- There is variation in cut-off values of chosen parameters.

Table 25.3 Some studies evaluating surgical outcome using CPET parameters

Publication	Patient group	Summary
Older et al. 1993	• Elective major abdominal surgery • Age >60 years • n = 187	• AT <11 mL/kg/min associated with increased mortality (18%) versus AT >11 mL/kg/min (0.8%) • Highest mortality (42%) in patients with AT <11 mL/kg/min and history of ischaemic heart disease
Older et al. 1999	• Elective major abdominal surgery • Age >60 or <60 if history of ischaemic heart disease or heart failure • n = 548	• Patients stratified into 3 management groups by AT and ECG evidence of ischaemia during CPET • No deaths in low risk group managed on ward (AT >11 mL/kg/min and no myocardial ischaemia) • Mortality 4.8% in high-risk group managed on ICU (AT <11 mL/kg/min)
Carlisle et al. 2007	• Elective abdominal aortic aneurysm repair • n = 130	• Ventilatory equivalents of CO_2 >42 associated with decreased 30-day and midterm survival (median 35 months)
Hartley et al. 2012	• Elective abdominal aortic aneurysm repair. Open and EVAR • n = 415	• AT <10.2 mL/kg/min associated with increased 30-day mortality • Peak VO_2 <15 mL/kg/min associated with increased 90-day mortality
Moyes et al. 2013	• Patients undergoing elective, curative oesophago-gastric cancer resection • Median age 66 years (range 38–84) • n = 108	• 42% of patients with AT <9 mL/kg/min developed cardiopulmonary complications • 20% of patients with AT >11 mL/kg/min developed cardiopulmonary complications
West et al. 2014	• Patients undergoing major colon surgery • n =136	• Increased complications at day 5 in patients with AT <10.1 mL/kg/min, and VO_2 at peak with a <16.7 mL/kg/min

- Most studies do not weigh the risks due to the presence of systemic disease (e.g. IHD, renal disease) in addition to CPET measured variables.
- It is possible that cutoff values for measured parameters of exercise capacity may be surgery-specific. No uniform value exists for the wide spectrum of surgical patients.

SUMMARY

CPET is increasingly utilised to provide objective functional assessment of patients undergoing high risk surgery. The most appropriate parameters to reference and the values of these are evolving as the evidence base increases. Most anaesthetists are likely to encounter patients who have undergone CPET and should be familiar with the principles and variables relevant to preoperative assessment.

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DAY-CASE SURGERY

SANTOSH PATEL

A day-case (ambulatory) patient is one who is an inpatient for several hours for purposes of investigation or minor surgery and does not stay overnight in the hospital.

ADVANTAGES

- No dependency on hospital inpatient beds.
- Less disruption to family life – useful for children and elderly patients.
- More pleasant for the patient and the family.
- Cost effective – less cost to hospital or individuals (early return to work).
- Greater flexibility to schedule surgical list.
- Improve efficiency to conduct surgical list.
- Waiting time is reduced.

- Less risk of hospital-acquired infections in susceptible individuals.
- Incidence of postoperative respiratory complications is decreased.
- Reduced postoperative confusion in the elderly.

LIMITATIONS

- Unplanned postoperative admissions may result.
- Constraints to schedule as a day case – medical (e.g. bleeding disorders) and social (lack of home support).
- If any complication occurs requiring hospital admission, an inpatient bed may not be available, delay in patient management, safety concerns and even transfer to another facility or hospital may be needed.

PREOPERATIVE ASSESSMENT

An anaesthetic assessment clinic approach is used in most centres. Patients should not be seen more than about 3–4 weeks before admission; otherwise, the patient's condition might have changed. Patients are usually seen and assessed by a nurse using a questionnaire-type instrument with further referral on to a consultant anaesthetist for advice in specific patients. Any blood tests or other investigations are ordered at the clinic visit. If any doubt remains, the surgeon and anaesthetist discuss the case individually. Patients are advised to telephone and defer their surgery if they develop an acute upper respiratory tract infection.

A self-assessment questionnaire is used in some centres. This is sent to the patient in advance of their admission with a request to complete and return it. This works well, provided the questions are relevant and well worded. The range of topics which can be covered is wide (Table 25.4), and each day-case unit will have its own individual version. Interactive assessment by computer is being used in some centres.

PREOPERATIVE STARVATION

The normal rules apply. It is imperative to emphasize the importance of preoperative starvation to the

Table 25.4 Example patient information sheet

Date, time, place of admission

Nature of procedure and anaesthetic

Preoperative starvation rules and surgical preparation requirements

What to do about taking any preoperative medication

Arrangements for a responsible adult for transport home

No driving, alcohol, operating machinery, etc. for 24 h

Postoperative analgesic advice

Day-case unit phone numbers (routine and emergency)

What to do if there is an acute change in health, a cold develops or the patient thinks she might be pregnant

Advice on time off work, follow-up appointments, etc.

Table 25.5 Guidelines for patient selection for anaesthesia

ASA status I or II

ASA III is acceptable with stable comorbidities and the decision made on an individual basis

Avoid procedures which are likely to last for longer than about 2 hours

Avoid procedures where severe postoperative pain is likely

Avoid procedures where significant postoperative bleeding is likely

Patient should live within 1 h drive of the unit

There must be a responsible adult to escort the patient home

patient because it is less easy to be certain that the patient has not eaten than when an in-patient.

INVESTIGATIONS

These are organized at the original booking clinic or the assessment clinic. There are no routine investigations which are always indicated. There should be a valid reason for each test. Some may need repeating (e.g. blood sugar, coagulation) on the morning of surgery.

SELECTION CRITERIA

Inappropriate selection will hamper the smooth working of the unit and lead to operations being cancelled or deferred. The day-case unit requires a series of guidelines of patients and operations which are suitable (Table 25.5). Consider the potential suitability for a local or regional block.

Ages 6 months to 70 years are usually accepted. A minimum of 1 month may be accepted provided the infant was not a preterm delivery. Patients over 70 years of age are considered on an individual basis depending on comorbidities.

Systemic diseases, e.g. morbid obesity, obstructive sleep apnoea, uncontrolled type 1 diabetes and severe asthma need individual consideration.

There must be a responsible adult to accompany the patient home. If the patient lives a long distance

from the facility, they should stay overnight within 60 minutes journey time from the facility. The patient should be able to follow postoperative instructions.

Each patient should receive an information pack at the booking clinic. This is also an opportunity to list important points that the patient needs to know about the postoperative period (Table 25.5). The patient should also receive an information sheet on discharge, re-emphasizing the essential points.

Most minor, and many intermediate, procedures are suitable and the list is constantly growing as surgical techniques (e.g. laparoscopy) become more refined. Surgery that is likely to have significant postoperative complications, e.g. bleeding, significant pain, multiple drains, haemodynamic instability, delayed oral intake and specialist postoperative monitoring are not suitable as day cases.

ANAESTHETIC MANAGEMENT

- *Induction* – Use a short-acting agent, e.g. propofol, desflurane, sevoflurane.
- *Maintenance* – Desflurane and sevoflurane may be associated with a better recovery profile than the older agents. A total intravenous (TIVA) technique using propofol is also suitable and may be associated with less PONV although claims of earlier discharge and less readmissions with TIVA have not been substantiated.

- *Analgesia* – Alfentanil or fentanyl are popular because of their potency and short duration of action. Remifentanil may be used. Intravenous NSAIDs (e.g. ketorolac, parecoxib) are commonly used. Local infiltration, intra-articular, intraperitoneal and nerve blocks may facilitate analgesia. In selective cases, non-opioid adjuvant analgesics such as gabapentoids, dexamethasone, lidocaine and ketamine may be useful.
- *Muscle relaxants* – Short or intermediate duration agents are recommended (e.g. mivacurium, atracurium, vecuronium, rocuronium). The incidence of muscle pains following suxamethonium is high in ambulatory patients and it should be avoided. Mivacurium does not always need to be reversed with an anticholinesterase agent, which avoids the potential adverse effects of this family of drugs. Sugammadex may be useful.
- *Regional blocks* – A potentially better option for patients with comorbidities such as sleep apnoea and diabetes. There is better postoperative pain relief, less PONV, no hangover, shortened recovery time, less nursing dependence and improved patient satisfaction. They can however be time consuming and require additional resources (e.g. ultrasound machine). The occasional need for sedation, inadequate or failed block, unexpected prolongation of surgery and urinary retention may be disadvantageous. Intravenous regional anaesthesia is also a safe, reliable and easy option for forearm and hand surgery lasting for less than an hour.
- *Neuraxial blocks* – Haemodynamic effects of spinal anaesthesia may be minimised by a unilateral block. In the UK, hyperbaric 2% prilocaine has become popular for spinals in day-case surgery. Between 30 and 60 mg is normal but lower doses may be combined with 10–25 μg of fentanyl. Early onset and early return of sensory and motor functions and early return of bladder function are advantageous.

POSTOPERATIVE CARE

Postoperative pain is a common complaint in outpatient surgery. It is a common cause for delay in discharge, may lead to inpatient admission or readmission and nullify some benefits of day surgery, e.g. early return to work or school. A multimodal approach is advised. In some cases with difficult postoperative control, non-opioid adjuvants may be administered. Some centres have reported safe, reliable and efficacious use of patient-controlled regional analgesia with the use of catheters.

Risk factors for PONV should be identified at preoperative assessment. Appropriate prophylaxis with one or multiple antiemetics is then given during the intraoperative period. If PONV occurs, prompt control is desirable.

DISCHARGE

Return to 'street fitness' is the most important factor in discharge. Each day-case unit should have a list of guidelines for safe discharge of patients (Table 25.6). In general, most patients are fit enough to go home after about 3–4 h.

POSTOPERATIVE PROBLEMS

Major morbidity or mortality after day-case surgery has been reported to be about 1 in 1000. Principle predictors of postoperative major complications include COPD, hypertension, previous coronary interventions, previous transient ischaemic attack/cerebrovascular accident, obesity and prolonged surgical time. Early postoperative pneumonia, unplanned

Table 25.6 Discharge guidelines

Vital signs stable for at least 1 h
No respiratory depression or undue drowsiness
Able to eat and drink and to pass urine
Able to walk unaided
No significant pain, bleeding, nausea or vomiting
Written instructions have been given to the patient and read by either patient or escort or both
Responsible adult present

postoperative intubation and wound disruption are among the most common morbidities. The facility should always be ready for admission of a patient for overnight stay if necessary. Such reasons might include: extended surgery or surgical complications, perioperative complications, persistent PONV, difficulty with pain control, prolonged drowsiness and lack of a suitable escort.

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Thrombotic embolism, Chapter 30
Spinal, epidural, CSE, Chapter 29

PREOPERATIVE RISK ASSESSMENT

TOM WRIGHT

Risk is defined as the likelihood of an adverse event occurring. Risks are inherent to all medical interventions. Determination of risk is a core component of all preoperative assessment. The content of that assessment will vary depending on the individual circumstances of the patient and the nature of the proposed procedure.

Risk assessment of patients undergoing surgery has many purposes, including:

- To facilitate informed consent.
- To aid decision making with regard to whether to proceed with a surgical intervention.
- Plan for use of resources, e.g. critical care.
- To target strategies to reduce risk.

No risk assessment can determine with certainty what will happen. Appropriate use of a validated risk assessment tool allows description of the probability of an adverse outcome in a population of patients similar to the one to which it is applied.

A variety of risk assessment tools exist to aid determination of the likelihood of an adverse outcome. These range from assessing the probability of specific events (e.g. PONV) to the probability of major cardiorespiratory complications or death. For many adverse outcomes, risk assessment tools do not exist so an understanding of risk factors may aid the planning of perioperative management.

SCORING SYSTEMS FOR MORBIDITY AND MORTALITY

AMERICAN SOCIETY OF ANESTHESIOLOGISTS (ASA) PHYSICAL STATUS CLASSIFICATION

The ASA grading system is extensively used in daily practice. Initially formulated in 1962 by the ASA with five categories, a sixth was later added for organ donors. A suffix of 'E' is added to further describe the case as an emergency. The different grades are described as follows:

- ASA 1 is a healthy, non-smoking patient.
- ASA 2 patients have mild systemic disease, such as well-controlled asthma or diabetes.
- ASA 3 patients have severe systemic disease resulting in functional limitation, such as stable angina or symptomatic COPD.
- ASA 4 patients have severe systemic disease, which represents a constant threat to life. Examples include recent MI or sepsis.
- ASA 5 patients are moribund and not expected to survive without surgical intervention. For example, ruptured abdominal aortic aneurysm or bowel perforation with septic shock.

Although widely used, the ASA grading system has limitations, not least that there is significant inter-observer variation in grading. The list of conditions described and categorised by the ASA is not exhaustive. Its prognostic ability is limited, although mortality values have been described for the different grades. The grading system does not take into consideration other important factors, such as physical fitness, the effects of ageing or the degree of surgical insult the patient is to encounter.

The ASA system is quick and easy to use and allows communication between health professionals. It is also used to identify patients who should have appropriately experienced anaesthetists present, e.g. a department may specify ASA IV patients should be anaesthetised by a consultant.

ACUTE PHYSIOLOGY AGE AND CHRONIC HEALTH EVALUATION VERSION II (APACHE II)

APACHE II is used for critically ill patients within 24 hours of admission. It is of relevance in the context of critically unwell patients undergoing emergency surgery. In use since 1985, it consists of three scores in different domains.

- Physiology (12 separate variables)
- Age
- Chronic health

Patients are assigned a numeric score between 0 and 71. Higher scores are associated with higher risk of mortality. The figure assigned will vary depend on the presenting pathology. A score of 25 equates to a predicted mortality of approximately 50%.

PORTSMOUTH PHYSIOLOGICAL AND OPERATIVE SEVERITY SCORE FOR THE ENUMERATION OF MORTALITY AND MORBIDITY (P-POSSUM)

The original POSSUM equation was described in 1991 and amended in 1998 to become P-POSSUM. The score comprises two components: a physiology score and an operative severity score (Table 25.7). It is widely used preoperatively in assessing risk of mortality in patients undergoing emergency

Table 25.7 P-POSSUM variables

Physiological parameter	Operative parameter
Age	Operation type (minor, moderate, major, complex major)
Cardiac co-morbidity	Number of procedures
Respiratory co-morbidity	Operative blood loss
ECG findings	Peritoneal contamination
Systolic blood pressure	Malignancy status
Pulse rate	
Haemoglobin	
White cell count	
Urea	
Sodium	
Potassium	
GCS	

laparotomy. Criticisms are that it can overestimate risk and the operative parameters often change once operative findings are confirmed. Adaptations of the original POSSUM equation have been developed to apply to different surgical populations, such as V-POSSUM for vascular patients.

POSTOPERATIVE NAUSEA AND VOMITING (PONV)

PONV is a common and distressing symptom for patients. Assessment of the risk of PONV allows measures to be implemented to minimise the occurrence. The Apfel score is well recognised for stratifying patients into risk groups. Patients score 1 point for each risk factor present (Table 25.8) and potential PONV can then be assessed (Table 25.9).

PULMONARY COMPLICATIONS

Postoperative pulmonary complications may include pneumonia, atelectasis, respiratory failure and pneumothorax. Whilst no individual risk assessment tool exists to guide preoperative assessment of nonthoracic surgery, several factors associated with the development of postoperative pulmonary complications have been identified (Table 25.10).

Table 25.8 Risk factors associated with increased risk of PONV

Patient factors	Anaesthetic factors	Surgical factors
Female	Volatile anaesthesia	Ophthalmic surgery
Non-smoker	Use of nitrous oxide	Gynaecological surgery
Young	Opioids	Emergency surgery
ASA I or II status	Duration of anaesthesia	
History of PONV or motion sickness	Gastric insufflation from face mask ventilation	

Table 25.9 Apfel score and associated risk of PONV

No. of risk factors	Baseline % risk of PONV
0	10
1	20
2	40
3	60
4	80

PERIOPERATIVE ATRIAL FIBRILLATION (AF)

AF is the most commonly encountered arrhythmia perioperatively and is associated with a risk of stroke both short term and long term following surgery. There are several well-recognised risk factors for the development of AF (Table 25.11). Risk assessment for probability of stroke associated with AF is performed using the CHA_2DS_2-VASc score. This scoring tool contains seven variables with a maximum score of nine. The variables incorporated in the CHA_2DS_2-VASc are age, gender, history of heart failure, hypertension, stroke, vascular disease and diabetes. A score of two or greater is considered high risk and anticoagulation should be considered.

PERIOPERATIVE DELIRIUM/ COGNITIVE DYSFUNCTION

Delirium is an acute onset of disturbed mental function characterised by fluctuating consciousness and impaired attention. It is a common complication in older patients undergoing major surgery and is associated with prolonged length of stay and increased mortality. Postoperative cognitive dysfunction is cognitive impairment detected on neuropsychological testing following surgery. Awareness of the risk factors associated with deterioration in cognitive function following surgery allows high risk patients to be identified

Table 25.11 Risk factors for peri-operative AF

Risk factors for AF	
Increasing age	Chronic kidney disease
Hypertension	COPD
Ischaemic heart disease	Diabetes
Congestive cardiac failure	Hyperthyroidism
Alcohol consumption	Hypokalaemia/ hypomagnesaemia/ hypocalcaemia

Table 25.10 Risk factors for pulmonary complications

Patient factors	Anaesthetic factors	Surgical factors	Investigations
Advancing age	Increased duration of anaesthesia (>2.5 hours)	Aortic aneurysm surgery	Abnormal preoperative chest radiography
ASA III/IV status	General anaesthesia	Abdominal surgery	Albumin <35 g/L
History of smoking		Neurosurgery	
Daily productive cough		Emergency surgery	
Congestive cardiac failure			
Chronic respiratory disease			
Impaired functional status			

Table 25.12 Risk factors for peri-operative delirium

Nonmodifiable	Modifiable	Triggers
Age >65 years	Infection	Acute pain
Pre-existing cognitive impairment	Electrolyte disturbance	Physical restraint
Visual/hearing impairment	Hypoxia/hypercarbia	Urinary catheterisation
Impaired functional status	Use of benzodiazepines, opioids, anticholinergics or antihistamines	Addition of 3 or more new medications in 24 hours
Hip fracture	Polypharmacy	Anaemia from surgical blood loss
Vascular surgery	Malnutrition	

and strategies targeted to reduce risk. Risk factors for perioperative delirium are described in Table 25.12.

Regional techniques are associated with a lower incidence of delirium in comparison to general anaesthesia. Deeper levels of sedation are associated with greater incidence of delirium. The use of benzodiazepines should be minimised and where possible regional techniques employed to reduce opioid use.

COMMUNICATING RISKS OF ANAESTHESIA

Communication of risk is an integral part of informed consent. The nature of the risks that should be explained is not defined in absolute terms. Explaining to every patient every potential adverse outcome is impractical and would likely result in cancellation of a large numbers of procedures. The AAGBI guidance regarding consent states that when considering what information to discuss the anaesthetist should consider, 'What would this patient regard as relevant when coming to a decision about which of the available options to accept?'

In general, patients should be informed of the risks associated with anaesthesia (Table 25.13). Often, this can be discussed in the context of describing the usual experience of undergoing anaesthesia. Dental damage is the most common source of medico-legal claims against anaesthetists. As such, it is prudent to discuss this risk with all patients.

Major complications are those that require significant additional treatment or may result in harm to the patient (physical or psychological). Those complications relevant to making a decision should be discussed.

Table 25.13 Examples of risks associated with general anaesthesia and estimated incidence

Risk	Incidence	Risk	Incidence
PONV	1/4 overall	Anaphylaxis	1/10,000–1/20,000
Sore throat	LMA 1/5 ETT 2/5	Aspiration	1/900–1/10,000
Dental damage	1/100	Malignant hyperthermia	Exact incidence unknown Estimated 1/5000–1/50,000
Shivering	1/4	Death	1/100,000
Eye injury	Corneal abrasion 1/2800 Sight loss estimated 1/60,000–1/125,000		
Peripheral nerve injury	1/1000		
Awareness	GA with relaxant 1/8000 GA without relaxant 1/136,000		
Failed intubation	Overall 1/1000–1/2000 Obstetrics 1/250		

Table 25.14 Examples of risks associated with central neuraxial blocks and estimated incidence

Risk	Incidence
Postdural puncture headache	Overall approximately 1/200.
	Varies with gauge of needle. For spinals, a wider gauge needle is associated with higher risk.
Temporary nerve injury	Differing rates quoted in literature due to varying definition. Approximately 1/3900–1/5000.
Permanent nerve injury	1/23,500–1/50,500
Total spinal	No precise data. Thought to be <1/100,000
Meningitis	<1/50,000
Death or paraplegia	1/54,500–1/141,500

REGIONAL ANAESTHESIA

Peripheral nerve or plexus block have both generic and specific risks. Generic risks of all peripheral nerve blocks include:

- Temporary nerve injury
- Permanent nerve injury
- Allergic reaction
- Local anaesthetic systemic toxicity

The risks associated with central neuraxial techniques are described in Table 25.14. Some examples of specific complications to particular blocks are listed in Table 25.15.

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Table 25.15 Examples of complications associated with specific nerve blocks

Technique	Risk
Inter-scalene block	Horner's syndrome
Supra-clavicular block	Pneumothorax
Retrobulbar block	Globe perforation
Transverse abdominal plane (TAP) block	Bowel injury
Ilio-Inguinal nerve block	Leg weakness secondary to femoral nerve block
Lumbar plexus block	Retroperitoneal haemotoma

PREOPERATIVE ASSESSMENT – SPECIFIC MEDICAL PROBLEMS

TOM WRIGHT

PATIENTS WITH CARDIAC DISEASE UNDERGOING NON-CARDIAC SURGERY

Perioperative cardiovascular complications, including myocardial infarction, pulmonary oedema and serious arrhythmias, carry a high risk of mortality and long-term morbidity. Preoperative identification of patients at high risk of these complications is of paramount importance.

Several risk stratification tools have evolved to aid assessment of the patient with cardiac disease. The Goldman risk index (1977) comprises nine variables, each with different weighted scores.

Table 25.16 Risk of death, non-fatal MI or non-fatal cardiac arrest with increasing number of revised cardiac risk index predictors

No. of predictors	Risk of cardiac death, non-fatal MI or non-fatal cardiac arrest
0	0.4%
1	0.9%
2	6.6%
3 or more	>11%

This was modified by Detsky (1986) and again by Lee (1999) who produced the Revised Cardiac Risk Index (RCRI). RCRI is a simplified and easier to use scoring system which has been incorporated into cardiac risk evaluation guidelines. RCRI has been validated in many studies to estimate the risk of major cardiac complications (Table 25.16).

REVISED CARDIAC RISK INDEX

1. History of ischaemic heart disease
2. History of congestive cardiac failure
3. History of cerebrovascular disease (including TIA)
4. History of diabetes requiring preoperative use of insulin
5. Chronic kidney disease (creatinine >177 micromol/L)
6. Planned supra-inguinal vascular, intra-peritoneal or intra-thoracic surgery

CLINICAL FEATURES

Chest pain

Anginal chest pain can be graded using the Canadian Cardiovascular Society classification (Table 25.17).

Shortness of breath

The NYHA scale (Table 25.18) is a commonly used classification to grade the severity of symptoms, particularly shortness of breath associated with heart failure. It also incorporates other symptoms associated with heart failure, such as fatigue and chest pain.

Syncope

A history of transient loss of consciousness may reflect underlying cardiovascular disease and should

Table 25.17 Canadian Cardiovascular Society grading of angina

Grade	Description
I	Angina during prolonged or intense physical activity
II	Slight functional limitation only with vigorous activity
III	Symptoms during activities of daily living
IV	Unable to perform any task with angina or angina at rest

Table 25.18 NYHA grading of shortness of breath in heart failure

NYHA grade	Description
I	Cardiac disease, but no limitation in ordinary physical activity
II	Mild symptoms with slight limitation of physical activity
III	Marked limitation of activity due to symptoms
IV	Severely limited and experiences symptoms at rest

be taken seriously if elicited during preoperative assessment, particularly if the patient has established cardiac disease or risk factors. Examples of pathology associated with syncope which need excluding include:

- Aortic stenosis
- Bradyarrhythmias
- Hypertrophic obstructive cardiomyopathy

INVESTIGATIONS

Twelve lead ECG monitoring is commonly performed as part of preoperative assessment. Guidelines vary but it is generally recommended for:

- The presence of risk factors for coronary artery disease
- A clinical history of ischaemic heart disease, congestive cardiac failure or cerebrovascular disease
- ASA 3 or 4 patients undergoing all but minor surgery

ECG abnormalities associated with perioperative cardiac morbidity include:

- Rhythm other than sinus
- ST-T wave abnormalities
- Left ventricular hypertrophy
- Pathological Q-waves
- Conduction abnormalities

Echocardiography is not routinely recommended before surgery. Indications include suspected valvular lesions, unexplained breathlessness or syncope and to evaluate signs and symptoms of heart failure. Left ventricular ejection fraction of less than 30% is associated with worse perioperative outcomes in non-cardiac surgery.

Stress testing can be either exercise or pharmacological. Patients with elevated risk and poor/unknown functional capacity should be considered for stress testing, including CPET. Pharmacological stress testing includes Dobutamine stress echo and myocardial perfusion imaging. Negative stress testing is strongly predictive for low risk of myocardial infarction.

Angiography is the 'gold standard' to assess coronary anatomy, but not routinely recommended for preoperative assessment in patients with cardiac disease. The ACC/AHA recommends its use for patients who have a conventional indication to undergo coronary angiography. Where coronary revascularisation is indicated, it is recommended that this takes place prior to surgery. Patients who have undergone coronary angiography with PCI may need to have surgery deferred as discontinuing anti-platelet therapy risks stent thrombosis and subsequent MI. The length of delay prior to undergoing surgery depends on the nature of the coronary intervention made.

- Balloon angioplasty: 14-day delay
- Bare metal stent: 30-day delay
- Drug eluting stent: 365-day delay

Coronary computed tomography angiography is an increasingly used tool for assessment of coronary anatomy. Current indications include the diagnosis of coronary artery disease in low/intermediate risk patients, establishing in-stent stenosis and for establishing graft patency. It does not currently have an established role in preoperative assessment.

However, studies have been undertaken which suggest a potential role in future practice.

ADDITIONAL CONSIDERATIONS

The number of patients with pacemakers and ICDs is increasing significantly and these patients present for incidental surgery. Knowledge of the indication for use, the device and its current functioning are essential for the provision of safe anaesthesia.

PATIENTS WITH PULMONARY DISEASE

A thorough history is mandatory:

- Symptoms (dyspnoea, cough, haemoptysis, weight loss, chest pain)
- Smoking history
- Functional status, including the recent trend
- Recent exacerbations of disease, including hospital and ICU admissions
- Medications, including any recent changes
- Travel history
- Industrial exposure

Routine examination should include:

- Baseline observations (respiratory and heart rate, oxygen saturations)
- Inspection of the chest
- Percussion
- Auscultation

Assess for evidence of right heart failure (cor pulmonale):

- Raised JVP
- Hepatic congestion
- Peripheral oedema

Preoperative chest X-rays are not a routine screening test as it has been shown to not have any significant influence on patient management. Preoperative chest X-rays are appropriate in the following:

- Age >60 years with cardiorespiratory disease
- Planned major abdominal surgery with cardiorespiratory disease
- Assessment of dyspnoea
- Assessment of cough

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- Heavy smokers who have not had a chest X-ray in the last 12 months
- Recent immigrants from TB endemic countries

In patients with obstructive airways disease, consider measuring PEFR. Outside of thoracic surgery the precise role of spirometry in preoperative assessment is not clearly defined. FEV_1 less than 1 L is associated with increased postoperative pulmonary complications. However, even patients with an FEV_1 of 0.5 L may cope without requiring postoperative ventilation. NICE recommends that ASA III or IV patients with respiratory disease undergoing intermediate or major surgery are discussed with a senior anaesthetist for consideration of spirometry.

Arterial blood gases may be indicated. A PaO_2 of less than 7.1 kPa or less than 70% of normal for age, in combination with dyspnoea at rest, predicts dependence on postoperative respiratory support in patients undergoing upper abdominal surgery. PaO_2 may be used with a graph of iso-shunt lines to assess pulmonary shunting and to estimate oxygen requirements.

$PaCO_2$ is of little predictive value for postoperative ventilation but may be useful for detection of those with a hypoxic respiratory drive in whom postoperative administration of oxygen will need careful monitoring. Detection of metabolic acidosis in the critically ill patient may indicate demands on respiratory work which cannot be met by spontaneous ventilation.

PATIENTS WITH ELECTROLYTE ABNORMALITIES

Electrolyte disorders are often found on biochemical testing. They may reflect the consequences of an underlying disease process or drug effect. The cause should be sought as treatment of the cause often corrects the biochemical abnormality. Of particular importance is to assess the volume status of the patient as many biochemical abnormalities occur alongside significant hypo- or hyper-volaemic states.

Patient assessment should additionally seek to identify any adverse effects (Table 25.19) resulting

Table 25.19 Electrolyte abnormalities and implications for anaesthesia

Biochemical abnormality and associated clinical features	Example causes	Implications for anaesthesia
Hyponatraemia (<130 mmol/L) • Drowsiness, confusion, seizures	Diuretics, excess water intake, hyper-aldosteronism, e.g. heart failure, liver disease	Rapid changes in sodium concentration associated with IV fluid administration can result in central pontine myelinolysis
Hypernatraemia (>145 mmol/L) • Signs of dehydration, drowsiness, confusion	Diabetes insipidus, dehydration, Cushing syndrome	Rapid IV infusions can result in cerebral oedema
Hypokalaemia (<3.5 mmol/L) • Arrhythmias, muscle weakness, ileus	Reduced intake, diuretics, IV fluid administration without potassium supplementation	Risk of arrhythmias
Hyperkalaemia (>5.5 mmol/L) • Muscle weakness, myocardial depression, nausea	Renal failure, adrenal insufficiency, ACE inhibitor use	Suxamethonium contraindicated
Hypomagnesaemia (<0.8 mmol/L) • Arrhythmia's, confusion	Diuretic use, malabsorption syndromes, prolonged vomiting or diarrhoea	Prolonged neuromuscular blockade
Hypocalcaemia • Parasthesia, muscle cramps, prolonged QT	Hypoparathyroidism, hypomagnesaemia, vitamin D deficiency, renal failure	Increased risk of arrhythmias, e.g. Torsades de Pointes
Hypercalcaemia • Hyperparathyroidism, malignancy	Polyuria, polydipsia, neuropsychiatric disturbance	Commonly associated with hypovolaemia

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from the electrolyte disturbance, such as arrhythmias and alteration in conscious level. Particular patient groups with greater risk of electrolyte disturbance include:

- Elderly
- Patients with diabetes
- History of renal disease
- History of cardiovascular disease (often secondary to effect of medication, e.g. diuretics)
- Malnutrition
- History of gastrointestinal disease
- Alcohol misuse

DO NOT ATTEMPT RESUSCITATION (DNAR) ORDERS

Patients who have a DNAR in place presenting for anaesthesia can represent a difficult challenge. Reasons for patients with a DNAR to present for surgery may include fixation of fractured neck of femur, stoma formation for bowel obstruction or insertion of a PEG.

The difficulty arises from the likely cardiorespiratory instability associated with anaesthesia and that several resuscitative measures are commensurate with routine anaesthetic conduct, such as advanced airway management and the use of vasoactive drugs. In addition, there are important ethical considerations in particular respect to a patient's autonomy. There are considered to be three options with regard to managing a DNAR in the perioperative period.

1. Suspend the DNAR temporarily for the period of anaesthesia and recovery.
2. Modify the DNAR to permit use of certain drugs and techniques whilst the patient is under anaesthesia.
3. No changes made to the DNAR.

The assessment and planning of anaesthesia in this patient cohort is of paramount importance. Where a patient is competent they should be fully involved in discussions with senior anaesthetists and the lead surgeon with regard to the management of DNAR status in the perioperative period. Explicit agreement should be made by all parties prior to proceeding and documented in the patient notes. In the event of a patient lacking capacity to discuss the issue, then a decision should be made in the patient's best interests in conjunction with next-of-kin as per the Mental Capacity Act 2005.

Suspending a DNAR order is the most straightforward means to proceed. However, this may not be ethically appropriate with regard to respect for patient autonomy. If anaesthesia is to be conducted with a modified DNAR, then the interventions that would be provided or withheld must be agreed upon prior to proceeding. Examples may include defibrillation or DC cardioversion, not providing chest compressions or not providing postoperative ventilation in intensive care. Proceeding with anaesthesia with a full DNAR in place is controversial and can be considered incompatible with the provision of anaesthesia in many circumstances.

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PREOPERATIVE DRUG ADMINISTRATION

SANTOSH PATEL

One of the important goals of preoperative assessment is to understand the drugs that patients are taking for medical or other reasons. Many patients are prescribed a number of medications. Decisions to continue or withhold require understanding of pharmacological principles and implications for anaesthesia and surgery (Tables 25.20 through 25.26). Appropriate alternative therapy may be necessary. Preoperative knowledge of drug therapy also can be useful to guide medication management for the postoperative period. Key questions are

- What are the indications, pharmacokinetics and pharmacodynamics of drugs prescribed?
- What are the side effects?
- Are there any potential drug interactions?
- Are there any possible interactions with anaesthetic drugs?

- Are there any implications for anaesthesia techniques including regional anaesthesia?
- Will it be possible to administer oral drugs via enteral routes in the perioperative period?

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Table 25.20 Cardiovascular

Group	Advice	Risks	Comments
Beta blockers, e.g. Atenolol, Bisoprolol, Carvedilol, Propranolol, Sotalol	Continue and give dose on the day of surgery	Hypotension Bradycardia Abrupt withdrawal may increase CV morbidity and mortality	Remember drug interactions with other CV drugs
ACE inhibitors, e.g. Lisinopril, Perindopril, Ramipril, Enalapril	If for Hypertension omit on the day of surgery If for cardiac failure consider to continue on the day of surgery	Hypotension	Caution in the presence of dehydration and renal damage. Remember drug interactions with other drugs e.g. NSAIDs
Angiotensin receptor antagonists, e.g. Candesartan, Irbesartan, Losartan, Valsartan	If for hypertension omit on the day of surgery If for cardiac failure consider to continue on the day of surgery	Hypotension	Caution in the presence of dehydration and renal impairment Interactions with other drugs e.g. NSAIDs
Calcium channel blockers, e.g. Diltiazem, Verapamil, Amlodipine	Continue and give dose on the day of surgery	Verapamil – bradycardia, conduction blocks Diltiazem – dizziness, oedema Amlodipine – oedema	Caution if LVF or severe left ventricle dysfunction Drug interactions with anaesthetic and other CV drugs
Antianginals, e.g. Isosorbide mononitrate, Nicorandil	Continue and give dose on the day of surgery	Orthostatic hypotension with other peripheral vasodilators	
Diuretics, e.g. Bendroflumethiazide, Furosemide, Spironolactone	Continue if on for congestive heart failure.	Electrolyte disturbances. Spironolactone - hyperkalaemia with potassium supplements or potassium rich food	Avoid thiazide and Furosemide if hypovolemia or hypokalaemia.
Alpha blockers, e.g. Doxazosin, Prazosin, Phenoxybenzamine	Continue and give dose on the day	Hypotension	
Centrally acting, e.g. Clonidine, Methyldopa	Continue and give dose on the day	Hypotension	
Antiarrythmics, e.g. Amiodarone, Sotalol, Procainamide, Diltiazem, Verapamil	Continue and give dose on the day	Bradycardia. Amidarone has wide range of systemic toxicity	Ensure normal electrolytes and acid base balance
Digoxin	Continue and give dose on the day if no digoxin toxicity	Conduction abnormalities	Electrolyte abnormalities, hypoxia and acidosis and impaired renal function increase susceptibility for digoxin toxicity. Serum levels preoperatively if digoxin toxicity is suspected.

Note: Common indications include hypertension, IHD, cardiac failure and arrhythmias. Often patients use combinations of medications. This may make management during the perioperative period difficult.

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Table 25.21 Respiratory

	Advice	Risks	Comments
Bronchodilators • Selective beta agonists – inhalers/nebulizers, e.g. Salbutamol, Salmetarol • Theophyllines • Steroids (inhalers/nebulizers), e.g. Fluticasone, Budesonide, Beclomethasone	Continue and give dose on the day If not possible to use inhalers give nebulised dose	Selective beta agonsits – hypokalaemia May have oral candidiasis due to steroid inhalers	Theophyllines – check plasma levels to prevent toxic effects
Other: Leukotriene receptor antagonist, e.g. Montelukast	Continue and give dose on the day		
Antihistamines,e.g. Cetirizine, Loratidine	Continue and give dose on the day		

Table 25.22 Anticoagulants and antiplatelets

	Advice	Risks	Comments
Aspirin	Continue: 1. If taking for secondary cardiovascular prevention. These patients are considered at moderate-high risk for cardiovascular events 2. Surgeries associated with an increased risk for perioperative cardiovascular events, e.g. major vascular Discontinue: 1. If used for primary cardiovascular prevention. 2. Risk of bleeding is high, e.g. central nervous system, middle ear, posterior chamber of the eye, spine, intracranial, transurethral prostatectomy	Risk of cardiovascular event in high-risk patients or stent thrombosis if antiplatelet drugs discontinued	Careful assessment of thrombosis (leading to medical complications) versus risks of bleeding (leading to adverse surgical outcome and increased blood transfusion). Discussion with cardiologist, neurologist, GP and surgeon. Low dose aspirin need not be discontinued in regional anaesthesia
Warfarin	Discontinue 5 days prior to surgery. Patients at moderate and high risk of thromboembolism may require perioperative bridging with low molecular weight heparin (LMWH) Continue for cataract surgery, cystoscopy (without biopsy), minor dental procedure, minor dermatologic procedure, endoscopy (without biopsy)	Discontinuation of therapy increases risk of thromboembolism in patients with prior DVT or PE, risk of CVA in patients with atrial fibrillation, or risk of clotting of mechanical heart valves in patients with prior mechanical valve replacements	Underlying disease requiring anticoagulation, concomitant cardiovascular risk factors and type of surgery determines bridging therapy with LMWH (therapeutic dose or prophylactic or no need) For emergency surgery Vit K, FFP or prothrombin complex concentrate may be needed depending on target INR

(*Continued*)

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Table 25.22 (Continued) Anticoagulants and antiplatelets

	Advice	Risks	Comments
Heparin – Unfractionated	Hold dose 2 hours before surgery	Thrombocytopaenia Check platelet count if receiving >5 days	Check APTT Increased risk in the presence of other anticoagulants and antiplatelet agents.
Heparin low MW, e.g. Enoxaparin, Dalteparin	Hold prophylactic LMWH for at least 12 hours before anticipated neuraxial block Hold LMWH for 24 hours if therapeutic dose being used prior to neuraxial block		Increased risk in the presence of other anticoagulants and antiplatelet agents

P2Y12 receptor Blockers, e.g. Clopidogrel, Prasugrel, Ticagrelor, Ticlopidine	Clopidogrel:
	  Consideration should be given to temporary discontinuation of clopidogrel 7 days prior to invasive procedures if the risk of increased bleeding exceeds the risk of thrombosis
	  Ticagrelor
	  Discontinue 5–7 days prior to elective surgery Consult anaesthetist/cardiologist for advice
	Prasugrel:
	  Discontinue 7 days prior to elective surgery, if antiplatelet effect not desired
	  Consult anaesthetist/cardiology for advice
	Patients on clopidogrel with prior CVA:
	  Continue, particularly if surgery within 3 months of CVA or TIA. If particular surgery carries significant bleeding risk or associated morbidity, consider switching patient from clopidogrel to aspirin 7 days preop
	  In patients on clopidogrel undergoing peripheral artery and carotid procedures, continue, bleeding risk appears low
		If patient within 30 days of bare metallic stent or 3–12 months of drug eluting stent insertion, depending on the stent, non-urgent surgeries should be delayed until minimal dual antiplatelet therapy is completed.
		If the risk of major bleeding appears greater than the risk of stent thrombosis, aspirin should be continued and P2Y12 receptor inhibitor therapy should be discontinued as briefly as possible. There are no proven bridging therapies in this context.
			Ticagrelor:
			If urgent surgery, wait until minimum 1 month after coronary event/stenting, stop ticagrelor 7–10 days prior to surgery, but continue aspirin 75 mg
			Expert consultation is advisable, e.g. cardiologist, haematologist, stroke physician, vascular surgeon

(*Continued*)

Table 25.22 (Continued) Anticoagulants and antiplatelets

	Advice	Risks	Comments
Direct thrombin inhibitor, e.g. dabigatran	Discontinue prior to surgery Normal renal function or mild impairment (CrCl >50 mL/min) – 2 days before surgery (3 days if high risk of bleeding) moderate renal impairment (CrCl 30–50 mL/min) – 3 days before surgery (4–5 days before surgery if risk of bleeding is high)		No reversal agent available If APPT is normal pharmacological effects of dabigatran are low but cannot be ruled out
Factor Xa inhibitor: Direct, e.g. Rivaroxaban, Apixaban	Discontinue prior to surgery. No evidence-based recommendations. Rivaroxaban: CrCl >50 mL/min: Discontinue 24 hours before surgery (48 hours if high risk of bleeding) CrCl 30–50 mL/min: discontinue 48 hours before surgery (3–4 days if high risk of bleeding) Apixaban: discontinue 24 hours before surgery (48 hours before if high risk of bleeding).	Discontinuation of therapy increases risk of thromboembolism in patients with prior orthopaedic surgery, DVT or PE, risk of CVA in patients with atrial fibrillation	Seek advice from expert, e.g. haematologist or refer to anticoagulation clinic No reversal protocol available
Glycoprotein IIb/IIIa inhibitors, e.g. Tirofiban, Abciximab	Use of glycoprotein IIb/IIIa inhibitors must be discontinued preoperatively for greater than 12 hours to allow normal haemostasis They are contraindicated in major surgery.		Discuss with anaesthetist prior to restarting antiplatelet agent if patient has an epidural catheter in situ
Factor Xa inhibitor: indirect, e.g. Fondoparinuax	Due to 17 hour half-life, hold at least 36–48 hours prior to major surgery Hold for 72 hours prior to neuraxial block		Avoid use in spinal injury or surgery patients Extreme caution must be considered before performing neuroaxial block

Table 25.23 Antiepileptics

Phenytoin Carbamazepine Valproic acid Levetiracetam Lamotrigine	Continue medications during the perioperative period	If patient is being treated with a drug for which there is no intravenous form and delay in postoperative oral intake is anticipated, preoperative conversion to a drug for which an intravenous form is available may be considered

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Table 25.24 Endocrine

	Advise	Risks	Comments
Steroids, e.g. prednisone methylprednisolone hydrocortisone	Continue and give dose on the day stress corticosteroid Coverage: Low risk surgery: 0–25 mg hydrocortisone IV every 8 hours × 1–3 doses or oral equivalent Moderate risk surgery: 25–50 mg hydrocortisone IV every 8 hours × 1–3 doses or oral equivalent High risk surgery: 50–100 mg hydrocortisone IV every 8 hours × 1–3 doses or oral	Infection Remember chronic side effects including hypothalamic-pituitary-adrenal axis suppression if patient is on steroid for long term	Steroid equivalencies: Prednisone 5 mg = methylprednisolone 4 mg = hydrocortisone 20 mg = dexamethasone 0.75 mg
Thyroid Levothyroxine Antithyroid – propylthiouracil methimazole	Continue during perioperative period	Features of hypo- or hyperthytoidism	Check thyroid function tests if not done in last 6 months
Antidiabetics (non-insulin)	Below advice only if one meal is missed. * Continue on the day of surgery: Metformin, Pioglitazone * Drugs to omit: sulfonylureas (e.g. glibenclamide, gliclazide, glipizide). * Variable approach: Acarbose, meglitinides (give morning dose if patient is eating and surgery in afternoon). * Variable advice (some advice to continue and some to discontinue on the day of surgery) Dipeptidyl peptidase-IV inhibitor, e.g. sitagliptin Glucagon-like peptide-1 analogue (e.g. liraglutide) injection	Hypo- or hyperglycaemia	Some advice to discontinue all oral antidiabetics on the day of surgery Monitoring of blood glucose on admission and subsequently All oral antidiabetics are stopped if patients are on variable rate intravenous insulin infusions until patient starts eating and drinking normally

(*Continued*)

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Table 25.24 (Continued) Endocrine

	Advise	Risks	Comments
Antidiabetics: Insulin It is important to understand the rationale of prescribed insulin and regimen	Below advice if only one meal is missed. * If evening single long-acting insulin – reduce the dose by 20% on the *previous* day of surgery * If morning single long-acting insulin – on the day of surgery decrease usual dose by approximately one-third for each expected omitted meal * Twice daily regimen (mixed or intermediate acting) – 50% of usual morning dose * If on more than 2 daily injectable insulins – omit doses usually given with or before any meals advised to miss (depending on surgery schedule morning or afternoon)	Hypo- or hyperglycaemia	Monitoring of blood glucose on admission and subsequently Variable rate intravenous insulin infusion is indicated if more than one meal is missed, if there is poorly controlled diabetes, or emergency surgery
Antidiabetics: Insulin pump	There are no specific guidelines Seek expert advice and refer to local protocols	Hypoglycaemia	Monitor blood glucose

Table 25.25 Immunosuppresants

Methotrexate	Some physicians recommend stopping 48 hours to 1 week before surgery	Effect of methotrexate on wound healing is controversial
Colchicine, gold, sulfasalazine, azathioprine, cyclophosphamide	Discontinue the night before surgery	Immunosuppression and associated risks
Etanercept	Some suggest stopping 1–2 weeks before surgery	
Infliximab	Some suggest stopping 2–3 weeks before surgery	

Table 25.26 Herbal medications

	Advice	Risks	Comments
St. John's wort (used as an antidepressant)	Stop 5 days prior to surgery	Induces cytochrome P450. Inhibits serotonin reuptake and dopamine and noradrenaline reuptake Also has a high affinity for GABA receptor causing sedation	May enhance metabolism of anaesthetic and other drugs Serotonin syndrome may occur when given with other serotonergic drugs
Ginger (anti-inflammatory)	Discontinue 2 weeks before	Risk of bleeding due to thromboxane inhibition Hyperglycaemia	Also has antiemetic effect
Garlic (to lower cholesterol and to prevent atherosclerosis, antihypertensive)	Discontinue 1 week before	Risk of bleeding due to antiplatelet effect	Also has antitussive and diuretic actions
Omega 3 fish oil	Stop 2 weeks before	Bleeding due to antiplatelet effect	
Ginseng (to relieve stress, to elevate mood, type 2 diabetes)	Discontinue 1 week before	Risk of bleeding by effect on coagulation pathway and inhibition of platelet aggregation Hypoglycaemia	Drug interaction with other sympathomimetics Caution if patient is fasting
Ginkgo biloba (to improve cognitive function and memory) Alzheimer disease	Stop 36 hours before surgery	Risk of bleeding due to antiplatelet effect	
Ephedra (to stimulate CNS, asthma, weight loss)	Stop 24 hours before surgery	Ephedrine is the active ingredient causing sympathomimetic effects, vasoconstriction and sensitization of myocardium to catecholamines, arrhythmias	Haemodynamic instability with prolonged use due to depletion of catecholamine stores Drug interactions with MAO inhibitors
Echinacea (stimulates immunity)	Discontinue at least 2 weeks before No data available	May cause liver dysfunction so avoid hepatotoxic agents	Avoid if concomitant use of immunosuppresants (e.g. organ transplantation)
Turmeric (anti-infective, antioxidant, analgesic, anti-inflammatory)	No specific recommendation	Enzyme inhibition leading to prolonged action of many anaesthetic drugs	

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SUBSTANCE ABUSE AND PSYCHIATRIC DISORDERS

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SUBSTANCE ABUSE

Substance abuse is the self-administration of a substance that is not for normal medicinal purposes and which may lead to physical and/or psychological dependence. Physical dependence occurs when the presence of the substance is necessary for normal physiological wellbeing and when specific symptoms ('withdrawal') occur if it is not taken. Psychological dependence occurs when the substance produces a desire to repeat the experience again and again. Tolerance to a substance may develop such that increasing doses are required to produce the same effect. There is an association with hepatitis, AIDS, personality disorders, unwanted pregnancy and antisocial behaviour. Drug overdoses are common due to mistakes, the desire to try more, unexpected variation in strength and mixing with other drugs or substances. Table 25.27 summarises anaesthetic implications of common substances abused.

PSHYCHIATRIC DISORDERS

ANXIETY DISORDERS

Anxiety usually is associated with stresses; it may not require treatment. Some patients experience anxiety frequently without any obvious external stress. The mainstay of treatment is benzodiazepines although small doses of beta adrenergic blocking agents may be beneficial. There may be fear of anaesthesia and/or surgery and thoughtful preoperative counselling is valuable. The administration of a benzodiazepine premedication is useful.

Some patients also give history of panic attacks which can cause tachycardia, hypertension, tachypnoea, sweating, nausea and vomiting.

DEPRESSION

Depression may be endogenous or reactive although the distinction can be unclear. Reactive depression is usually triggered by external events, e.g. bereavement. Symptoms include fatigue, mood disturbances, insomnia, loss of appetite, decreased ability to concentrate and a general feeling of loss of worth. Suicidal thoughts may prevail. It is more common in women than men and familial tendencies exist. The exact pathophysiology is not known but disturbances in central amine levels may be present. It may be difficult to distinguish from dementia in the elderly. Treatment is pharmacological (Table 25.28) and/or ECT.

MANIC DISORDERS

The clinical presentation is one of hyperactivity and heightened mood. It may progress to hallucinations and delusions. It appears to be inherited in an autosomal dominant manner and pathophysiological changes include abnormalities in central neurotransmitter regulation. The routine treatment is lithium.

SCHIZOPHRENIA

These patients may exhibit a wide variety of symptoms including hallucinations, withdrawal from society, flat affect and disinterest in personal appearance. A wide variety of drugs (Table 25.28) is used in its treatment.

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Intraoperative arrhythmias, Chapter 30

SURGICAL POPULATIONS

SANTOSH PATEL

THE ELDERLY PATIENT

Age is not a contraindication for surgery. Two separate issues exist: firstly, the presence of age-related pathology, and secondly, the physiological effect of ageing in the apparently well person.

Presence and severity of medical illnesses are more predictive of perioperative outcome than age itself. One-third of elderly patients have three or more medical problems necessitating multiple drug therapy.

The influence of cultural factors must not be underestimated. Deafness and pre-existing dementia and confusion may make communication and consent difficult. Assistance from a family member, next of kin or support worker may be helpful.

IMPORTANT PHYSIOLOGICAL AND PHARMACOLOGICAL IMPLICATIONS

Cardiovascular system

Loss of large artery elasticity, decreased resting heart rate, underdamped autonomic circulatory control, impaired diastolic function, sclerosed and calcified valves.

Cardiac symptoms may not be evident because of age-related decrease in exercise. Functional limitations may be due to arthritis or cognitive problems. As myocardial ischaemia and infarction are common postoperative complications, preoperative assessment for coronary artery disease is important.

Respiratory system

Increased residual volume and closing capacity, ventilation-perfusion mismatch, reduced vital capacity, progressive age-related decline of arterial oxygen tension, less chest compliance. Protective mechanisms (cough, mucociliary clearance, ventilator response to hypoxia) are blunted.

Central nervous system

Neuronal death and dysfunction are progressive and manifest by reduced conduction velocity and deafferentation. Transmitter deficiencies may result in specific neurological syndromes (e.g. Parkinson disease, dementia). These may be symptomless until an advanced degree of neurological deterioration has occurred. Thermoregulatory responses are blunted. MAC for inhalational anaesthetics is reduced. Loss of neuronal mass may produce sensitivity to CNS depressant drugs.

Renal

Reduced renal blood flow and GFR, impaired fluid and electrolyte handling because of reduced tubular function.

Gastrointestinal

Liver mass is reduced and function impaired. Action of drugs dependant on liver for metabolism and clearance may be prolonged.

Metabolism and endocrine

Muscle mass is reduced. Basal metabolism is reduced. Glucose handling is impaired due to increased insulin resistance.

Pharmacology

Increased adipose tissue mass increases the reservoir of lipid-soluble agents leading to increased duration of many drugs. Decrease in total body water decreases the volume of distribution for water-soluble drugs. Higher peak plasma levels may produce exaggerated effects. Liver and renal mass are reduced affecting metabolism and excretion, respectively. Blood flow to these organs is reduced which may further affect handling of drugs. Hypoalbuminaemia is common, leading to reduced protein binding and higher free level of drugs. This increases the risks of drug toxicity.

KEY PRINCIPLES

- Geriatrician led multidisciplinary team involvement during preoperative assessment

Table 25.27 Systemic changes and anaesthetic considerations of specifc substance abuse

Substance abused	Systemic effects	Anaesthetic implications
Alcohol Varying degrees of severity	Many of the central effects of alcohol appear to result from an action on the GABA system. Alcohol increases GABA-mediated increase in chloride conductance and is closely related to the action of benzodiazepines and barbiturates. Withdrawal of alcohol produces tremor, hallucinations, agitation, confusion, delirium tremens, insomnia, tachycardia, hypertension and convulsions. Chronic effects – liver disease, pancreatitis, malnutrition, cardiomyopathy. Longer-term chronic effects include cerebellar neurone loss associated with vitamin B1 deficiency (Wernicke's encephalopathy or Korsakoff's psychosis)	In acute alcohol intoxication delay anaesthesia if possible. Aspiration prophylaxis. Hypoglycaemia may develop during the hangover phase following the acute period; blood glucose should be measured at regular intervals. The sedative and respiratory depressant effects of opioid and inhalational anaesthetic agents may be potentiated. In the chronic alcoholic tolerance to anaesthetic agents is often present. In the later stages impaired hepatic function may lead to slower drug metabolism and reduced plasma proteins causing an exaggerated response to some agents. Hepatic cirrhosis and nutritional deficiencies may also be present. Regional techniques may prove attractive but care should be taken if any neurological deficits are present. Withdrawal symptoms may need treatment with alpha-2 agonists, benzodiazepines or alcohol.
Opioids – Several routes. May be prescribed drugs, e.g. morphine, meperidine or codeine. Illicit drugs, e.g. heroin (may be mixed with other illicit drugs). Deaddiction drug – methadone	Dependence, tolerance. Overdose – slow respirations, pinpoint pupils, low GCS, slurred speech. Inhaled or injected heroine – pulmonary infections, viral infections, glomerulonephritis, nephropathy. Withdrawal symptoms – sympathetic stimulation.	History may not be reliable Poor nutritional status; blood borne infections may be present; high risk of sepsis. Administer their routine dose. Patient may be on detoxification programme. Continue if on methadone maintenance programme; marked haemodynamic instability can occur, acute tolerance decreases MAC for inhalational agents, cross-tolerance to other sedatives. Management of withdrawal symptoms – clonidine can be used. Non-opioid-based anaesthesia is possible. If possible use regional technique but anticipate exaggerated hypotension. It may be necessary to calculate morphine-equivalent dose for other opioids.

(Continued)

Table 25.27 (Continued) Systemic changes and anaesthetic considerations of specific substance abuse

Substance abused	Systemic effects	Anaesthetic implications
Cocaine	Stimulatory effects mediated through enhancement of adrenergic and dopaminergic pathways. Withdrawal causes fatigue, depression and increased appetite. Acute administration can cause tachycardia, arrhythmias (including VF), hypertension, coronary spasm and myocardial ischaemia or infarction. Smoking it may also cause lung damage, pulmonary oedema and atrophy of the nasal septum. Anxiety, psychosis or mydriasis can occur. Agitation, paranoid thoughts, hyperreflexia, hyperpyrexia and convulsions may also be seen.	For agitation and anxiety, benzodiazepines may be necessary. Requirements for anaesthetic agents may be increased. Hypertension should be managed by nitrate or hydralazine. Calcium channel blocker may be useful. Clonidine or dexmedetomidine may control blood pressure as well as agitation. Beta blockers may have unopposed alpha effects and cardiovascular consequences. Avoid volatile agents which can sensitise the myocardium to catecholamines. Thrombocytopaenia has been described – check platelet count before considering a regional block.
Ecstasy (many street names)	Euphoria, memory loss, cognitive and behavioural disorders.	Correction of hyponatraemia.
3,4-Methylenedioxy-methamphetamine (MDMA)	Serotonin syndrome (neuromuscular excitability, autonomic disturbances, mental disturbances) can occur. Sympathetic stimulation. Hyperthermia can occur. Rhabdomyolyisis can lead to renal and cardiac failure, profound sweating leading to increased water intake, hyponatremia and seizures. Coagulopathy. Fatty liver changes.	Temperature control – cooling, if excessive muscle damage dantrolene may be useful. Avoid suxamethonium. Pharmacokinetics of drugs may be altered because of impaired liver and kidney function. Profound hypotension on induction may be seen which may not respond to ephedrine. Metaraminol is preferable.

(Continued)

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Table 25.27 (Continued) Systemic changes and anaesthetic considerations of specifc substance abuse

Substance abused	Systemic effects	Anaesthetic implications
Marijuna (Cannaboid) Inhalational, oral routes. May be mixed with other recreational agents.	Euphoria. Hallucinations. Agitation. Cannabis psychosis rarely. May cause drowsiness, tachycardia and postural hypotension. Acute toxicity rare but can cause anxiety, tachycardia, conjunctival congestion. Long-term use may cause tar deposits in the lungs. Effects can be complex, unpredictable and widespread.	Airway irritability and bronchospasm may occur. If tachycardia is present avoid other drugs which cause tachycardia. Delayed recovery and respiratory depression are possible. There is otherwise little effect on anaesthesia.
Other psychoactive drugs: Lysergic acid diethylamide (LSD), phencyclidine (PCP) Ketamine Mescaline Oral route Often mixed with other drugs and alcohol.	LSD – acts on serotonin receptors. PCP – interferes with catecholamine uptake. Anxiety, agitation, panic attacks. Hallucinations (visual, tactile). Tachycardia, hypertension, hyperthermia, dilated pupils due to sympathetic stimulation. Seizures, coma and death may occur due to overdose. Psychological addiction occurs.	Marked sympathetic stimulation should be controlled to prevent myocardial infarction, cardiac failure and cerebral side effects. Antiarrythmic drugs may be required. If regional technique is used, sedation may be necessary to control anxiety and agitation.
Benzodiazepines	Chronic benzodiazepine ingestion produces physical dependence. Tolerance also develops. Depression of respiration may occur with overdose but is much more likely if another substance (e.g. alcohol) has been taken in addition.	Beware of using flumazenil in acute benzodiazepine overdose as convulsions may occur. Withdrawal symptoms may appear during perioperative period. Cross-tolerance to anaesthetic agents occur leading to the need for higher doses.

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Table 25.28 Psychiatric-related drugs

Drug/drug group	Administration during perioperative period	Specific anaesthetic implications	Side effects
Tricyclics, e.g. Amitriptyline, Nortriptyline, Imipramine	Treatment need not be interrupted during the operative period.	Anesthetic requirement may be increased. Avoid sympathomimetic agents. Orthostatic hypotension. A directly acting agent such as metaraminol may be preferable to manage hypotension.	Mild sedation, dry mouth, blurred vision, urinary retention (anticholinergic effects), ECG changes (increased PR and QT intervals). Overdose may cause potentially fatal cardiac arrhythmias. Increased risk of serotonin syndrome in patients who receive methylene blue intraoperatively.
Others, e.g. Bupropion, Venlafaxine			Agitation, lack of sleep, diarrhoea. Increased risk of serotonin syndrome in patients who receive methylene blue intraoperatively. Combination should be avoided unless benefit outweighs risk.
Selective serotonin reuptake inhibitors (SSRIs) (including agents with partial SSRI activity), e.g. Fluoxetine, Paroxetine	Treatment need not be interrupted during the operative period.	Fluoxetine – enzyme inhibition so potentiation of effects of drugs dependant on liver metabolism There have been reports of serotonin syndrome after concurrent use with tramadol.	Serious drug interactions with other antidepressants and antipsychotic drugs, e.g. serotonin syndrome.
Monoamine oxidase inhibitors (MAOIs), e.g. selegiline, phenelzine	Continue during perioperative period. It is no longer recommended that MAOIs should be stopped 21 days before a general anaesthetic.	Avoid opioids. Avoid meperidine and indirect sympathomimetics (i.e. ephedrine); may cause neuroleptic malignant syndrome. Avoid or minimize sympathetic stimulation. Exaggerated response to sympathomimetic drugs (e.g. topical or subcutaneous vasoconstrictors). Avoid inhalational anaesthetic agents which sensitize myocardium to catecholamines. Exaggerated hypotension with central neuraxial block.	Increased risk of serotonin syndrome in patients who receive methylene blue intraoperatively. Combination should be avoided unless benefit outweighs risk.

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Table 25.28 (Continued) Psychiatric-related drugs

Drug/drug group	Administration during perioperative period	Specific anaesthetic implications	Side effects
Atypical antipsychotics, e.g. olanzapine, risperidone, quatiepine, zotepine	Continue preoperatively.	Postural hypotension may occur and there may be an exaggerated response to hypotensive drugs (including anaesthetic agents), fluid loss and IPPV.	These drugs possess a wide variety of side effects including extrapyramidal symptoms and signs, anticholinergic effects, sedation, dyskinesias and Parkinsonism.
Typical antipsychotics, e.g. haloperidol, thoridazine, chorpromazine	Continue preoperatively.	Postural hypotension; tachycardia may be present.	Acute dystonia, tardive dyskinesia. Anticholinergic effects – dry mouth, urinary retention, blurred vision. Alpha1 receptor blockade. Antihistaminic effects – sedation.
Lithium	Continue unless risks outweigh benefits. Minor surgery – not stopped. Major surgery – discuss with psychiatrist. If discontinuation is required, stop 24–72 hours before surgery.	Very narrow therapeutic index. Measure plasma level preoperatively. Toxicity appears when the level is over 1.5 mMol L^{-1}. If levels rise above 2 mMol L^{-1} it can be fatal. May potentiate the effect of neuromuscular blocking agents.	Signs of lithium toxicity include muscle weakness, sedation, ataxia, hypotension and ECG changes. Hypothyroidism, polyuria and polydipsia may occur with long-term treatment.

is useful to identify and optimize high-risk patients.

- Preoperative communication may be difficult because of hearing, vision or speech problems. Mental and decision making capacity may be impaired. Information from relatives or support workers may identify specific issues with regards to communication and capacity.

- Preoperative assessment must consider physiological, medical, psychological, functional and social aspects. Elderly patients of the same age may present with variable issues.

- High risk of cardiopulmonary, renal and CNS complications. Recognition and modification of risk factors are important to improve perioperative outcome.

- Preoperative factors for postoperative delirium like electrolyte and blood sugar imbalance, hypoxia, hypovolaemia, malnutrition, and infection should be corrected. Delirium increases postoperative morbidity and hospital stay significantly.

- Functional assessment is assisted by inquiring about activities of daily living. If more than six activities are impaired, hospital mortality and nursing home stay is increased significantly.

- Preoperative frailty is associated with postoperative complications and delayed discharge. Assessment of frailty may allow risk prediction and in some cases improvement. Several physical characteristics may suggest its presence such as weakness (e.g. grip strength), unintentional weight loss (>10 pounds in last year), speed (time to walk 15 feet), and level of activity (e.g. Minnesota leisure time activity questionnaire). Improvement in muscle strength and nutrition may reduce frailty.

SPECIFIC CLINICAL PROBLEMS

Dementia

Alzheimer disease is the most common form of dementia. Its pathology is primarily a degeneration of the processing functions of the brain, rather than its somatosensory function. Pain threshold and pain perception are unimpaired, but the ability to rationalize about a painful stimulus may be affected. These patients may suffer acutely with painful movements (e.g. coughing after abdominal surgery) and be unable either to take measures to lessen the pain or understand what is happening. This could lead to a state of continuous arousal of the CNS pain pathways.

Dementia must be distinguished from other causes of apparent cognitive dysfunction in the elderly patient. The presence of corroborating evidence from relatives and carers is important. The differential diagnosis includes acute confusional states (pain, infection, hospitalization and alcohol or caffeine withdrawal), depression and deafness. Acute confusional states may improve with successful management of the underlying condition. Dementia presents in varying degrees and the patient who is mildly demented may live safely in the community. The added stress of hospitalization, together with perioperative cerebrovascular complications or unsuitable drug use (e.g. anticholinergics) may make it difficult for full rehabilitation to be achieved. Dementia is a predictor of perioperative morbidity and mortality.

ANAESTHESIA FOR HIP FRACTURE SURGERY

Hip fracture surgery demonstrates many of the problems with anaesthesia for the elderly. It is important to ascertain the circumstances behind the injury. A degree of delirium is not unexpected as a consequence of pain or hospitalization – in one series up to 44% of such patients were so described.

Systematic reviews comparing regional with general anaesthesia have demonstrated that regional anaesthesia is associated with improved short-term survival and reduced thromboembolic complications.

THE PAEDIATRIC PATIENT

Anatomical, physiological and pharmacological considerations

- *Airway* – Large head and tongue, anterior and high larynx, tonsils and adenoids hypertrophy, short trachea, narrowest part cricoid ring.

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- *Respiratory* – High rate, low FRC, lower lung and higher chest compliance.
- *CVS* – Rate dependant cardiac output.
- *Fluid balance* – Higher total body water content; sensitive to fluid loss.
- *Metabolic* – Higher metabolic rate.
- *Thermoregulation* – Heat loss is higher because of low fat content and higher surface-to-body weight ratio. Thermoregulatory mechanisms are not mature. In neonates, non-shivering thermogenesis is mainly responsible for heat production from brown fat. Highly susceptible to hypothermia.
- *Pharmacological* – Immature liver so drug metabolism impaired. Total body water increased so volume of distribution of drugs altered. Minimal alveolar concentration for inhalational anaesthetic agent is high.

Fasting

- Clear liquids – 2 hours; breast milk – 4 hours; other milk and food – 6 hours.

Prematurity

Infants born preterm have lung immaturity, pulmonary dysplasia and pulmonary hypertension. Prolonged tracheal intubation and ventilation may result in subglottic stenosis. These infants are at risk of apnoea and bradycardia.

Upper respiratory tract infections

The causative organism may be viral or bacterial. Symptoms include nasal congestion, nasal discharge, sneezing, cough, sore throat and fever. There may be airway hyperactivity, decreased mucociliary clearance and decreased pulmonary reserve. Increased incidence of laryngospasm, bronchospasm, reduced oxygen saturations and airway obstruction has been reported.

There is an increased risk of respiratory complications with a history of asthma, prematurity, one parent smoking, presence of airway anomaly and a history of snoring. Lack of experienced anaesthetist, airway surgery and surgery requiring tracheal intubation also increase respiratory complications.

Chest X-ray may be required in severe cases. If there are compelling risks for development of respiratory complications, surgery should be postponed.

Heart murmur

Infants with a heart murmur should be referred to a paediatric cardiologist. Often CHD is associated with other congenital diseases. A history of breathlessness, failure to grow, syncopal attack and squatting suggest an underlying serious problem. Examination for any CHD associated syndromes, cyanosis, clubbing and cardiac failure should be carried out. ECG, ECHO and referral to specialist centre may be necessary.

Vaccination

Recent vaccinations may increase the risk of perioperative complications. The immunosuppressive effects of anaesthesia and surgery may make a vaccine less effective.

EMERGENCY SURGERY

The issue is that there is not enough time available for the patient to be fully prepared and optimised. There may be existing homeostatic disturbances and not all test results may be available.

If the nature of the surgery is compelling and immediate, no time should be wasted. If it is possible to introduce some delay, however short, this is beneficial in order to allow the patient fuller assessment and optimisation.

Assessment should be the same as for elective procedures if time permits. Assess as fully as possible within the constraints of the degree of urgency. If the patient cannot give any history, look for warning cards and 'Medic-Alert' tags or bracelets. Look at previous hospital notes and emergency department records. Ask friends or relatives if present. Perform appropriate investigations depending upon available time. Obtain the results for those already instituted. Look for the presence of alcohol or drug intoxication, particularly in cases of trauma. If there is insufficient time to obtain the results of blood tests, they should still be requested and

then action taken if necessary when the results do become available.

Review previous anaesthetic charts and information. Preoperative assessment may need to be carried out in uncommon places, e.g. A&E. An ABCDE approach may be useful.

Airway and breathing

Assess airway. If intubated, check the size, type and patency of tracheal tube. If receiving IPPV, check the mode and parameters. Look for respiratory problems, e.g. chest infection, secretions or bleeding. Check arterial blood gases.

Circulation

There may be normal haemodynamic parameters due to compensation. Anything from negligible haemodynamic insufficiency to severe shock is possible. If shocked, is it hypovolaemic, septic or cardiogenic?

Evaluate fluid balance and look for signs and severity of hypovolaemia and bleeding. Remember that loss may be concealed. Insert at least one large bore cannula and resuscitate with fluids and blood as needed. Most patients for major surgery will need invasive lines for monitoring and vasopressors/inotropes infusion.

Drugs

Note all routine medications and any new drugs started and their timing for administration. If patients are receiving any inotropes or other drug infusions, check the rate and volume remaining in the syringe or bag. Establish any drug allergy.

Equipment

Prepare for transport if needed. Oxygen supply, ventilator, backup manual ventilation, monitoring, and infusion pumps should all be available. Critically ill patients require intensive care throughout.

Examination

Perform rapid cardiovascular and neurological examination. Note vital parameters. Examine available medical record, e.g. early warning scores or ICU observation charts.

Fasting

Assume that the patient has a full stomach. Even if the last food intake was over 6 h ago, the stomach may not be empty. In cases of trauma, the stomach ceases to empty at the time of injury. Attempts at emptying the stomach using a nasogastric tube are not usually successful and should not be relied upon.

Group and save

In case of massive haemorrhage or DIC, ensure blood and blood components are available. If necessary, alert blood bank and initiate massive haemorrhage protocol.

Help

Request help – at the right time of the right colleague. Communicate clearly what is required. Help may be required from another speciality.

Investigations

Check recent blood results and arterial blood gas analysis. Check any radiological investigations available which may give additional information not revealed by systematic examination.

Invasive lines

Note and take care of all invasive lines and tubes in situ.

EMERGENCY LAPAROTOMY

Morbidity (25%–40%) and mortality (10%–15%) is high.

Key principles:

- Identify and optimize organ function and systemic problems.
- Timing of surgery is important. Immediate surgery if patient is bleeding; with septic shock <3 hours; with severe sepsis and evidence of organ dysfunction <6 hours.
- APACHE II and P-POSSUM scoring systems to assess risk.
- Senior anaesthetist and surgeon.
- Resources such as radiology, theatre and postoperative care facilities must be available.

- Development of emergency laparotomy pathways based on national standards and available local quality data improve the care of patients and prevent complications.

CANCER SURGERY

Cancer patients are often at the extremes of age and primary systemic comorbidities may be present. Thorough systematic evaluation and detailed investigations and preparation of cancer patients are necessary for safer outcome.

- Anaemia, hypoproteinaemia, weight loss and nutritional deficiencies are common.
- Cancer mass can cause anatomical changes (e.g. compression or invasion of airway or vascular structures).
- CT scan reports may give information about local or distant spread.
- Metastasis can occur to lung, liver, bone and brain (Table 25.29).
- Preoperative nausea and vomiting due to chemotherapy and radiotherapy.
- Increased risk of deep vein thrombosis.
- Increased risk of infection.
- Cancer pain may be present due to nerves, nerve roots or major plexus involvement, bone metastasis or ischaemia due to vascular compromise. Radiotherapy may cause tissue necrosis, bone necrosis and neuropathy. Patients may be on several analgesics including opioids.
- Some cancers (e.g. lung, breast and ovary) cause paraneoplastic syndromes due to release of hormones or cytokines. These include hematologic and coagulation abnormalities, fatigue, electrolyte abnormalities (e.g. hypercalcaemia due to osteolysis, hyponatraemia due to SIADH) and adrenal insufficiency.
- Chemotherapy can cause systemic side effects and functional limitations (Table 25.30). Chemotherapy depends on site and type of cancer. During preoperative assessment, side effects (Table 25.31) of chemotherapy should be considered and investigated initially by blood results.
- Radiation site and dose may produce adverse effects including radiation

Table 25.29 Metastatic sites of cancer

Primary cancer	Site of metastasis
Colorectal	Liver, lung, peritoneum, brain
Ovary	Liver, lung, peritoneum
Pancreas	Liver, lung, peritoneum
Kidney	Adrenal gland, bone brain, liver, lung
Lung	Adrenal gland, bone brain, liver, lung, pleura
Prostate	Adrenal gland, bone brain, liver
Breast	Bone, brain, liver, lung
Bladder	Bone, liver, lung
Uterus	Bone, liver, lung, vagina, peritoneum

Table 25.30 Chemotherapeutic agents for common cancer

Cancer	Potential chemotherapeutic agents
Colon and rectum	Irinotecan, Fluorouracil, Cisplatin, Folinic acid, methotrexate, Cetuximab
Bladder	Doxorubicin, cisplatin, cyclophosphamide, Fluorouracil, methotrexate
Prostate	Doxorubicin, Docetaxel, Prednisone, cisplatin, cyclophosphamide, Leuprolide, Vinblastine
Uterus	Paclitaxel, Carboplatin, Tamoxifen, Fluorouracil, Doxorubicin, cisplatin
Ovary	Paclitaxel, Capecitabine, Doxorubicin, Irinotecan, cyclophosphamide, cisplatin, methotrexate
Breast	Tamoxifen, Paclitaxel, Doxorubicin, cyclophosphamide, cisplatin, Capecitabine, methotrexate, etoposide
Pancreas	Fluorouracil, gemicitabine, Carmustine, mitomycin, doxorubicin, cisplatin
Lung and bronchus	Several drugs and regimens
Leukaemia	Several drugs and regimens
Lymphoma	Several drugs and regimens

Table 25.31 Toxicity of chemotherapeutic agents

Toxicity/side effects	Chemotherapeutic agents
Anaemia	Methotrexate, 6-Merceptopurine, 5-Fluorouracil, Busulfan, Chlorambucil, Doxorubicin, Mithramycin, Carmustine
Bone marrow suppression	Methotrexate, 6-Merceptopurine, 5-Fluorouracil, Busulfan, Chlorambucil, Doxorubicin, Mithramycin, Carmustine
Immunosupression	
Cardiac	Doxorubicin, Daunorubicin, Cisplatin
Hepatic	6-Merceptopurine, Thiogaunine, Methotrexate, Mitharamycin, Azathioprine, L-Asparginase
Pulmonary	Bleomycin, methotrexate, cyclophosphamide, chlorambucil, busulfan
Renal	Methotrexate, 6-Merceptopurine, cyclophosphamide, Vincristine, Busulfan, Carmustine, L-Asparginase
CNS	Methotrexate, vincristine, cisplatin

enteritis, pneumonitis, neuropathy, cystitis, pericarditis and hepatic injury. Radiation also causes direct damage to skin and soft tissues with tissue necrosis and oedema leading to fibrosis and contracture. In some critical areas such as head and neck it can cause difficulty in intubation and insertion of invasive lines.

- Cancer patients may have psychosocial issues. An empathetic discussion during preoperative visit is essential.

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Blood transfusion, Chapter 30
Fluid and electrolyte balance, Chapter 30
Transport of the critically ill, Chapter 30

Airway

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AIRWAY AND ASPIRATION RISK

NARCIS UNGUREANU

Inhalation of gastric contents into the tracheobronchial tree produces chemical pneumonia due to the acidic pH of the gastric contents. In the general population, the incidence of aspiration is low; however, during unexpected difficult intubation the risk is higher. Positive pressure ventilation through face mask and supraglottic devices using a high peak pressure may lead to distension of the stomach and regurgitation of stomach contents.

Aspiration after tracheal intubation in elective patients has an incidence of 1.25 per 10,000 patients. Difficult intubation during elective and rapid sequence induction increases the risk. The fourth national audit project in the UK identified risk factors for aspiration in 35% of all patients and in 50% of patients with morbid obesity. It is important to identify patients with increased risk of aspiration. The sign-in component of a WHO checklist has a specific question on aspiration risk prompting the team to choose an appropriate strategy to prevent aspiration.

RISK FACTORS FOR ASPIRATION

- Full stomach
- Raised intra-abdominal pressure
- Gastro-intestinal obstruction
- Delayed gastric emptying due to drugs (opioids), pain and autonomic neuropathy
- Gastro-oesophageal reflux
- Hiatus hernia
- Previous oesophageal surgery
- Pregnancy
- Difficult intubation
- Straining or coughing during light plane of anaesthesia
- Acute alcohol intoxication

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PREVENTION OF ASPIRATION

- Avoid general anaesthesia if possible.
- Delay non-emergency surgery (for 6 hours) to empty the stomach.
- Prophylactic drugs.
- Rapid sequence induction of general anaesthesia.
- Induction of anaesthesia in head up position or left lateral head down position.

The 'classical' rapid sequence induction (RSI) is designed to achieve rapid anaesthesia and intubation without interposed mechanical ventilation to minimize the risks of gastric aspiration. This technique includes:

- Pre-oxygenation of the patient with 100% oxygen through a tight-fitting face mask, with eight vital capacity breaths or for 3–5 minutes of tidal volume breathing to denitrogenate the lungs.
- Protection of tracheobronchial tree by cricoid pressure (Sellick's manoeuvre).
- Rapid relaxation after administration of predetermined dose of intravenous induction agent. The neuromuscular blocking agent should only be administered after ensuring loss of consciousness to prevent awareness.
- Placement of the tracheal tube under vision followed by auscultation, $ETCO_2$ measurement and release of cricoid pressure only after confirmation of correct placement of tracheal tube.

A modified version of the RSI technique has been increasingly used in recent years. Propofol has replaced thiopentone and the majority use an opioid at induction. Suxamethonium was the muscle relaxant most commonly used but rocuronium is now often used due to its rapid onset ability to be quickly reversed using sugammadex. Gentle mask ventilation is recommended to prevent hypoxaemia during the apnoeic period.

CRICOID PRESSURE

A properly trained assistant is mandatory. A bimanual technique is preferred by some – the neck support prevents the head from flexing on the neck. Others prefer the single-handed technique leaving the assistant's other hand free to help with intubation. In the obese or patients with difficult neck anatomy, an ultrasound can be used to locate the cricoid cartilage prior to commencing anaesthesia.

Cricoid pressure should be applied to the awake patient with a force of 10 N (1 kg), after preoxygenation but before intravenous induction and increased to 30 N (3 kg) after loss of consciousness. The assistant should practice the correct application of force on a weighing scale.

COMPLICATIONS OF CRICOID PRESSURE

In an awake patient, cricoid pressure can cause discomfort and anxiety. It can distort the anatomy, worsen the laryngoscopic view, cause airway obstruction and restrict facemask ventilation. Airway obstruction is directly related to the force applied – 40 N causes airway obstruction in about 35% of patients. When a difficulty laryngoscopy is encountered, cricoid pressure may need to be released to facilitate laryngoscopy or insertion of a supraglottic airway device.

PROPHYLACTIC DRUGS

- H_2 receptor antagonist (ranitidine 150 mg PO or 50 mg IV given slowly): administered 30 minutes prior to induction of anaesthesia.
- Metoclopramide (10 mg PO or IV): administered 30–60 minutes prior to induction of anaesthesia to stimulate gastric emptying.
- Nonparticulate antacid (sodium citrate 30 mL): administered immediately prior to induction of anaesthesia.

MANAGEMENT OF SUSPECTED ASPIRATION

- Administer 100% O_2.
- Head down tilt.
- Oropharyngeal suction to clear the airway.
- Secure the airway with a tracheal tube.
- Immediate tracheal suction.
- Insert a nasogastric tube and empty the stomach.

- Mechanical ventilation and PEEP to treat hypoxaemia.
- Bronchodilators if needed.
- Chest X-ray to diagnose collapse or pneumonia.
- Supportive care includes fluid management and H_2 receptor antagonists for prophylaxis against stress ulcers.

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CROSS-REFERENCES

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Emergency anaesthesia, Chapter 25

ARTIFICIAL AIRWAYS

CYPRIAN MENDONCA

Recent advances in technology have enabled the development of various airway devices to ensure safe airway management. The most commonly used airway devices are shown in Figure 26.1.

FACE-MASK

Designed to fit the face, along with a self-inflating bag or reservoir bag, it facilitates ventilation when tightly applied over the face. The body of the mask has an air-filled cushion to ensure a tight fit on the face. The proximal end of the mask has a 22 mm inlet connection to the angle piece. Transparent single use masks are available in different sizes to fit neonates to large adults.

ORAL/NASAL AIRWAYS

An airway will often relieve an obstruction, but if the patient is too 'light', it may provoke coughing, breath holding and laryngospasm. A nasal airway should be used in patients with fragile teeth, crowns or bridges. Avoid nasal airways in patients with bleeding disorders and suspected base of skull fractures.

SUPRAGLOTTIC AIRWAY DEVICES

Supraglottic airway devices are used in approximately 56% of all general anaesthetics administered in the UK. The currently available ones are extensively used and have good safety profiles. Supraglottic airway devices are classified as first generation or second generation. The second generation devices offer improved safety against gastric aspiration and provide a higher oropharyngeal leak pressure. The two distinctive features that these devices most commonly have are the gastric drainage port and the integrated bite block. The examples of second generation SADs include I-Gel, ProSeal LMA (PLMA), LMA Supreme, Laryngeal tube suction II (LTS-II) and Streamlined liner of the pharynx airway (SLIPA).

FIRST GENERATION SUPRAGLOTTIC AIRWAY DEVICES

Laryngeal mask airway and laryngeal mask

The laryngeal mask airway (LMA), also known as classic LMA (cLMA), is a laryngeal mask with a protected name produced by the original manufacturers and made from latex-free medical grade silicone rubber. Similar devices on the market which are manufactured by other companies are called laryngeal masks (LM), are single use and made from polyvinyl-chloride (PVC). The flexible LM consists of an airway tube made of soft silicone with wire reinforcement. This avoids kinking of the airway tube when bent.
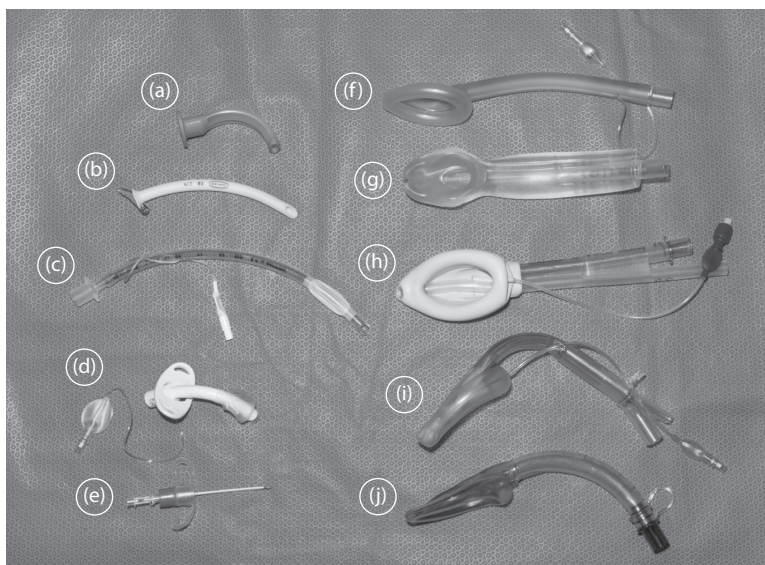
Figure 26.1 Artificial airways. (a) Oropharyngeal airway. (b) Nasal airway. (c) Tracheal tube. (d) Tracheostomy tube. (e) Ravussin style jet ventilation catheter. (f) Laryngeal mask. (g) I-Gel. (h) ProSeal LMA. (i) LMA supreme. (j) Air-Qsp.

The above three devices are all considered *first generation* supraglottic airway devices that consist of an airway tube, a mask and a mask inflation line. When correctly placed, the tip rests against the upper oesophageal sphincter and the airway channel opens to the glottis. A seal around the larynx is achieved by inflating the cuff. For easy insertion of an LM, the patient must be deeply anaesthetized. Propofol provides the best conditions for insertion. Positive pressure ventilation can be performed. A bite block should be used and the device should be left in until the patient is awake. The LM is a very useful aid to fibre-optic guided intubation. It is available from sizes 1 to 6 to fit neonates to large adults. The cLMA has bars in the mask aperture, which prevents the obstruction of the airway channel by the epiglottis.

SECOND-GENERATION SADS

I-Gel

The I-Gel airway is similar to the LM but does not have an inflatable cuff. It is made of medical grade thermoplastic elastomer called styrene-ethylene-butadiene-styrene (SEBS), which is soft, gel-like and transparent. It incorporates a gastric channel and an integrated bite block that reduces the risk of airway channel occlusion. The gastric channel facilitates passage of an orogastric tube and suctioning of regurgitated gastric contents. It is a single use device and available in sizes of 1 to 5 to fit neonates to large adults.

ProSeal LMA

The ProSeal LMA (PLMA) is designed to improve performance during controlled ventilation. The posterior cuff and the increased bulk of the PLMA mask substantially increases the pharyngeal seal. It differs from the LM by having a drainage tube that runs parallel to the airway tube and an integral bite block. When correctly placed, the drain tube opens to the oesophagus and the airway tube opens to the larynx. It is available in sizes of $1^{1}/_{2}$ to 5 to fit infants to large adults.

LMA supreme

The LMA supreme is a single use supraglottic airway device with features similar to the PLMA. It has a slightly larger cuff for a better anatomical fit. The tip of the cuff has been reinforced to prevent folding on the mask aperture. The airway tube is semi-rigid and anatomically curved with integral bite block and incorporates a gastric drainage tube. Pharyngeal seal

is intermediate between cLMA and PLMA at 26–30 cm H_2O. The airway tube is divided by the drain tube in two narrow lumens. This limits its use as a conduit for intubation.

AIR-Q_{SP}

This has a self-pressurising mechanism of sealing. The design allows positive pressure ventilation to self-pressurise the mask cuff. The intra-cuff pressure cycles between the peak airway pressure (usually between 15 and 30 cm H_2O) and the level of PEEP <10 cm H_2O on expiration, maintaining a better seal only when higher inflation pressures are required. The resultant seal pressure on expiration averages 17–20 cm H_2O. Therefore, there is theoretical benefit of decreasing the risks associated with continuous higher sealing pressure such as vascular and nerve pressure injuries.

TRACHEAL TUBES

A tracheal tube is placed in the trachea and considered a definitive airway. The currently available tubes are single use and are made of polyvinyl chloride (PVC) or ivory PVC. Reinforced (armoured) tubes are made of medical grade silicone and are reinforced with wire.

The cuff, when inflated, provides an airtight seal between the tube and trachea. Uncuffed tubes are used in children with a view to avoiding trauma on the delicate tracheal mucosa. There are two types of cuffs: low pressure-high volume and low volume-high pressure. In low pressure-high volume cuffs, the pressure is distributed over a larger area and hence the risk of mucosal ischaemia is lower. The other problems associated with cuffs include herniation of the cuff causing airway obstruction, failure to deflate at extubation and expansion of cuff volume when nitrous oxide is used.

TYPES OF TRACHEAL TUBES

- Oral tubes
- Nasal tubes
- Armoured tracheal tubes
- North and south facing RAE tubes
- Microlaryngeal tubes

- Flexible aluminium shaft (Mallinckrodt laserflex) or stainless steel shaft laser tubes
- Double lumen tubes

SIZE OF TUBES

The cross-sectional area of the narrowest point of the glottic opening (true cords) corresponds to that of an 8.0 mm tracheal tube in men, and a 7.0 mm tube in women. Smaller sizes can be used, since resistance to gas flow is only clinically significant below 6.0 mm and perhaps truly relevant in prolonged ventilation, like the ones in intensive care setting. There is little to be gained and a price to be paid (insertion is more difficult or impossible and the incidence of sore throat is higher), if larger sizes are used.

ORAL OR NASAL TUBE?

Nasal tubes – for

- Often necessary when mouth opening is limited. Fibre-optic intubation using the nasal route is usually easier.
- Provides better surgical access for intraoral surgery.
- Is easier to fix, the patient cannot bite it and it is seen as more comfortable for the patient. There are fewer episodes of accidental extubation and main bronchus intubation.

Nasal tube – against

- Mucosal damage is common, both in the nasal cavity and to the posterior pharyngeal wall. Damage to, or even avulsion of, the turbinates occurs. These can be avoided by using a suction catheter or fibre-optic endoscope as an introducer.
- Severe bleeding can occur – bleeding disorders and infected blood represent contraindications. Mucosal engorgement in pregnant women at term is a relative contraindication.
- Closed base-of-skull fractures can be converted to open fractures by nasal intubation.
- Bacteraemia is common after nasal intubation. Half of nasally intubated patients develop bacterial sinusitis if intubated for more than 4 days.

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TISSUE DAMAGE AND OROTRACHEAL INTUBATION

Sore throat and hoarseness are common, but should settle within 48 hours. Persisting or severe symptoms require investigation. Use of smaller size tracheal tubes, limiting the tracheal tube cuff pressure and use of videolaryngoscopes for intubation have shown to reduce the risk.

Cord palsy after intubation has been variously ascribed to recurrent laryngeal nerve damage and arytenoid dislocation. Dental damage due to laryngoscopy usually affects the left upper first or second incisor. Displaced teeth should be replaced immediately or stored in milk or saline. Perforation of the pharyngeal mucosa by stylets, bougies or tubes can cause mediastinitis or retropharyngeal abscess. Various complications such as palatopharyngeal arch injury, perforation of the soft palate and injury to tonsilar pillars have been reported during videolaryngoscopy.

EXTUBATION

- Desaturation is probably more common at extubation than at any other time. Preoxygenation prior to extubation is recommended.
- Direct laryngoscopy and suction should be performed prior to extubation.
- Laryngospasm, breath holding and severe coughing can complicate extubation, unless the patient is wide awake (patient takes his or her own tube out) or deeply anaesthetized (no reaction to laryngoscopy and suction).
- An 'airway exchange catheter' can be useful in cases where reintubation may have to be performed. These hollow catheters allow oxygenation and are well tolerated by patients.

Current practice is to extubate the trachea with the patient awake and the use of short-acting anaesthetic agents with good recovery profiles (i.e. TCI Remifentanil with an effect site concentration of 1–2 ng/mL) facilitates awake extubation.

The Difficult Airway Society UK (DAS) has published guidelines on extubation. A plan for extubation should be in place prior to induction of anaesthesia and should be reviewed throughout. Further, patient factors (temperature, cardiovascular, respiratory, metabolic and neuromuscular) and the local environmental factors (location, help, monitoring and equipment) should be optimised during preparation stage. A decision should be made whether it is safe to remove the endotracheal tube. If it is unsafe to remove the tube, then a tracheostomy or postponement of extubation are the best options.

CRICOTHYROIDOTOMY

This can be life-saving in airway obstruction leading to a can't intubate, can't oxygenate (CICO) scenario. There are two main types of cricothyroidotomy techniques:

- *Surgical cricothyroidotomy* – Scalpel cricothyroidotomy is recommended by DAS 2015 guidelines as the preferred rescue technique and should be practised by all anaesthetists.
- *Cannula cricothyroidotomy* – Reserved for anaesthetists experienced in its use.

Scalpel cricothyroidotomy involves a stab incision with a scalpel over the cricothyroid membrane and direct placement of a 6 mm cuffed tube. This will protect the airway from aspiration, provide a route for exhalation, allowing low-pressure ventilation using standard breathing systems, and permits end-tidal CO_2 monitoring. The technique incudes continuing to give 100% oxygen via the upper airway, ensuring neuromuscular blockade and extending the patient's neck. In an adult, it requires three simple pieces of equipment – scalpel with number 10 blade, a bougie and a cuffed tracheal tube of 6 mm ID. Once the cricothyroid membrane is identified, a transverse stab incision is made through the cricothyroid membrane, followed by a 90° rotation of the scalpel. The coudé tip of the bougie is inserted along the blade into the trachea. Finally, the lubricated 6.0 mm cuffed ETT is railroaded over the bougie into the trachea. In cases where the cricothyroid membrane is not palpable, an 8–10 cm vertical neck skin incision and blunt dissection should precede the above steps.

Cannula cricothyroidotomy can be performed using a narrow bore cannula with an approximate internal diameter (ID) of 2 mm or a large bore cannula with

approximate ID of 4 mm or greater. With narrow bore cannula, a suitable high-pressure ventilation system (a Sanders injector or manual jet insufflator) has to be readily available. Barotrauma is a possible complication of high-pressure jet ventilation. Other simpler O_2 delivery devices such as Rapid O_2 (Meditech) (a Y-shaped tubing with a large exhaust port that permits exhalation through the cannula) or Ventrain (Ventinova) have become available as alternatives to jet ventilation, claiming reduced chances of causing barotrauma.

Large bore cannulas such Melker (Cook Critical Care) and Quicktrach (VBM Medical) are available as both cuffed and non-cuffed versions. A cuffed tube inserted through a cricothyroidotomy has the advantage of providing effective ventilation.

As a part of preoperative airway assessment, the cricothyroid membrane should be located. In patients where the cricothyroid membrane is not palpable, ultrasound can be used for locating it.

TRACHEOSTOMY

A tracheostomy tube is a curved tube, inserted into the trachea through a tracheostome. Tracheostomy is usually performed through the second, third or fourth tracheal ring. There are many different types of tube available based on the internal diameter, length, presence of fenestrations and presence of a cuff. Some of them have two tubes (inner and outer tubes); the inner tube can be removed for cleaning at regular intervals. Tracheosotomy is indicated in intensive care patients who require prolonged ventilation. It helps to reduce the amount of sedation used, facilitates weaning and prevents some of the complications of long-term endotracheal intubation such as vocal cord damage.

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DIFFICULT AIRWAY – OVERVIEW

CYPRIAN MENDONCA

The primary aim of airway management is to provide oxygenation. When difficulty arises, a safe method of oxygenating the patient should be adopted. To do so, a basic plan and a structured approach in managing both anticipated and unanticipated difficult airway is essential. Any anticipated difficulty should be communicated with the team during team brief and sign in as a part of the WHO surgical safety checklist. If plan A fails, one should have plan B or plan C ready. When in doubt, additional assistance and help should be requested.

CAUSES AND DEFINITIONS

A difficult airway can be encountered during any phase of anaesthesia. It may also be encountered outside the operating theatre in the ICU, emergency department and pre-hospital environment. The incidence is influenced by several factors: environment, experience of the anaesthetist, patient, available equipment, assistance and the urgency of the procedure. A difficult airway is not limited to difficult intubation, but it may be manifest as difficulty in mask ventilation, difficulty in securing a supragottic airway, difficulty in securing a surgical airway or difficulty experienced at extubation.

Failed mask ventilation occurs in about 1:1500 and failed intubation 1:2000 general surgical patients. Totally satisfactory definitions for difficult intubation or difficult mask ventilation are elusive since difficulty is subjective and a clinical situation is often a mixture of problems.

CAUSES

- *Anatomical* – Short neck, receding mandible, high arched palate, buck teeth, large breasts.
- *Congenital* – Down syndrome, Pierre-Robin syndrome, Treacher Collins syndrome.
- Inflammatory diseases: rheumatoid arthritis, ankylosing spondylitis.
- Infections – Quinsy, epiglottitis.
- *Reflexes* – Laryngospasm, breath holding, regurgitation.
- *Stiffness* – Temporomandibular joint arthritis, cervical spine fixation devices, contractures, scleroderma.
- *Swelling* – Ludwig's angina, trauma, burns, cervical haematoma, anaphylaxis, tumours (e.g. cystic hygroma, goitre, oropharyngeal and laryngeal cancer).
- *Others* – Radiotherapy to neck, acromegaly, obesity and pregnancy.

DEFINITIONS

Difficult airway

The ASA Task Force on Management of the Difficult Airway defined a difficult airway as 'the clinical situation in which a conventionally trained anesthesiologist experiences difficulty with mask ventilation, difficulty with tracheal intubation, or both'. In clinical practice, the difficult airway is a complex interaction between patient factors, clinical setting and the skills and experience of the individual anaesthetist. The severity of difficulty experienced is subjective to each individual's usual practice and experience.

Difficult mask ventilation

In 2013, ASA defined difficult mask airway ventilation as follows:

It is not possible for the anesthesiologist to provide adequate ventilation due to one or more of the following problems: inadequate mask seal, excessive gas leak, or excessive resistance to the ingress or egress of gas.

Signs of inadequate face mask ventilation include absent or inadequate chest movement, absent or inadequate breath sounds, cyanosis, gastric air entry

or dilatation, decreasing or inadequate oxygen saturation, absent or inadequate exhaled carbon dioxide, absent or inadequate spirometric measures of exhaled gas flow and haemodynamic changes associated with hypoxaemia or hypercarbia.

In clinical practice one may experience a varying degree of difficulty with mask ventilation. An oropharyngeal airway, nasopharyngeal airway or both, high gas flow of >15 L/min and two-person bag and mask ventilation may be needed. Finally, despite all measures it may be impossible to ventilate as judged by lack of chest wall movement and absence of $ETCO_2$.

Causes

- *No seal between face and mask* – Reasons range from absence of teeth and presence of beard to massive facial trauma.
- *Reduced cross-sectional area of airway* – Tissue swelling from infection, trauma, burns, tumour or oedema (cervical haematoma, anaphylaxis).
- *Standard airway-opening manoeuvres not possible* – Cervical rigidity or poor mouth opening and/or mandibular protrusion may make it impossible to counteract the effects of anaesthesia on airway patency. Patients in halo-body frames or with interdental wiring are examples of this problem.

Difficult supraglottic airway device placement

- The situation where supraglottic airway placement requires multiple attempts in the presence or absence of tracheal pathology.

Difficult laryngoscopy

A situation where it is not possible to visualize any portion of vocal cords after an optimal attempt at laryngoscopy using a laryngoscope with which the operator is most experienced. Cormack and Lehane grading (Figure 26.2) of glottic view is still widely used. Grade 2 view is subdivided into grade 2a (visualization of parts of the laryngeal inlet), and grade 2b (visualization of only the arytenoids) and grade 3 view into 3a and 3b where 3a describes visualization of lifted epiglottis and 3b visualization of epiglottis closely applied to the posterior pharyngeal wall. Laryngoscopic view can also be described as percentage of the glottic opening visible. For practical
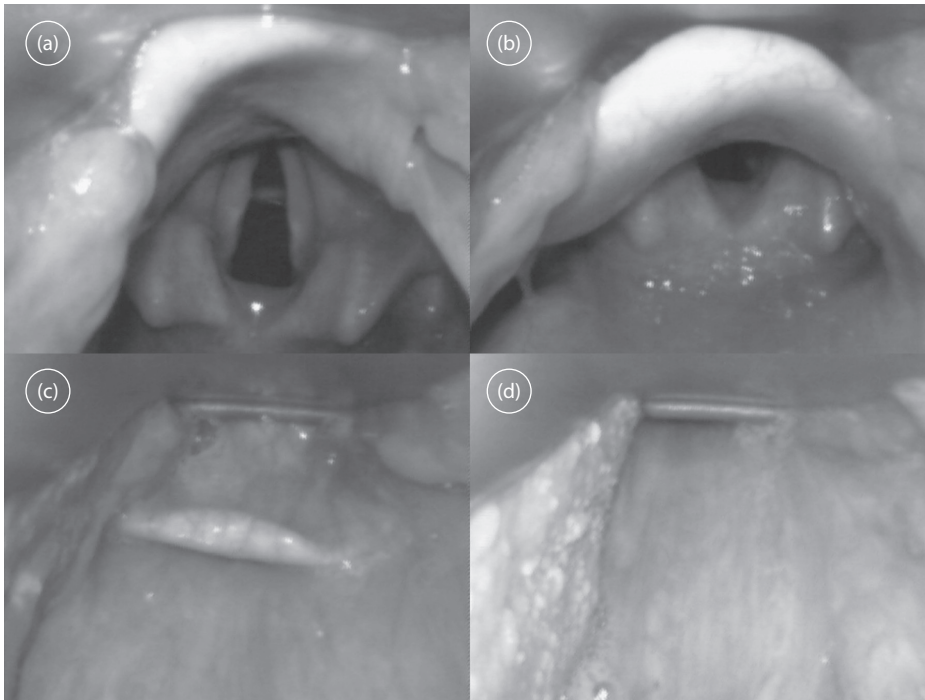
Figure 26.2 Cormack and Lehane's classification of laryngoscopic view.

purposes when a part of the laryngeal inlet is visible, tracheal intubation is possible.

Causes

- Difficult laryngoscopy is caused by poor mouth opening, short muscular neck, protruding upper teeth, receding mandible and stiffness of the cervical spine, particularly the craniocervical junction. Swelling of the oropharyngeal tissues can also prevent vision.
- Diseases regularly associated with difficulty include arthritis (particularly rheumatoid arthritis), oropharyngeal infections and tumours, ankylosing spondylitis, acromegaly and the Klippel–Feil abnormalities of the cervical spine.

Difficult tracheal intubation

In day-to-day practice, difficult intubation tends to be synonymous with difficult laryngoscopy. However, with videolaryngoscopy, despite a good view of the cords, difficulty may be encountered in advancing the tube through the glottis. In 2013, the ASA defined difficult tracheal intubation as a situation where tracheal intubation requires multiple attempts, in the presence or absence of tracheal pathology.

The Canadian Task Force defined difficult intubation as when multiple attempts or more than one operator is required or an adjunct such as a bougie or an alternative intubation device is required after unsuccessful use of the primary device.

Difficult cricothyroidotomy

The Canadian Task Force defined a difficult transtracheal surgical airway as one that requires excessive time or multiple attempts.

THE DIFFICULT AIRWAY SOCIETY GUIDELINES

The Difficult Airway Society (DAS) 2015 guidelines on managing unanticipated difficult intubation emphasises optimising the first attempt at laryngoscopy,

recognising failure early and avoiding airway trauma by multiple attempts at laryngoscopy and maintaining oxygenation. The basic structure of the DAS 2015 guideline includes four plans (Figure 26.3).

- *Plan A* – Facemask ventilation and tracheal intubation.
- *Plan B* – Maintaining oxygenation and supraglottic airway device insertion.
- *Plan C* – Facemask ventilation.
- *Plan D* – Emergency front of neck access.

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CROSS-REFERENCE

Difficult airway management, Chapter 26

Figure 26.3 Difficult Airway Society difficult intubation guidelines: overview. (Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. C. Frerk, V. S. Mitchell, A. F. McNarry, C. Mendonca, R. Bhagrath, A. Patel, E. P. O'Sullivan, N. M. Woodall and I. Ahmad, (2015). Difficult Airway Society intubation guidelines working group. *British Journal of Anaesthesia*, **115**(6): 827–848 doi:10.1093/bja/aev371.)

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AWAKE FIBRE-OPTIC INTUBATION

Nasal intubation is frequently easier in this group of patients because of limited mouth opening, and the poor 'angle of attack' with the oral route.

Topical anaesthesia is more effective if glycopyrrolate is given to dry the mucosa. A vasoconstrictor should be applied to the nasal mucosa (xylometazoline or phenylephrine) before endoscopy.

Lidocaine is poorly absorbed from the nasopharynx; doses up to 8 mg/kg are acceptable. Lidocaine is an irritant to the nasal and glottic mucosa; initial application should be with a small quantity of 4% solution. Application in the form of atomised spray using the Mackenzie technique or mucosal atomisation device is generally well tolerated by patients. The epiglottis, glottis and trachea are liberally sprayed with 4% lidocaine through the endoscope (about 4 mL) using the 'spray as you go' technique (SAYGO). An alternative is to inject 3–4 mL 4% lidocaine through the cricothyroid membrane. Coughing can be very vigorous, but will result in satisfactory anaesthesia. The glottis will not be anaesthetized if the patient does not cough.

Tube size is an important factor contributing to the success of fibre-optic intubation. Tube sizes between 6 and 7 mm ID are ideal. The larger the size, the more likely is the difficulty in railroading the tube over the fibrescope. Flexible metal-reinforced tubes with short and soft bevel are recommended, and rotation of the tube as it is passed through the cords is helpful. Severe laryngeal damage has been reported after awake fibre-optic intubation; the glottic reflexes must be obtunded, force not used, and multiple attempts not made.

It is sensible to administer some sedation during the procedure. Target controlled infusion of remifentanil (1–4 ng/mL) or propofol (0.5–1.0 mcg/mL) is commonly used for providing conscious sedation.

Alternatively, if the mouth opening is about 20–25 mm or greater, awake tracheal intubation can also be performed using videolaryngoscopes.

In certain scenarios such as paediatric patients, patients with learning disabilities, hypersensitivity to local anaesthetics and uncooperative patients, it may not be possible to perform awake intubation.

CYPRIAN MENDONCA

A difficult airway generally means difficult tracheal intubation. In clinical practice there may be difficulty with mask ventilation, difficulty with obtaining an adequate view of the larynx and difficulty in placing the tube in the trachea despite an adequate view of the larynx. The management depends on whether the difficulty is anticipated or unanticipated. The management plan is also influenced by the degree of difficulty anticipated or encountered, location, availability of equipment and expertise. A structured approach to the management and having a series of plans in place (management strategy) is more likely to result in a successful outcome.

The basic principles of airway management involve:

- Preoperative assessment and anticipation
- Preparation and choosing appropriate plans (primary plan and alternative plans)
- Maintaining oxygenation throughout the procedure
- Planning extubation
- Maintaining records and communication

ANTICIPATED DIFFICULT AIRWAY

The management depends on

- Severity of difficulty expected
- Presence or absence of upper airway obstruction

SEVERE DIFFICULTY EXPECTED

Awake fibre-optic intubation is the method of choice, and is acceptable to most patients. Fibre-optic nasendoscopy can be regarded as a part of preoperative examination and should be presented to the patient in that manner. The cardiovascular stability associated with fibre-optic intubation under topical anaesthesia is an attractive feature of the technique.

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ANTICIPATED DIFFICULT AIRWAY WITH AIRWAY OBSTRUCTION

The management depends on:

- Urgency of action required (severity of airway obstruction and cause of airway obstruction)
- Site of airway obstruction

Acute airway obstruction can occur in patients with a previously normal airway due to angioneurotic oedema, foreign body inhalation and post-surgery neck haematoma.

Chronic airway obstruction can occur with benign and malignant diseases involving the glottis and supraglottic regions, radiotherapy to the neck and previous surgery on the neck.

Stridor is the cardinal sign of a narrowed airway, which is said to occur at rest when the airway diameter is reduced by 50%. The diameter of the airway can be dangerously reduced without stridor being present. Complaints of awakening at night with sensations of choking are characteristic of glottic obstruction. Patients with airway obstruction should be allowed to adopt their preferred position and interfered with as little as possible. If the patient panics, the situation is likely to become truly dreadful.

Cervical haematomas (thyroid or anterior cervical surgery) produce oedema of the glottis and periglottic tissue. The swelling may be largely due to lymphatic obstruction and the haematoma may be small. The wound should be opened immediately to relieve lymphatic and venous obstruction.

When immediate intervention is required, basic life support protocol including providing high flow oxygen to the patient should be initiated. Inhalation of nebulised epinephrine (1 mL of 1:1000, in 10 mL of saline) may buy some time. In cases of anaphylaxis, epinephrine (0.5–1.0 mL of 1:10,000) should be given intravenously. Heliox may be beneficial in improving the turbulent flow across the obstruction.

- Recognition and evaluation of the critical airway obstruction is paramount.
- Team management in the form of senior anaesthetist, ENT surgeon and trained ODP is essential.

Patients with stridor can be divided into two groups.

- *Severe stridor* – Nasendoscopy reveals intubation is impossible. In this group, the patient should have tracheostomy under local anaesthetic.
- *Moderate stridor* – Nasendoscopy reveals intubation is possible. In this situation, a decision should be made whether to proceed for intubation using fibrescope or to undertake controlled intravenous induction with neuromuscular blockade. An ENT surgeon gowned and gloved, ready to perform tracheotomy or to perform rigid bronchoscopy with jet ventilation facility, should be present during induction.

Patients may find it difficult to tolerate a supine position, so induction of anaesthesia and initial preparation of surgery should be performed in the head-up position.

There are two possible problems with awake fibre-optic intubation in these patients.

- Local anaesthetic spray can lead to laryngospasm and complete airway obstruction.
- Advancement of fibre-optic scope through the lesion can cause 'cork in the bottle' phenomenon leading to complete airway obstruction.

Pre-oxygenation using high flow humidified nasal oxygenation is useful if induction of general anaesthesia is planned as it has been shown to extend the duration of apnoea without desaturation. High flow nasal oxygenation should be continued during apnoea and attempts should be made to maintain the airway patency.

OROPHARYNGEAL TUMOURS

In patients with oropharyngeal tumours, awake fibre-optic intubation is a suitable option. The other possible option includes induction of anaesthesia following prophylactic placement of cricothyroid cannula.

SUBGLOTTIC OR INFRAGLOTTIC TUMOURS

The site and extent of the airway obstruction should be determined using a CT/MR scan. If assessment of the airway does not indicate difficult larygoscopy, then conventional intubation can be planned. Evaluation of the airway beyond the tube should be examined with the fibre-optic bronchoscope after the intubation.

ANTERIOR MEDIASTINAL MASSES

Obstruction of the trachea or main bronchus can occur during anaesthesia in symptomless patients. In a patient with a history suggestive of mediastinal mass, chest X-ray and CT scan can demonstrate the site of airway obstruction. Flow/volume loop studies are useful in quantifying the degree of airway obstruction and also in differentiating extrathoracic from intrathoracic airway obstruction. If the obstruction is likely to hamper the placement of a small tube and for lower tracheal and bronchial obstruction, cardiopulmonary bypass should be considered.

EXTUBATION AFTER INTUBATION FOR AIRWAY OBSTRUCTION

The minimum period of intubation should probably be 24 hours. Adequate sedation must be prescribed to prevent accidental extubation. A small tube should have been passed so that deflation of the cuff and blocking the tube can demonstrate a satisfactory airway.

LARYNGOSCOPY AND THE UNSTABLE CERVICAL SPINE

All airway manipulations will cause a degree of movement at the craniocervical junction and in the cervical spine. There is no evidence that laryngoscopy with manual inline stabilisation is more dangerous than any other method of intubation. Many patients with severe instability are in cervical fixation devices, which restrict mouth opening and cervical movements. In these circumstances, awake intubation is probably the method of choice. In trauma scenarios, manual inline stabilization of the cervical spine results in suboptimal conditions for a direct laryngoscopy with Macintosh blade. Use of an alternate blade such as McCoy or a videolaryngoscope may have a role in these cases.

UNANTICIPATED DIFFICULT AIRWAY

This is a situation when a preoperative assessment reveals a normal airway but, following induction of anaesthesia, there is difficulty with mask ventilation or with tracheal intubation or with both. The scenario where both mask ventilation and tracheal intubation is impossible (can't intubate, can't oxygenate) is very rare.

The principles of management involve:

- Maintenance of oxygenation
- Prevention of the airway trauma due to repeated attempts
- Recognising the difficulty and requesting help
- Choosing an alternate technique/device rather than persisting in attempts with the same technique/device

The above principles can be implemented by adhering to a definitive, clear algorithm-based pre-existing plan. In the UK, the Difficult Airway Society 2015 guidelines are recommended. A flow chart, consisting of plans A, B, C and D has been described for managing unanticipated difficult tracheal intubation in adults (Figure 26.4).

PLAN A

Plan A is facemask ventilation and tracheal intubation. The first attempt at the laryngoscopy should be performed under optimal conditions. It includes an adequately anaesthetised/paralysed patient and optimum position of the head and neck. Anaesthesia should be induced following adequate pre-oxygenation. In obese patients, ramping is used instead of 'sniffing the morning air' position.

External laryngeal manipulation or BURP (backward, upward and right sided pressure on thyroid cartilage) manoeuvre improves the laryngeal view. If the best first attempt results in a poor view, then the decision should be made either to use a different laryngoscope (videolaryngoscope/direct laryngoscope) or

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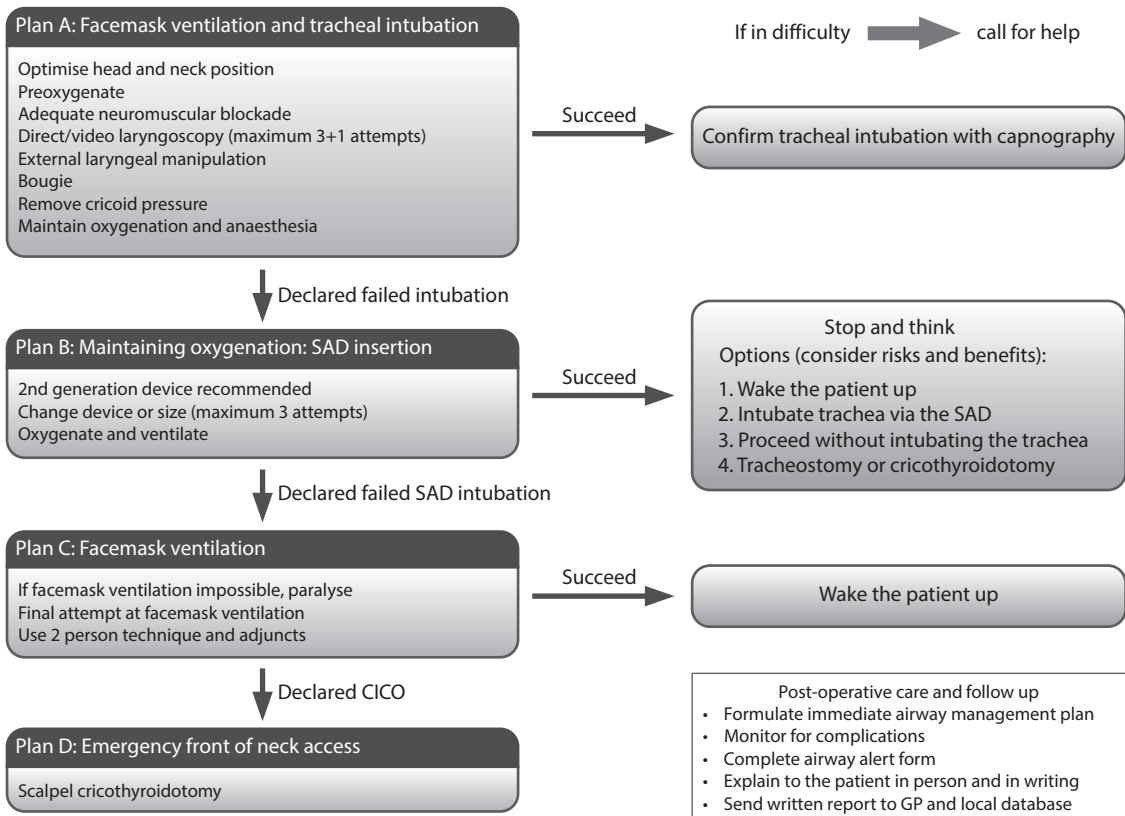
Figure 26.4 Management of unanticipated tracheal intubation in adults. (Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. C. Frerk, V. S. Mitchell, A. F. McNarry, C. Mendonca, R. Bhagrath, A. Patel, E. P. O'Sullivan, N. M. Woodall and I. Ahmad, (2015). Difficult Airway Society intubation guidelines working group. *British Journal of Anaesthesia* **115**(6): 827–848. doi:10.1093/bja/aev371.)

bougie or to move on to plan B. Adequate oxygenation should be ensured between the attempts at laryngoscopy.

The bougie is an outstandingly useful item and should always be ready for use. Blind insertion of a bougie and hold up sign (indicating the bougie tip reaching the small bronchi) can be associated with airway trauma. Some videolaryngoscopes (non-channelled) require a preshaped bougie or purpose-made stylet to facilitate passage of a tube through the cords.

The maximum number of laryngoscopy attempts should be limited to three and another attempt can be performed by a more experienced anaesthetist.

It is well recognized that excessive cricoid pressure may lead to poor laryngoscopic view. Therefore, cricoid pressure should be limited to 30 N. If required, pressure should be released with suction in hand.

PLAN B

Plan B is maintaining oxygenation using a supraglottic airway device (SAD). DAS 2015 Guideline recommends a second generation SAD with integrated gastric drainage tube, minimizing the risk of aspiration. Once satisfactory oxygenation is achieved, a short pause is essential to think and choose further airway management options. In most situations, waking up the patient is the most appropriate action.

PLAN C

Plan C involves an optimum final attempt at facemask ventilation using a two-handed technique. One should ensure adequate paralysis before declaring failed mask ventilation.

PLAN D

Failure of plans A, B and C leads to a situation of can't intubate and can't oxygenate (CICO). In this circumstance, an emergency front of neck access (Figure 26.5) is required without delay. Success depends on the familiarity of the technique, decision making and immediate availability of equipment and help. The available methods of front of neck access (cricothyroidotomy) have been briefly described in the previous section (refer to artificial airways).

Call for help

Continue 100% O_2
Declare CICO

Plan D: Emergency front of neck access

Continue to give oxygen via upper airway
Ensure neuromuscular blockade
Position patient to extend neck

Scalpel cricothyroidotomy

Equipment: 1. Scalpel (number 10 blade)
2. Bougie
3. Tube (cuffed 6.00 mm ID)

Laryngeal handshake to identify cricothyroid membrane

Palpable cricothyroid membrane
Transverse stab incision through cricothyroid membrane
Turn blade through 90° (sharp edge caudally)
Slide coude tip of bougie along blade into trachea
Railroad lubricated 6.0 mm cuffed tracheal tube into trachea
Ventilate, inflate cuff and confirm position with capnography
Secure tube

Impalpable cricothyroid membrane
Make an 8–10 cm vertical skin incision, caudad to cephalad
Use blunt dissection with fingers of both hands to separate tissues
Identify and stabilise the larynx
Proceed with technique for palpable cricothyroid membrane as above

Post-operative care and follow up
- Postpone surgery unless immediately life threatening
- Urgent surgical review of cricothyroidotomy site
- Document and follow up as in main flow chart

Figure 26.5 Failed intubation, failed oxygenation in the paralysed anaesthetized patient. (Reproduced from Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. C. Frerk, V. S. Mitchell, A. F. McNarry, C. Mendonca, R. Bhagrath, A. Patel, E. P. O'Sullivan, N. M. Woodall and I. Ahmad, (2015). Difficult Airway Society intubation guidelines working group. *British Journal of Anaesthesia* **115**(6): 827–848. doi:10.1093/bja/aev371.)

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Artificial airways, Chapter 26
Difficult airway: overview, Chapter 26

DIFFICULT AIRWAY – NEW DEVICES

CYPRIAN MENDONCA

An ideal device intended for use in a difficult airway scenario should be easy to use, simple and quick to set up, suitable for both paediatric and adult use, suitable for both nasal and oral route, portable and reliable. The term 'videolaryngoscopes' is now commonly used in relation to the recently introduced new laryngocscopes based on the principle of indirect laryngoscopy.

DIRECT LARYNGOSCOPY

A 'line of sight' must be established from eye to glottis by aligning the oral, pharyngeal and laryngeal axes (Figure 26.6). This requires:
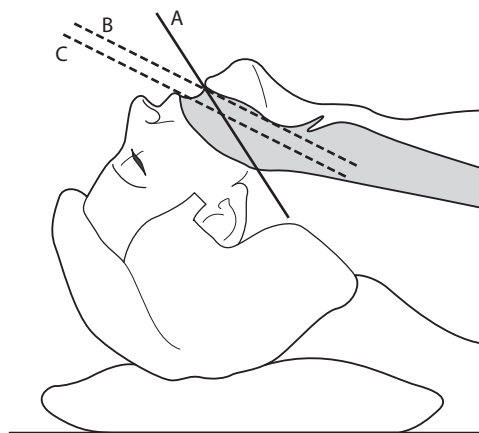
Figure 26.6 Head in neutral position, none of the axes are aligned. A, oral axis; B, pharyngeal axis; C, laryngeal axis. All three axes are aligned by flexion of cervical spine (elevation of head) and extension at the atlanto-occipital joint.

- Extension of the head at the atlanto-occipital joint, combined with a slightly flexed cervical spine – the 'sniffing the morning air' position, described by Magill.
- Artificial protrusion of the mandible, tongue and hyoid bone with the blade of the laryngoscope.

It is not always possible to achieve a line of sight; an inadequate view of the larynx is the result. A poor laryngoscopic view using direct laryngoscopy necessitates additional force, external laryngeal manipulation, use of a gum elastic bougie or stylet or an alternate technique to achieve success.

INDIRECT LARYNGOSCOPY

A simple example of indirect laryngoscopy is visualizing the image of the larynx using an otolaryngoscopic mirror illuminated with light, placed in the patient's mouth. Indirect laryngoscopy does not require the alignment of oral, pharyngeal and laryngeal axes. Therefore, it requires less force to be applied and comparatively fewer haemodynamic changes. A classification of indirect laryngoscopes is shown in Table 26.1.

OPTICAL STYLETS

The Bonfils (Karl Storz Endoscopy Ltd, Tuttlingen, Germany) is a rigid fibre-optic stylet, of 40 cm in

Table 26.1 Classification of indirect laryngoscopes

Optical stylets			Bonfils	Shikani		
Video-laryngoscopes	With tube channel		Airtraq	Pentax Airway Scope	King Vision	
	With guiding plate		Venner APA			
	Without tube channel		Bullard	C-Mac	Glidescope	McGrath

length and 5 mm in outer diameter, with an angulated distal end. It can be used in patients with limited mouth opening.

The Shikani optical stylet (Clarus Medical, Minneapolis, MN) is a high-resolution endoscope and has a malleable stainless-steel sheath. It is available in two different sizes (adult and paediatric). It can be used in conjunction with a Macintosh laryngoscope.

Both of the above devices can be used with an eyepiece or can be connected to a video camera system. Both have been shown to cause less movement of the upper cervical spine when compared to the Macintosh laryngoscope.

VIDEOLARYNGOSCOPES

Videolaryngoscopes (Figures 26.7 and 26.8) transmit the image to an external monitor, from a miniature video camera placed at the distal end, via a fibre-optic bundle or a system of prisms. They can be broadly divided into two main types: those with

an anatomically shaped blade with an inbuilt tube channel, which directs the tracheal tube into the larynx (channelled videolaryngoscopes); and those with blades resembling the Macintosh blade (non-channelled videolaryngoscopes). Angulated 'difficult airway' blades, characterized by an angle that is sharper than the curvature of the standard Macintosh blade, are often manufactured for the non-channelled videolaryngoscopes. These blades provide a better view of the anterior larynx, but require a rigid stylet to direct the tracheal tube into the glottis.

C-Mac

The C-Mac® video laryngoscope (Karl Storz Endoscopy Ltd, Tuttlingen, Germany) consists of a reusable blade, or a single use blade (C-Mac S), which is similar in shape to the Macintosh blade. It exhibits a curvature of 60° with a digital camera view of 80°. A small digital camera and a high power light emitting diode are located at the distal
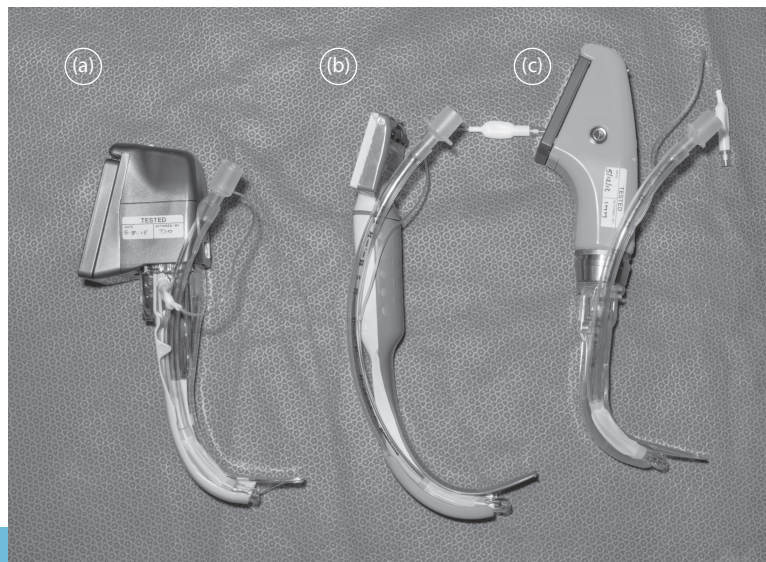
Figure 26.7 Videolaryngoscopes with tube channel. (a) Airtraq; (b) King Vision; (c) Pentax Airway Scope.

Figure 26.8 Video laryngoscopes without tube channel. (a) C-Mac; (b) Glidescope; (c) McGrath.

third of the blade. The camera is connected to a high-resolution, 7 inch TFT (thin-film transistor) monitor with a single cable. The blade is available in three different sizes (2, 3 and 4). A more curved version of the blade (D-Blade) has been introduced as an aid to difficult intubation.

GLIDESCOPE

The Glidescope® (Verathon Medical, Burnaby, Canada) consists of a video baton that incorporates a high resolution camera, a light emitting diode and a portable LCD (liquid crystal display) monitor. The disposable plastic blades are available in different sizes that need to be mounted on the video baton. A paediatric version of the video baton is also available. Successful placement of the tracheal tube requires the use of an angulated stylet.

McGrath

The McGrath® (Aircraft Medical Ltd, Edinburgh, UK) is a battery-powered portable laryngoscope with a 33 mm by 22.5 mm LCD screen mounted on top of the laryngoscope handle. The camera stick incorporates a miniature camera and a light source. A disposable, clear acrylic blade (McIntosh design) covers

the camera stick. A more angulated difficult airway blade (X blade) is also available, which requires an angulated stylet to facilitate tube placement.

VENNER APA

The Venner APA™ (Venner Medical International, St. Helier, Jersey) consists of an integrated display screen, handle and camera stick with a high intensity LED light source. Interchangeable, disposable blades are mounted onto the camera module. Four blade types are available: size 3 and 4 Macintosh style blades, an angulated difficult airway blade and an angulated blade with a guiding plate. With the latter, passage of the endotracheal tube can be achieved without a stylet, as the plate directs the tube towards the glottic opening.

PENTAX AIRWAY SCOPE (AWS)

The Pentax AWS® (Hoya Corporation, Tokyo, Japan) is also a battery powered laryngoscope consisting of a handle with a built-in 2.4 inch LCD screen and a 12 cm cable with a miniature video camera. The disposable polycarbonate blade (PBLADE) incorporates a tube channel, which guides the tube into the glottic opening when the image is correctly aligned on the monitor.

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AIRTRAQ

The Airtraq® (Prodol Meditec SA, Vizcaya, Spain) is a single use device which consists of an anatomically shaped laryngoscope with a built-in tube channel. A battery operated light emitting diode is present at the tip and provides the illumination. The laryngeal view is transmitted to the proximal viewfinder through a combination of lenses and prisms. Facilities are also available for transmitting the image to an integrated monitor or an external wireless monitor.

KING VISION

The King Vision® videolaryngoscope (Ambu, Ballerup, Denmark) is a battery operated videolaryngoscope that consists of an anatomically shaped, single use blade/handle unit, and an integrated reusable display monitor. The blade has an integrated tube channel which directs the tube towards the glottis.

LARYNGOSCOPY VERSUS TRACHEAL INTUBATION

The process of tracheal intubation can be divided into two steps.

- Visualisation of the glottis
- Placement of the tube in the trachea

In general, visualization of the glottis is easy with most indirect laryngoscopes but further skill is required for placing the tube in the trachea. The success rate and learning curve for correct tube placement can be variable amongst the different videolaryngoscopes. Despite a grade 1 laryngeal view, tracheal tube placement can be difficult. This is because oral, pharyngeal and tracheal axes are not aligned and therefore the tube must be inserted around a curvature, without direct vision. Therefore, an angulated stylet or tube introducer is required.

There are several benefits and roles for videolaryngoscopes:

- Videolaryngoscopes have a role in teaching and evaluating the laryngoscopy procedure.

- Videolaryngoscopes enable an anaesthetic assistant to be more effective in assisting intubation. The assistant can perform the external laryngeal manoeuvres and cricoid pressure more effectively as he or she can observe the laryngeal view on the monitor.
- Videolaryngoscopes can contribute to better record keeping, guarding against claims of airway injuries, through recording images and videoclips. Images obtained by the videolaryngoscopes in cases of difficult airway can be used to plan future airway management.
- They require less force and therefore cause less haemodynamic changes.
- They are used for managing difficult intubation in both anticipated and unanticipated situations. They are included in the plan A of Difficult Airway Society guidelines in managing an unanticipated difficult intubation.

There are reports of airway trauma during videolaryngoscopy. During videolaryngoscopy, the operator's attention is diverted from the direct view of the proximal airway to the indirect view of the glottis on the monitor. Advancement of tracheal tube and stylet blindly into the proximal airway can result in airway trauma such as palatal perforation, palatopharyngeal arch tear and injury to tonsilar pillars. Therefore, when using a videolaryngoscope the tip of the tube and stylet should be introduced into the oropharynx under direct vision and then advanced as guided by the indirect view on the monitor.

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DIFFICULT AIRWAY – PREDICTION

ALEKSANDRA NOWICKA

Prediction of a difficult airway is based on history, clinical examination and investigations. Previous 'difficult airway alerts', surgery, injuries or radiotherapy in the head and neck region, the presence of head and neck pathology (e.g. tumours, oedema, snoring, obstructive sleep apnoea) can suggest a possible difficult airway. A detailed clinical examination involves predictive tests followed by further investigations such as neck X-rays, CT scans and nasendoscopy as indicated. Poor airway assessment and failure to alter the airway management technique in response to findings at assessment commonly contribute to the failure and adverse outcomes. Predicting a difficult airway should therefore go beyond simply recording the history, examination and investigations. It requires application of clinical judgement to formulate an airway strategy based on these findings.

Airway assessment should be focused on predicting difficulties during each stage of airway management. These include mask ventilation, insertion of a supraglottic airway device, tracheal intubation, cricothyroidotomy and, finally, extubation.

PREDICTING DIFFICULT MASK VENTILATION (DMV)

Predictors of DMV include full beard, Mallampati grade 3 or 4, age more than 55 years, body mass index >30 kg m^{-2}, limited jaw protrusion, edentulous, history of snoring and sleep apnoea. These can be remembered using the mnemonic 'OBESE' (obese, bearded, elderly, snorers, edentulous). The reported incidence of impossible bag and mask ventilation is 0.15% and history of previous neck radiotherapy is the most important predictor of this rare but potentially catastrophic event.

Obesity is an independent predictor of difficult mask ventilation and is associated with obstructive sleep apnoea. Despite preoxygenation these patients can desaturate quickly during induction due to reduced oxygen reserve. Limited protrusion of the mandible, Mallampati and large neck circumference are predictors of difficult mask ventilation.

PREDICTING DIFFICULT SUPRAGLOTTIC AIRWAY DEVICE INSERTION

Limited mouth opening with inter-incisor distance <2 cm is likely to cause difficulties with insertion of all airway devices. Short thyromental distance and limited neck movement are also risk factors for difficult ventilation via a supraglottic airway.

PREDICTING DIFFICULT INTUBATION

Cases with readily identifiable problems, such as facial injuries, can be predicted. Serious difficulty is, fortunately, very rare in apparently normal people. It is therefore unlikely that any single predictive method will be successful.

Various tests have been described (Mallampati, Patil), which appear to perform well when applied retrospectively. However, prospective trials have shown that the false-positive rate associated with prediction in a general population is very high. It is

also unfortunately the case that the available tests have sensitivities of about 50% (i.e. half the cases are missed).

If fibre-optic intubation is planned, factors such as patency of nasal passages, presence of nasal polyps, coagulopathy or excessive airway secretions should be taken into consideration.

AVAILABLE TESTS

MALLAMPATI TEST (WITH SAMPSON AND YOUNG'S MODIFICATION)

The patient sits opposite to the anaesthetist with mouth wide open and tongue protruded. Depending on the view of the pharynx, four classes have been described.

Class 1 – Faucial pillars, soft palate, posterior pharyngeal wall and uvula are seen.
Class 2 – Soft palate, part of posterior pharyngeal wall and base of uvula are seen.
Class 3 – Only soft palate visible.
Class 4 – Soft palate is not visible.

The test estimates the size of the tongue in relation to the oral cavity. Class 3 and 4 are associated with difficult laryngoscopy using a Macintosh laryngoscope. There is interobserver variation and, as a stand-alone test, it has been found to have poor sensitivity and poor positive predictive values in general surgical patients. In a recent meta-analysis only 35% of patients with a difficult intubation were identified as Mallampati class 3 or 4.

INTER INCISOR GAP

With the mouth maximally open, the gap between the incisors is measured. If <3 cm, difficult laryngoscopy is likely.

MANDIBULAR PROTRUSION

Mandibular protrusion can be assessed on an ABC basis:

Class A – Able to protrude the lower incisors anterior to the upper incisors.

Class B – Lower incisors reach just the margin of the upper incisors.
Class C – Lower incisors cannot protrude to the upper incisors.

Direct laryngoscopy is always difficult in patients in class C but it is a rare finding, largely confined to patients with rheumatoid arthritis.

THYROMENTAL DISTANCE (PATIL'S TEST)

Thyromental distance is an indicator of mandibular space. Measure the distance between the uppermost part of the thyroid cartilage to the tip of the chin (mentum), with the neck fully extended and mouth closed. If <6 cm, it predicts difficult laryngoscopy.

STERNOMENTAL DISTANCE

Sternomental distance is the distance from the sternal notch to the tip of the chin with the neck fully extended and mouth closed. If <12.5 cm, it predicts difficult laryngoscopy.

MOVEMENT OF CERVICAL SPINE

Flexion and extension movements of the cervical spine and atlanto-occipital movement are important for direct laryngoscopy. Cervical spine movement can be assessed by placing a finger on the patient's chin and the other one on the occipital protuberance. The head is extended maximally and the position of chin in relation to occipital protuberance noted.

- If chin is higher than the occipital protuberance – Normal cervical spine mobility.
- If at the same level – Moderate limitation of cervical spine mobility.
- If chin is lower than the occipital protuberance – Severe limitation of cervical spine mobility.

SCORING SYSTEMS

A combination of the above tests has a better predictive value than any single test. A meta-analysis by Shiga et al. showed that combining Mallampati score and thyromental distance is a better predictor of difficult

laryngoscopy. It has also been suggested that a ratio of the neck circumference to the thyro-mental distance of >0.5 predicts difficult intubation in obese patients. Several weighted, multivariate scoring systems, such as Wilson score or the Simplified Airway Risk Index (SARI), have been published that increase the positive predictive value of testing.

DIFFICULT CRICOTHYROIDOTOMY

Obesity, goitre, neck tumours, previous neck surgery or radiotherapy and limited extension of the neck may result in problems with performing emergency cricothyroidotomy. The choice and success is likely to depend on the ability to identify the cricothyroid membrane. During preoperative airway assessment, an attempt should be made to palpate the cricothyroid membrane. In cases where the cricothyroid membrane is not palpable due to difficult neck anatomy, ultrasound can be used to locate the criothyoroid membrane.

EXTUBATION

While predicting problems during extubation, difficulties during initial airway management, perioperative airway deterioration and limited access to the airway should be taken into account. Other risk factors include the reactive airway (smoking, asthma, respiratory tract infections), impaired respiratory, cardiovascular or neurological function, hypothermia and electrolyte and acid base abnormalities. A structured approach to extubation and the decision-making process has been published by The Difficult Airway Society. The algorithms are available at https://www.das.uk.com/content/das-extubation-guidelines.

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EFFECT OF GENERAL ANAESTHESIA ON THE AIRWAY AND UPPER ALIMENTARY CANAL

CYPRIAN MENDONCA

Most of the trouble we experience, such as coughing, biting, breath-holding, laryngospasm and regurgitation, occur during light planes of anaesthesia, usually during induction or emergence.

OROPHARYNGEAL AND GLOTTIC STRUCTURES

Induction of anaesthesia usually causes obstruction of the upper airway. Alterations in the tone of the

skeletal muscles of the pharynx and neck are thought to be responsible. Radiographic and MR studies have shown that, at induction, the most important cause of obstruction is approximation of the soft palate to the posterior pharyngeal wall.

Increasing depth of propofol anaesthesia is associated with increased collapsibility of the upper airway, which is associated with profound inhibition of genioglossus muscle activity. This dose-related inhibition seems to be the combined result of depression of central respiratory output to upper airway dilator muscles and of upper airway reflexes. The standard manoeuvres employed to clear an obstructed airway – head tilt, chin lift and jaw thrust – stretch the anterior neck tissues, which lifts the glottic opening from the posterior pharyngeal wall.

RECOGNITION OF AN OBSTRUCTED AIRWAY

Patients with a tendency to upper airway obstruction during sleep are vulnerable during anaesthesia and sedation. These include obesity, maxillary hypoplasia, mandibular retrusion, bulbar muscle weakness and specific obstructive lesions such as nasal obstruction and adenotonsillar hypertrophy. Such abnormalities also make intubation difficult. Preoperative identification of at-risk patients and generation of an appropriate management plan are key to enhancing patient safety.

A spontaneously breathing patient will generate large negative intrathoracic pressures, which will cause:

- Noisy inspiration, due to turbulent gas flow (a completely obstructed airway is silent).
- Signs of respiratory distress including tracheal tug and intercostal recession.
- Paradoxical respiratory movements.
- Negative pressure pulmonary oedema can develop if the obstruction continues.

A ventilated patient will have high inflation pressures.

PULMONARY OEDEMA FOLLOWING RELIEF OF AIRWAY OBSTRUCTION

Negative pressure pulmonary oedema occurs as a result of generation of a large negative intra-thoracic

pressure in patients with upper airway obstruction. It is a type of noncardiogenic pulmonary oedema and often occurs in otherwise young, fit and healthy patients. Negative pressure pulmonary oedema typically occurs at the end of anaesthesia when a patient bites and occludes the laryngeal mask or endotracheal tube. The underlying pathophysiology is incompletely understood; however, it is believed that the generation of a large negative transpulmonary pressure gradient causes fluid to collect in the alveoli resulting in pulmonary oedema. In the UK, the 4th National Audit Project identified 13 patients who experienced postoperative pulmonary oedema as a result of airway obstruction. Twelve made a full recovery, and one suffered a fatal cardiac arrest.

The clinical features may include pink frothy sputum, desaturation, increased respiratory rate, audible wheeze and crackles.

Negative pressure pulmonary oedema is an anaesthetic emergency. Management includes:

- Sitting the patient upright.
- Administration of high concentrations of oxygen.
- Application of PEEP/CPAP.
- Positive pressure ventilation may be required and an ARDS-like clinical picture may result.
- There is limited evidence for the use of diuretic.

LARYNGOSPASM

Glottic closure is a normal physiological mechanism to protect the airway from aspiration. Laryngospasm can be defined as sustained closure of the vocal cords resulting in complete or partial airway obstruction. It is estimated that laryngospasm occurs in roughly 1% of anaesthetics. It commonly occurs during patient stimulation in light planes of anaesthesia, for example, during airway manipulation during induction/emergence. Laryngospasm is an anaesthetic emergency. Seeking help and clear communication with all team members is imperative for successful management.

Management includes:

- Cessation of any stimulation
- Increasing inspired oxygen concentration
- Increasing the depth of anaesthesia
  - Administration of propofol

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- Increasing the inspired concentration of volatile anaesthetic (consider switching volatile anaesthetic to sevoflourane)
- Application of CPAP/PEEP
- Administration of neuromuscular blocking drug such as suxamethonium

Although there is limited evidence, Larson's manoeuvre (bilateral firm digital pressure over the styloid processes posterior to the rami of the mandible) can be attempted.

OESOPHAGEAL FUNCTION AND ANAESTHESIA

THE LOWER OESOPHAGEAL SPHINCTER

The intraluminal pressure at the gastro-oesophageal junction is 15–25 mmHg above gastric pressure, which normally prevents gastro-oesophageal reflux. The pressure is produced by smooth muscle cells of the lower oesophageal sphincter. Contraction of the surrounding skeletal muscle of the diaphragmatic crura increases the intraluminal pressure during inspiration, and also during straining. Straining does not cause gastro-oesophageal reflux in normal conscious patients. Reflux does not occur spontaneously during anaesthesia, but diaphragmatic tone decreases and thus its protective effect may be lost. Reflux is associated with hiccup, straining, deep inspiration with surgical stimulus and bucking on the tracheal tube, all features of light anaesthesia. The effect of various drugs on the lower oesophageal sphincter tone is described in Table 26.2.

THE OESOPHAGUS

The oesophagus is a muscular tube about 25 cm in length, which begins at the caudal border of the cricoid cartilage and ends at the cardiac orifice of the stomach, usually about 1.5 cm below the diaphragm. The upper quarter is composed of skeletal muscle only, the lower third is smooth muscle only and the middle is a mixture of the two types. The oesophagus can contain large volumes of fluid (up to 200 mL). Refluxed gastric contents are cleared by oesophageal peristalsis, which is initiated by swallowing or local reflexes.

Table 26.2 The effect of drugs on the lower oesophageal sphincter tone

Drugs increasing the tone	Drugs reducing the tone
Anti-emetics	Inhalational anaesthetics
Domperidone, metoclopramide, cyclizine	Desflurane, isoflurane
Neuromuscular blocking drugs	Intravenous induction agents
Suxamethonium, pancuronium, vecuronium	Propofol, thiopentone
Beta-blockers	Opioids
Metoprolol	Morphine, pethidine
	Calcium channel blockers
	Verapamil
	Cholinergics
	Atropine

Both general anaesthesia and intravenous atropine inhibit oesophageal motility. Oesophageal clearance may not occur during general anaesthesia. The refluxed contents will remain in the oesophagus, increasing the risk of regurgitation into the pharynx, until swallowing recommences as the patient awakes.

THE UPPER OESOPHAGEAL SPHINCTER

The upper oesophageal sphincter is formed by the lamina of the cricoid cartilage anteriorly and the striated muscle cricopharyngeus posteriorly. Resting upper oesophageal sphincter pressure is about 40 mmHg. Relaxation of the upper oesophageal sphincter at induction of anaesthesia can precipitate regurgitation.

Both intravenous thiopentone and suxamethonium decrease upper oesophageal sphincter pressure to less than 10 mmHg, a pressure low enough to allow regurgitation of oesophageal contents. Intravenous induction with ketamine maintains upper oesophageal sphincter pressure, in the absence of neuromuscular blockade. Upper oesophageal sphincter pressure may rise to over 100 mmHg during coughing and straining under light anaesthesia, and prevent regurgitation.

Intravenous benzodiazepines, such as midazolam, reduce upper sphincter pressure. They also depress laryngeal reflexes. Heavy sedation may allow aspiration.

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HYPOXAEMIA UNDER ANAESTHESIA

WILLIAM TOSH

Hypoxaemia is defined as a decrease in the partial pressure of oxygen in blood and is considered to be severe when oxygen saturation falls below 90%. Acute hypoxaemia will eventually cause circulatory arrest due to myocardial hypoxia and at some point around the time of arrest, irreversible cardiac damage occurs. Following cardiac arrest, consciousness is lost within 10 seconds and irreversible brain damage can occur within 4–5 minutes. The period of anoxia necessary to produce circulatory arrest will depend on cardiac health, the oxygen content of the body prior to the anoxic episode and oxygen consumption.

The oxygen content of the blood depends on oxygen saturation and haemoglobin in the blood and can be calculated from the following equation:

$$\text{Arterial oxygen content} = (Hb \times 1.36 \times SaO_2) + (0.003 \times PaO_2)$$

where Hb is the haemoglobin, SaO_2 is the percentage of haemoglobin saturated with oxygen and PaO_2 is the partial pressure of arterial oxygen in mmHg.

The oxygen delivery (oxygen flux) to the tissues is calculated by multiplying cardiac output (CO) and arterial oxygen content (CaO_2) of the blood.

$$\text{Oxygen flux} = (HR \times SV) \times [(Hb \times 1.36 \times SaO_2) + (0.003 \times PaO_2)]$$

The term hypoxia defines deficiency of oxygen at a tissue level. There are several types of hypoxia including anaemic, hypoxaemic, stagnant and tissue. Hypoxaemic hypoxia is due to reduced oxygen saturation in arterial blood.

PERIOPERATIVE CAUSES OF HYPOXAEMIA

A systematic approach to the management of hypoxia under anaesthesia is essential. The first response to falling oxygen saturations is to increase the inspired oxygen (FiO_2) to 100%. Following this, a systematic, stepwise approach to the underlying cause should begin.

EQUIPMENT FAILURE

A rapid check of all equipment should be performed. In particular, the patency, correct connections of the anaesthetic circuits and artificial airways should be confirmed. Commencing hand ventilation may provide vital information on compliance of the lung and factors associated with the anaesthetic machine. It may be necessary to suction down artificial airways to remove secretions and ensure patency. Confirming the pulse oximeter probe position and a good quality waveform may help to rule out erroneous readings.

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LOW INSPIRED OXYGEN CONCENTRATION

This can be due to an accidental decrease in FiO_2 due to equipment failure, misconnections or low flow anaesthesia.

HYPOVENTILATION

Hypoventilation results from central or peripheral depression of ventilation, and/or an obstructed airway. The effect of hypoventilation on oxygen saturation is complex and depends on the inspired oxygen concentration. When a patient with normal lungs breathes 21% oxygen, PaO_2 does not fall below 100 mmHg (13.2 kPa) until alveolar ventilation is approaching 3 L/min. As inspired oxygen concentration is raised, the level of alveolar ventilation required to maintain PaO_2 above 100 mmHg falls. With an oxygen concentration in excess of 40% alveolar ventilation of 1 L/min will normally maintain PaO_2 at greater than 100 mmHg. For the exact relationship between alveolar ventilation and alveolar gas tensions, the reader is referred to texts on respiratory physiology.

APNOEIC OXYGENATION

During apnoea the alveoli will continue to take up oxygen in the absence of respiratory effort. Approximately 250 mL min^{-1} of oxygen is removed from the alveoli into the blood down a concentration gradient. Conversely, 20 mL min^{-1} of CO_2 is transferred from the blood to the alveoli. Thus, there is a net loss of approximately 230 mL volume of gas from the alveoli. This loss of volume generates a sub-atmospheric pressure within the alveoli during periods of apnoea. If a high concentration of oxygen is delivered to the upper airway during periods of apnoea, oxygen will be drawn down the airway into the alveoli by means of concentration and pressure gradient provided the airway is at least partially open.

Apnoeic oxygenation can prevent desaturation during lengthy periods of apnoea, since the $PaCO_2$ only rises by about 0.5 kPa min^{-1}. During prolonged laryngoscopic attempts, high flow nasal oxygenation (15 L min^{-1}) can be used to prevent desaturation.

SHUNT

The term 'shunt' is used here to mean failure of oxygenation of blood during passage through the pulmonary circulation ('venous admixture'). In most cases, this is due to ventilation/perfusion mismatch. The normal shunt fraction is about 2% and is due to the bronchial and thebesian veins draining directly into the left atrium without being oxygenated. Anatomic shunts are caused due to right to left intracardiac shunts (TOF or reverse flow through ASD and VSD) and intrapulmonary fistulae (connection between branches of pulmonary artery and vein). The other causes of shunt include general anaesthesia, intermittent positive pressure ventilation (IPPV), bronchial intubation, aspiration, oesophageal intubation and pulmonary oedema.

LOW CARDIAC OUTPUT AND HYPOXIA

A fall in cardiac output will decrease oxygen delivery to the tissues. Consequently, in order to meet tissue and cellular demands for oxygen, the amount of oxygen extracted from the blood by the tissues increases. In a healthy subject, the normal oxygen extraction ratio (O_2ER) is about 24%. O_2ER can be calculated from the following equation:

$$O_2ER = VO_2/DO_2 \times 100$$

where VO_2 = oxygen uptake and DO_2 = oxygen delivery.

It is evident from the formula that a reduction in oxygen delivery will result in an increase in the O_2ER.

An increase in oxygen extraction will produce a reduction in mixed venous oxygen saturation. As the primary cause of the hypoxia is cardiovascular, increasing the FiO_2 may be of limited benefit. Tissue oxygenation can be improved by increasing cardiac output.

Reductions in cardiac output may result in areas of ventilated lung being under-perfused leading to increased V/Q mismatch. Increasing cardiac output may improve ventilation/perfusion profiles. It is therefore necessary to ensure that cardiac output is adequate, particularly when the patient has pulmonary pathology. At higher levels of shunt, any drop

in cardiac output may result in a significant fall in saturation and tissue oxygen delivery.

DEAD SPACE

Dead space is that part of inspired air that fails to take part in gas exchange. The volume of conducting airways leading up to the alveoli constitutes the anatomical dead space. The part of the alveolar air that does not take part in gas exchange accounts for alveolar dead space. The sum of the anatomical and alveolar dead space equates to the physiological dead space. The alveolar dead space increases in pulmonary embolism and in conditions with reduced cardiac output.

HYPOXIA DURING AIRWAY MANAGEMENT

FAILED AIRWAY MANAGEMENT

Maintenance of oxygenation and ventilation when failed intubation has occurred is fundamental to patient safety. Repeated failed attempts at intubation are associated with increased patient morbidity and mortality. Further, facemask ventilation may become more difficult, due to increasing soft tissue swelling, trauma and bleeding within the airway. When unexpected difficult intubation arises, the priority is to maintain oxygenation and to limit the number of airway interventions.

BRONCHIAL INTUBATION

Bronchial intubation is usually suspected when desaturation occurs following a successful tracheal intubation. This is confirmed by unilateral chest movement and unilateral breath sounds. Fibre-optic bronchoscopy can confirm the placement of tracheal tube in the trachea or main bronchus. Once confirmed, the tube should be gradually withdrawn under direct vision and reassessed. Failure to recognise and correct an endobronchial intubation may lead to hypoxaemia, lung or lobar collapse and barotrauma.

OESOPHAGEAL INTUBATION

Oesophageal intubation if unrecognised is a cause of significant morbidity, hypoxic brain injury and death. Following intubation, the correct placement of the tube can be confirmed by visualisation of it passing through the vocal cords, bilateral chest wall movement, bilateral breath sounds, presence of a sustained capnography trace and use of ultrasound to confirm bilateral ventilation of the lungs. In addition, a flexible fibrescope can be used to check the correct placement of the tube in the trachea.

UPPER AIRWAY OBSTRUCTION

Upper airway obstruction is one of the common causes of hypoxaemia under anaesthesia. Reasons include artificial airway kinking, failure to maintain the upper airway or laryngospasm. Postoperative nocturnal airway obstruction in patients with obstructive sleep apnoea is an increasing problem, due to the prevalence of obesity. Opiate drugs will increase the tendency to obstruction, both whilst they are administered and for some nights after cessation of treatment, because of a rebound increase in REM sleep. Patients known to suffer obstructive sleep apnoea should be nursed in HDU whilst suspected cases and obese patients should receive nocturnal oxygen.

ASPIRATION

Aspiration can be potentially fatal. It can cause acute desaturation and hence hypoxaemia. It may require admission to ICU.

PREOXYGENATION

Adequate preoxygenation is essential and helps to prevent desaturation during induction of anaesthesia. The Farmery and Roe model predicts that the SaO_2 will decline to 60% after 9.9 minutes if the subject was breathing 100% oxygen before the apnoea, and in 2.8 minutes after breathing air.

The primary purpose of preoxygenation is to extend the safe apnoea time (the time taken for critical arterial desaturation to occur following the cessation of breathing). Factors influencing safe apnoea time include FRC, preoxygenation, maintenance of a patent airway, metabolic rate, physiological shunt and dead space. The FRC is the most important store of oxygen in the body.

Preoxygenation aims to de-nitrogenate the FRC. In a healthy adult, following effective preoxygenation, the lungs would contain about 2000 mL of oxygen. During apnoea, the FRC serves as a store for oxygen and the greater the FRC, the longer the period of apnoea which can be tolerated. Patients with a reduced FRC such as those with underlying lung disease, pregnancy or obesity will desaturate and reach critical hypoxaemia more rapidly. In this patient population, adequate preoxygenation is essential.

Techniques commonly employed involve asking the patient to breathe 100% oxygen through a tight-fitting facemask for 3 minutes, asking the patient to take 8 vital capacity breathes in 1 minute or 4 vital capacity breaths in 30 seconds. The adequacy of preoxygenation can be assessed by the end-tidal oxygen concentration. Patients should be preoxygenated until the end-tidal oxygen concentration is >80%.

Preoxygenation using a 20°–25° head-up position, continuous positive airway pressure and humidified high flow nasal oxygenation can further extend the duration of apnoea and is particularly useful in high-risk patients.

OBESITY AND HYPOXAEMIA

An obese patient has decreased vital capacity, expiratory reserve volume, inspiratory capacity and FRC. The supine position further decreases expiratory reserve volume and FRC due to small airways collapse, cephalad displacement of diaphragm and increased thoracic blood volume. FRC declines steeply with increasing BMI and reaches values of around 1 L or less in subjects whose BMI exceeds 40 kg m^{-2}. The time to develop hypoxaemia is significantly shorter in obese patients. In addition, there are several other problems during airway management, such as difficult mask ventilation, difficult tracheal intubation and increased risk of aspiration. For morbidly obese patients (BMI 40 kg m^{-2}), preoxygenation in the head-up position increases FRC and achieves better oxygenation.

With increasing BMI, closing volume can encroach on FRC during normal tidal ventilation, leading to airway closure and V/Q mismatch. A modest preoperative (A–a) O_2 gradient and shunt fraction can deteriorate markedly on induction of anaesthesia requiring high FiO_2 and PEEP to maintain an adequate arterial PO_2.

The incidence of obstructive sleep apnoea increases with obesity and increasing age. Depressant drugs, including many anaesthetic agents and analgesics, accentuate this. The combination of reduced chest wall and diaphragmatic tone during general anaesthesia, the increased incidence of atelectasis and secretion retention render the morbidly obese patient at risk of rapid desaturation during periods of hypoventilation or apnoea. These problems persist into the postoperative period. In addition to supplemental oxygen, a multimodal approach, involving breathing exercises, physiotherapy, and in some cases continuous positive airway pressure (CPAP), may be necessary in the immediate postoperative period.

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PAEDIATRIC AIRWAY

BENJAMIN ROBINSON
AND CAROL L BRADBURY

ANATOMY AND PHYSIOLOGY

The paediatric airway is smaller and anatomically distinct from that of the adult. Infants are obligatory nasal breathers with narrow nasal passages and relatively large tongue. An adult intubation position of 'sniffing the morning air' may therefore be unhelpful due to a large head and prominent occiput.

The infant larynx is more anterior and cephalad; the epiglottis long, floppy and U-shaped in cross-section. Intubation using a straight-bladed laryngoscope, directly lifting the epiglottis, may result in better visualisation of the vocal cords. Intubation with a curved blade is common in older children as the larynx reverts towards its adult anatomy.

The narrowest part of the conical shaped paediatric larynx is the cricoid cartilage which is circular in cross-section. This differs from the adult where the vocal cords themselves are the narrowest cross-section, and becomes important when placing endotracheal tubes. Uncuffed endotracheal tubes can be placed to form an effective seal at the cricoid level.

Physiologically the neonate/infant has an immature respiratory centre (increased risk of apnoea), stiffer lungs (lower airway compliance), higher closing volume and increased metabolism and oxygen consumption, which together result in a small oxygen reservoir and rapid onset of hypoxia and desaturation.

As a result of the smaller paediatric airways, partial obstruction caused by mucosal oedema is common and can be caused by tracheal intubation and worsened by any pre-existing medical conditions such as upper respiratory tract infection.

PREOPERATIVE ASSESSMENT

Difficult paediatric airways are uncommon, but can often be predicted by conducting a thorough history and clinical examination. Points pertinent to the paediatric patient include:

- Presence of coryzal or other symptoms of upper respiratory tract infection.
- Presence of symptoms of obstructive sleep apnoea (snoring, restless sleep, apnoea) and tonsillar enlargement.
- Examination of the head and neck looking for dysmorphic features, micrognathia and cleft lip or palate. The Mallampati test can be impractical in young children.

MANAGEMENT OF THE PAEDIATRIC AIRWAY

Preparation is vital. Children have smaller oxygen reserves and desaturate faster than adults. Unexpected difficulty can be encountered in the routine induction of general anaesthesia, during mask ventilation or intubation. A predetermined plan is recommended and must be clearly communicated with the relevant personnel to ensure all airway equipment can be available when requested.

Excellent algorithms have been devised by the Difficult Airway Society (DAS) and Association of Paediatric Anaesthetists of Great Britain and Ireland (APA) to help aid decision making in an unanticipated difficulty in mask ventilation or tracheal intubation during the induction of anaesthesia in children aged 1 to 8 years. These are available online: http://www.das.uk.com/guidelines/paediatric-difficult-airway-guidelines.

INDUCTION

During induction, children are predisposed to upper airway obstruction. This is usually overcome by simple airway manoeuvres:

- Avoiding extension at the atlanto-occipital joint in infants.
- Head tilt chin lift in an older child.
- No pillow for small children and consider a pad under the shoulders for infants.
- Avoid compressing the soft tissues under the jaw as this may push the tongue base upwards and backwards, further obstructing the airway (exert pressure on the bony mandible only).

ORAL OR NASOPHARYNGEAL AIRWAYS

- Oral airways may cause laryngospasm or retching.
- Estimate size by comparing to child's face, should extend from lips to the angle of the jaw.
- Nasal airways are better tolerated; the correct diameter is the same as that for an orotracheal tube.

CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

CPAP is useful as it provides a pneumatic splint, helping to distend the structures of the oropharynx and minimise upper airway obstruction. It also helps to prevent distal airway collapse. It is best provided with a T-piece circuit and partial obstruction of the reservoir bag outlet.

LARYNGEAL MASK AIRWAY

Laryngeal mask (LM) insertion can be more difficult than in adults due to the acute angle between the palate and posterior pharynx in children. Simple variations in technique such as inserting the device upside down initially and then rotating it, partially inflating the device before insertion or guiding the LM around the posterior pharynx with a finger can help.

LM and other supraglottic airway devices are available in a range of sizes according to the child's weight (Table 26.3).

TRACHEAL INTUBATION

- Intubation of a neonate or infant is easiest with a straight blade laryngoscope.

- The laryngoscope blade can be slid underneath the epiglottis and the tip lifted so the glottis is exposed.
- Alternatively, the blade can be deliberately advanced into the oesophagus and then, with the tip lifted anteriorly, withdrawn slowly to bring the larynx into view.
- In order to avoid damage to the cricoid region, it is essential to choose a tube of the correct size (Table 26.4).

There are several formulae to calculate the length of the endotracheal tube from the mid-trachea to the incisors:

- An additional 2–3 cm is added for nasal intubations.
- Whichever method is used, tube position must be checked clinically.
- Endotracheal tubes must be carefully secured with adhesive tape or tied.

THE DIFFICULT PAEDIATRIC AIRWAY

Preparation is vital when a difficult airway is anticipated. Children desaturate faster than adults, so all potentially needed equipment should be immediately available for use. Backup plans should be determined before the child is induced and all personnel need to be clear on the plans.

It is advisable to keep the child spontaneously breathing and this is easiest to do with an inhalational induction. The use of 100% oxygen should be considered as the carrier gas. Monitoring and intravenous access should be acquired as soon as is practically possible. Before any attempt at intubation takes place, the level of anaesthesia must be allowed to deepen. This may take a while if partial airway obstruction is apparent. Simple airway manoeuvers and adjuncts, including LM insertion, may facilitate deepening of anaesthesia but this depends upon the site of the obstruction.

Table 26.3 Supraglotiic airway devices and their smallest size available

	Device	Smallest size available	Weight
First generation devices	LMA Classic	1	Up to 5 kg
	LMA Flexible	2	10–20 kg
Second generation devices	I-Gel	1	2–5 kg
	LMA Supreme	1	Up to 5 kg

Table 26.4 Paediatric endotracheal tube sizes

Age	Tube size (internal diameter in mm)	Depth of placement of endotracheal tube (to lips in cm)
Newborn	3.0 (premature babies may require a smaller tube size)	7-8-9 rule can be applied: 7 cm if 1 kg 8 cm if 2 kg 9 cm if 3 kg
6 months	3.5	11
1 year	4.0	12
Over 1 year	(Age/4) + 4	(Age/2) + 12 or (Weight/5) + 12

The exact technique chosen will vary with respect to the individual case. Many children will be intubated with direct laryngoscopy. Careful attention should be paid to optimise the child's position prior to attempted intubation. Simple measures such as application of a BURP manoeuvre (Backwards Upwards Rightwards Pressure on the Cricoid), use of a gum elastic bougie or a paediatric McCoy blade may assist intubation.

Videolaryngoscopes and fibre-optic intubation are useful advanced techniques. Fibre-optic intubation may proceed either nasally or orally. The nasal route allows the larynx to be approached at a less acute angle but risks trauma to the nasal mucosa.

Once surgery is complete, preparation for extubation should be as rigorous as for intubation. All the equipment that was made available for intubation should remain present for extubation. Any throat pack or debris that has been acquired during surgery should be carefully removed and generally the child should be fully reversed and wide awake for extubation.

Documentation of techniques used and any difficulties encountered is important. It can greatly assist the future airway management of a child with a known difficult airway.

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THE ANAESTHETIC MACHINE

MICHELE PENNIMPEDE
AND BAHA AL-SHAIKH

The anaesthetic machine provides an accurate and continuous flow of anaesthetic gases and vapours.

HISTORY AND EVOLUTION

After the introduction of O_2 and N_2O in the form of compressed gases in cylinders, there was a necessity for mounting these cylinders on a metal frame. This stimulated many people to attempt to construct the anaesthetic machine. In 1917, HEG Boyle modified Gwathmey's machine and this became popular as the Boyle anaesthetic machine.

Some of the milestones in the development of the anaesthetic machine are as follows:

1921 – Waters to and fro absorption apparatus was introduced.
1927 – Flow meter for CO_2 was included, the volatile controls were of the lever type and the familiar back bar made its first appearance.
1930 – The plunger of the vaporiser appeared in the 1930 model.
1930 – Circle absorption system was introduced by Brian Sword.
1933 – Dry bobbin flow meters were introduced.
1952 – Pin index safety system (PISS) by Woodbridge.
1958 – Introduction of Bodok seal.

Accurate delivery of gases is achieved by controlling the way in which the supplies of oxygen, medical air and nitrous oxide are mixed before the selected

Provides O_2.
Accurately mixes anaesthetic gases and vapours.
Enables patient ventilation.
Minimises anaesthesia-related risks to patients and
staff.

concentration of anaesthetic agent is added to it prior to being delivered to the patient. The development of non-flammable volatile agents has allowed subsequent integration of electronics into the anaesthetic machine allowing the development of the integrated 'Anaesthetic Workstation' (AWS). The AWS may have an electronically controlled ventilator as well as integral output monitoring devices and associated alarms. The AWS, typically, incorporates integrated patient monitoring and therefore provides a sophisticated comprehensive platform for the safe administration of anaesthesia. It is important to obtain specific training before using an unfamiliar anaesthetic machine or AWS.

A backup system to maintain anaesthesia (e.g. IV anaesthetic agent) as well as an independent means of ventilation with oxygen (self-inflating bag) must always be present in case of unexpected problems.

Electrically powered workstations should always be plugged directly into a wall-mounted mains socket, and never via a multisocket extension lead. Most include an integral battery that provides power in the event of electrical supply failure.

Modern machines comprise several basic elements:

PIPELINES

- Colour-coded hoses have Schraeder male probes at the distal end to insert into the piped gas supply. They are permanently connected to the machine via a *non-interchangeable screw-thread* (NIST). Pipeline gases are supplied at a working pressure of 400 kPa.
- Backflow check valves on the anaesthetic machine prevent retrograde gas leaks from the pipelines when these are not plugged into a gas terminal outlet.

CYLINDERS

- Cylinders are colour-coded and attached using yokes and wing nuts and a pin index system to prevent incorrect attachments.
- Cylinders are connected at their respective yokes via metal-edged rubber bonded disks, Bodok seals, to ensure a gas-tight seal. Cylinder valves should be opened slowly and closed without using undue force. Oil or grease should never be used, as combustion may occur.
- Cylinders can be used either as the primary gas supply, or as a reserve in the event of pipeline failure. If both sources are connected, gas from the pipeline supply is used preferentially.
- Non-return valves on the anaesthetic machine prevent gases escaping from the empty cylinder yoke when a cylinder is empty or is changed. However, the efficiency of these valves may deteriorate with time and if a cylinder yoke is to be left empty, blanking caps should be used to prevent back pressure leaks that could potentially alter the gas mixture delivered.

PRESSURE GAUGES

- Aneroid 'Bourdon gauges' monitor the gas pressures in the cylinders and pipelines. Each consists of a flexible tube which straightens when exposed to a gas pressure causing a gear mechanism to move a needle pointer. They are usually mounted in a colour-coded front facing panel on the anaesthetic machine, displayed as kPa × 100.
- Calibrated for individual gases or vapours, the pressure indicates the contents available except for N_2O as the cylinder contains both liquid and vapour.

PRESSURE REGULATORS

- Reduce the high and variable cylinder pressures to a safe constant working pressure and provide protection against pressure surges. Modern machines may have several regulators for each gas.

- Primary regulators are used to reduce high cylinder pressures to machine working pressures, often just below the pipeline pressure of 400 kPa (so that pipeline gas is used preferentially).
- Secondary regulators (flow restrictors) lie downstream of the primary regulators and act to further smooth out supply pressure from emptying cylinders and fluctuations in pipeline gas pressure.
- There are usually relief valves downstream of the regulators to allow escape of gas should the regulators fail.

FLOW CONTROL VALVES

- Manually operated fine-adjustment needle valves control the flow rates of gases.
- The oxygen control knob is larger than the others with a distinctive shape to avoid confusion, and is always on the left in the UK.
- Standards ensure that they are accurate, that the torque required to operate them is high enough to prevent accidental readjustment and that minimal variation in gas flow can occur as a result of axial forces on the valve spindle.
- They must prevent the supply of a hypoxic mixture, or trigger an alarm integrated into the anaesthetic machine if such a mixture is delivered. Measures to prevent the delivery of hypoxic mixtures often take the form of mechanical linkages between the oxygen and other control valves. Alternate approaches include pneumatic linkages or electronic controls.
- Some AWS use electronically controlled proportional flow valves instead.

FLOW METERS

- Flow meters, usually rotameters, enable flows of individual gases to be visualized and measured. Electronic versions also exist.
- Rotameters consist of tapered tubes with bobbins. Gas entering the tube pushes the bobbin up causing it to float and rotate within. The flow is measured at the top of the bobbin.
- Rotameter tubes are individually calibrated for their gases at 20°C and atmospheric pressure. They are non-interchangeable, using differing diameters, lengths or fittings. Stops at either end of the tube ensure that the bobbin is always visible. The glass is antistatic to prevent the bobbin sticking. They are positioned vertically to ensure accuracy.
- The bobbin has angled slots in its top flange which cause it to rotate. The resulting spin helps stabilize the bobbin in the centre of the gas flow and prevents sticking.
- The flowmeter block is arranged such that oxygen is the last gas to be added to the mixture, so even if there is a leak in the block a hypoxic mixture is not delivered to the patient.

VAPORISERS

- Plenum vaporizers have a vaporizing chamber and bypass channel, the control knob alters the 'splitting ratio', thereby determining the percentage of carrier gas that becomes fully saturated with the volatile anaesthetic. Vaporizers are calibrated for individual agents, and are compensated for temperature and flow rate changes.
- Vaporizers are mounted on the machine back-bar using a mechanism that prevents more than one vaporizer being active at any one time. Leaks may occur when the vaporizer is not seated properly, and this must be checked each time a vaporizer is replaced.
- Electronic vaporizers (using the fuel injection principle) are controlled from an electronic panel and do not have a control knob.

PRESSURE RELIEF VALVE

- This protects the machine from high pressure in the event of obstruction at the common gas outlet or breathing system. It typically opens at pressures exceeding 35 kPa.

OXYGEN FAILURE

- An audible oxygen supply failure alarm powered by the oxygen supply pressure alone must be present. The alarm usually sounds when the oxygen supply pressure falls below 200 kPa (half normal pressure) with an alarm lasting at least 7 seconds.
- Secondary alarms may operate to give a more persistent warning, e.g. a siren driven by nitrous oxide being vented.
- Some designs ensure that gases containing less than 21% oxygen are cut off in the event of oxygen supply failure. Others open the breathing system to the atmosphere.

OXYGEN FLUSH

- When pressed it provides pure oxygen at 35–75 L/min bypassing vaporizers.
- It is normally non-locking, but may have the facility to be locked on (risk of awareness/barotrauma if unnoticed).

COMMON GAS OUTLET

This is the final pathway for gases to leave the anaesthetic machine with a male 22 mm taper and a female 15 mm taper.

ANAESTHETIC GAS SCAVENGING SYSTEM

An active system using assisted flow is commonly used. It is frequently integrated into the modern anaesthetic machine.

COMPRESSED OXYGEN OUTLETS

One or more compressed oxygen outlets allow oxygen at pipeline pressure (400 kPa) to be utilised for various functions such as driving ventilators, or for use with a Sanders type jet ventilator device.

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Breathing circuits, Chapter 27

BREATHING SYSTEMS

JANINE MA THOMAS AND BAHA AL-SHAIKH

Breathing systems deliver anaesthetic gas mixtures to the patient. Several classifications have been described (Table 27.1) whether they allow rebreathing, and whether they have an 'adequate' or 'inadequate' fresh gas flow to meet the patients minute volume, e.g. the Mapleson systems (adequate) or the circle system (inadequate).

The most common classification used is the Mapleson nomenclature (Table 27.2). His classification of five systems has since been expanded to six systems labelled A to F, arranged in decreasing levels of adequacy of ventilation.

Table 27.1 A classification of breathing systems

	Definition	Examples
Open	Entirely open to the atmosphere, ambient air is the fresh gas supply	Rag and bottle
Semi-open	Mainly open to atmosphere with some restriction to ambient air which acts as the fresh gas supply	Schimmelbusch mask
Semi-closed	Air intake prevented but venting of excess gas to the atmosphere	Mapelson systems
Closed	No air intake or venting of exhaust gases	Totally closed circle system

SYSTEMS WITH INADEQUATE FGF

Systems that allow the use of 'inadequate FGF' reduce the amount of volatile anaesthetic agent required, minimising costs and pollution. They also allow for effective warming and humidification of gases. However, the composition of gases at the patient end of a system can be very different from those of the FGF. They should therefore always be used with monitoring of the inspired gases.

For systems using an 'inadequate' gas flow, a method must be used to remove CO_2 which would otherwise build up. This is normally achieved by the use of chemical CO_2 absorption. Several versions are available:

- Soda lime (94% $Ca(OH)_2$, 5% $NaOH$, 1% KOH with silicates to make granules)
- Baralyme (80% $Ca(OH)_2$, 20% $Ba(OH)_2$)
- Amsorb ($CaCl_2$, $Ca(OH)_2$)

Indicator dyes are included which change colour when the absorbent is exhausted. Moisture is required for efficient CO_2 absorption as well as to prevent CO formation (and compound A formation with Sevoflurane). Other substances that can potentially accumulate in such systems include methane, acetone, ethanol and hydrogen. However, they do not generally become clinically significant. CO accumulation and subsequent carboxyhaemoglobin formation is said to occur at less than 0.1% per hour, so may become significant in smokers when ultra-low flows are used. Oxygen flushes of the system (e.g. once an hour) will prevent this.

WATERS' CANISTER (BIDIRECTIONAL FLOW)

- An obsolete system with a cylinder containing soda lime with a reservoir at one end and a facemask, fresh gas inlet and expiratory valve at the other.
- Its usefulness is limited by its cumbersome size, while its efficiency is limited by the rapid exhaustion of soda lime nearest to the patient (results in increasing dead space) and channelling of gas flow.
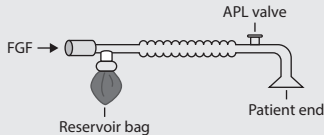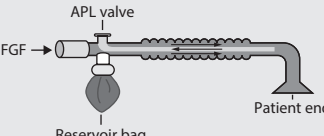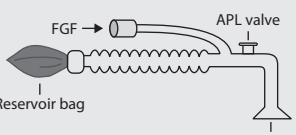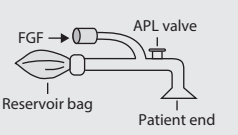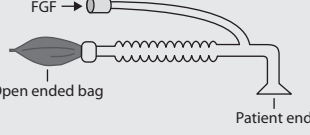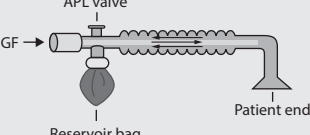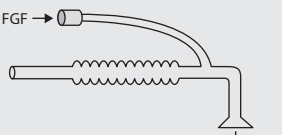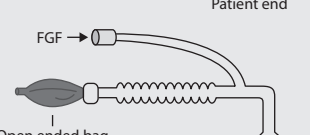
CIRCLE SYSTEM (UNIDIRECTIONAL FLOW)

- The lower the FGF entering the circle, the longer the time constant for the system to reach steady state and the less control there is over the concentrations within the system.

Time constant = Volume of circle/(FGF – uptake).

- In the absence of monitoring, low flow use with the circle system can result in the delivery of hypoxic mixtures. In addition, there may be inadvertent wrong dosage of inhalational agent and rebreathing due to unrecognised exhaustion of CO_2 absorbent.
- Monitoring minimises these risks and is essential with the circle system. Oxygen concentration, volatile agent concentration, capnography and circulating gas volume (e.g. by rising of ventilator bellows) should be monitored.

Table 27.2 Mapleson breathing systems

Name	Alternative names	Diagram	FGF requirements for	
			Spontaneous ventilation	Controlled ventilation
Mapleson A	Magill system	APL valve / FGF → / Reservoir bag / Patient end	1 × MV	3 × MV
	Lack's system	APL valve / FGF → / Reservoir bag / Patient end		
Mapleson B		FGF → / APL valve / Reservoir bag / Patient end	1.5 × MV	1 × MV
Mapleson C	Waters system	FGF → / APL valve / Reservoir bag / Patient end	1.5 × MV	1 × MV
Mapleson D		FGF → / Open ended bag / Patient end	2 × MV	1 × MV
	Bain's system	APL valve / FGF → / Reservoir bag / Patient end		
Mapleson E	Ayre's T-piece	FGF → / Patient end	3 × MV	3 × MV
Mapleson F	Jackson-Rees	FGF → / Open ended bag / Patient end	3 × MV	3 × MV

FGF = fresh gas flow. M.V. = resting minute ventilation of an anaesthetised patient (approximately 70 mL kg min⁻¹).

SYSTEMS WITH ADEQUATE FGF

Non-rebreathing valve systems *(unidirectional flow)* require FGF to match the patient's minute ventilation exactly. Too high a flow may result in the valve jamming in inspiration, causing barotrauma, hypoventilation and falling cardiac output. They are not used.

Reservoir systems *(bidirectional flow)* have a reservoir where bidirectional flow and storage of gases occur (see Figure 27.1). The reservoir portion of the system should be approximately equal to the patient's tidal volume. It may be situated either in the exhaust limb in *efferent* reservoir systems, the supply limb in *afferent* reservoir systems or at the junction of the supply and exhaust limb in *junctional* reservoir systems.

MAPLESON A

Also known as the Magill system, its coaxial variant is known as the Lack system. It is an *afferent* reservoir system.

Advantages
- Maximal efficiency in spontaneous ventilation.
- Good heat and moisture exchange.

Disadvantages
- Very inefficient in controlled ventilation.
- Cannot be used with a ventilator.
- The APL valve at the patient end places weight and bulk near the patient and makes adjustment and scavenging difficult.

MAPLESON B AND C SYSTEMS

Junctional reservoir systems that, while differing in length of tubing to the reservoir, act in an equivalent fashion. The Mapleson C system is also known as a Water's circuit.

Advantages
- Reservoir bag close at hand for monitoring respiration and controlling ventilation with simple interchange between spontaneous and controlled ventilation.

Disadvantages
- Intermediate efficiency in all modes of ventilation.
- APL valve at patient end makes scavenging and adjustment difficult. Weight and bulk near the patient.

MAPLESON D

Its coaxial form is known as the Bain system. It is an *efferent* reservoir system.

Advantages
- Minimal dead space, so may be used for children and adults.
- Simple interchange between spontaneous and controlled ventilation.
- Useful for limited access to head and neck.
- Efficient in controlled ventilation.
- Scavenging convenient.
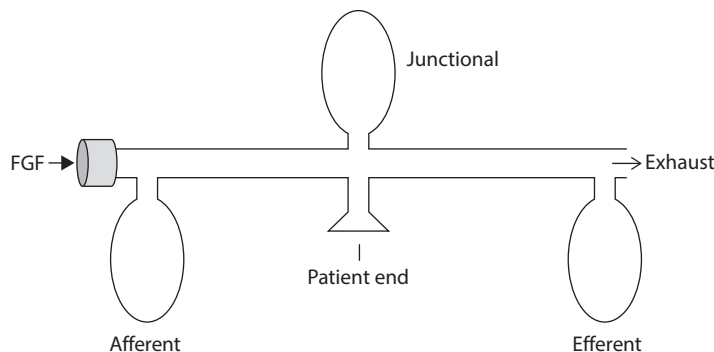
Figure 27.1 The reservoir is where storage of gases occurs. It may be situated in the exhaust limb in efferent reservoir systems (Mapleson D, E, F), the supply limb in afferent reservoir systems (Mapleson A) or at the junction of the supply and exhaust limb in junctional reservoir systems (Mapleson BC).

Disadvantages

- Inefficient in spontaneous ventilation.
- Bain system inner tube disconnection results in a very large dead space.

MAPLESON E

Also known as the Ayre's T-piece. It is an *efferent* reservoir system.

Advantages

- Its low resistance to breathing makes it useful for spontaneous ventilation in small children.
- Minimal dead space, so may be used for children.

Disadvantages

- Inefficient in spontaneous and controlled ventilation.
- Difficult to scavenge.
- Difficult to apply CPAP.

MAPLESON F

Also known as the Jackson Rees modification of the Ayre's T-Piece or the Rees T-piece, it was added to Mapleson's classification by Rees. An open-ended bag is added to the T-piece. Versions with an integrated APL valve and closed reservoir bag are available. It is an *efferent* reservoir system.

Advantages

- Minimal dead space, so may be used for children.
- Reservoir bag close at hand for monitoring respiration and controlling ventilation with simple interchange between spontaneous and controlled ventilation.

Disadvantages

- Scavenging difficult (unless APL valve included).
- Inefficient in spontaneous ventilation.

COMBINATION SYSTEMS

Combination systems attempt to combine the advantages of efferent and afferent reservoir systems. For example, the Humphrey ADE system combines the Mapleson A, D and E systems in one unit with interchange enabled by use of the Humphrey block valve assembly.

ENCLOSED AFFERENT RESEVOIR SYTEM (EAR SYSTEMS)

These systems are similar to the Mapleson D but have an additional reservoir on the FGF limb enclosed within a chamber connected to the main limb (Figure 27.2). Despite their advantages, such systems are not widely used. A further variation – the enclosed efferent afferent reservoir system (EEAR) – has also been described which is more efficient still.
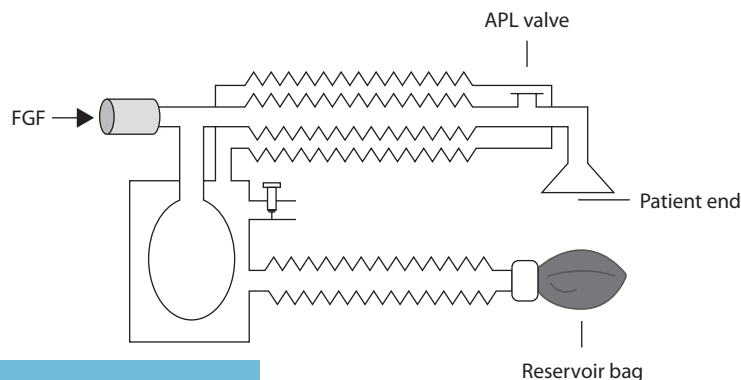
Figure 27.2 Enclosed afferent reservoir (EAR) system. APL, adjustable pressure-limiting; FGF, fresh gas flow.

Advantages

- Efficient for spontaneous and controlled ventilation.
- Convenient interchange between modes of ventilation.
- Satisfactory humidity even without HME.
- Easy scavenging.
- Low dead space so suitable for children.

Disadvantages

- Difficult to understand how it works.
- Internal leak results in large dead space.

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DEPTH OF ANAESTHESIA

JOHN COOMBES AND BAHA AL-SHAIKH

'Depth of anaesthesia' can be represented as a balance between the factors causing hypnosis and the stimulating factors which lighten anaesthesia. Inadequate attention may risk patient awareness due to imbalance of stimulating factors and hypnotic factors.

There is no measure of depth of anaesthesia; however, the following can be used to give an indication.

NON-SPECIFIC

- Clinical signs (PRST, blood *p*ressure, heart *r*ate, *s*weating, *t*ears). These are influenced by other factors such as inadequate analgesia or drugs that affect the autonomic nervous system (e.g. atropine, atenolol).
- End-expiratory volatile agent concentration and comparing to MAC.
- Estimated plasma concentration (Cp_{50}) from target controlled infusion (TCI).

SPECIFIC

- *Non-EEG* – The isolated forearm technique, lower oesophageal contractility, frontalis muscle activity (partly used in entropy, see later), and heart rate/ECG variability.
- *EEG* – Brain activity (measured as EEG) can form the basis of 'depth of anaesthesia' measurement as it is the 'end-organ' for anaesthetic effect.
  - Raw EEG changes with 'depth of anaesthesia', but this requires specialist interpretation and is not easily quantifiable.
  - *Processed EEG* – Early methods looked at spectral analysis. More recently, more complex methods have evolved which use advanced analysis and processing of the EEG.

BISPECTRAL INDEX (BIS)

BIS is the most established of the commercially available monitors where changes to the EEG waveform that occur during anaesthesia are analysed. A mathematical algorithm is used to produce a simplified output variable generated in real time with a range between 0 and 100; '100' represents the fully awake patient and '0' represents cortical electrical silence (Table 27.3). The BIS monitor uses a fronto-temporal electrode array connected to a microprocessor.

Bispectral analysis, a technique used for analysing complex waves, was originally used to analyse complex oceanographic waves in several dimensions; a similar process can be used to analyse the EEG. The algorithms used to compute BIS are available and were derived using EEG data recorded from healthy volunteers undergoing repeated transitions between consciousness and unconsciousness. These were then fitted into a mathematical model to generate a single number or BIS index. BIS has been

Table 27.3 The range of the BIS values in relation to the clinical state

BIS index	Clinical state
100	Awake
>70	Light sedation
60–70	Deep sedation
60	General anaesthesia
40–60	Moderate hypnotic state
40	Deep hypnotic state (burst suppression)
0	No cortical electrical activity

studied widely with patients and healthy volunteers and refined a number of times to enhance accuracy; however, there is contradictory evidence regarding as to whether it reduces rates of awareness.

EEG ENTROPY

Entropy utilizes a measure of the 'regularity' or the amount of disorder of the EEG signal. High levels of entropy during anaesthesia show that the patient is awake, and low levels of entropy correlate with unconsciousness. The EEG signal is recorded using electrodes applied to the forehead and side of the head.

- State entropy (SE) index is calculated from a low frequency range (under 32 Hz) corresponding predominantly to EEG activity.
- Response entropy (RE) index uses a higher frequency range (up to 47 Hz) and includes electromyographic (EMG) activity from the frontalis muscle.
- The concept of Shannon entropy is then applied to normalize the entropy values to between zero (total regularity) and one (total irregularity).

The commercially available M-entropy module (GE Datex-Ohmeda) converts the entropy scales of zero to one into a scale of 0 to 100. The conversion is not exactly linear to give greater resolution at the most important area to monitor.

Both RE and SE are displayed with RE from 100 to 0 and SE from 91 to 0. In practice, 0 corresponds to a very 'deep' level of anaesthesia and values close to 100 correspond to the awake patient. Values between 40 and 60 represent clinically desirable depths of anaesthesia. At this level, the SE and RE indexes should be similar if not identical.

As the patient awakens, an increase in the difference between the SE and RE values is seen due to a diminishing effect of drugs on the CNS and an increasing contribution from frontalis EMG.

LIMITATIONS

- In cerebral atrophy (e.g. dementia) the contribution from the EMG is proportionally increased over the normally dominant EEG signal. This produces a difference in SE and RE values which may not be due to a lightening of anaesthetic depth.
- Ketamine, nitrous oxide and xenon produce different patterns of EEG changes to other anaesthetic drugs. This limits the value of the device.
- Intense surgical stimulation may produce changes in the difference between SE and RE, which could be interpreted as 'inadequate anaesthesia'. In these situations, the anaesthetist must interpret the data in the clinical context to decide whether the patient needs more analgesia or a deepening of anaesthesia.

AUDITORY EVOKED RESPONSE (AER)

AER has been used to assess the integrity of the auditory neuronal pathway. More recently, AER has been incorporated into a commercially available monitor to produce an index from 0–100.

Auditory stimuli (usually clicks at 2 Hz frequency) are applied to the ears via headphones or ear pieces. The EEG signal following each click is recorded from bipolar surface electrodes placed on the centre of the head (vertex) and the temporal lobe (on the mastoid process). It is digitalized and averaged so that the EEG response corresponding to the click emerges from background noise (which cancels out due to its random nature).

The early cortical or mid-latency auditory evoked response shows graded changes with general anaesthesia which has been exploited to measure 'depth of anaesthesia'. The brainstem waves of the AER (prior to 30 ms) appear stable to changes in the level of arousal.

The late cortical waves change dramatically during natural sleep and are not present during anaesthesia.

In the commercially available aePEX device, 7 Hz clicks are used as the stimulus. Like all systems that use EEG as a signal, it is affected by sources of electrical interference.

Factors affecting AER:

- Age
- Conductive and sensorineural hearing disorders
- Brain ischaemia
- Temperature
- Tumours affecting the specific nerve tracts

NAP5 AND NICE GUIDANCE

Inadequate depth of anaesthesia leading to awareness is one of the most common fears of patients undergoing surgery. This was the subject of the 5th National Audit Project (NAP5). The incidence of accidental awareness was found to be around 1:19,000 general anaesthetics but higher in some subspecialties with depth of anaesthesia monitors used in only 2.8%.

NICE recommended the use of EEG-based monitoring of depth of anaesthesia in high-risk patients and when total intravenous anaesthesia is used.

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MONITORING

SANJAY AGRAWAL AND BAHA AL-SHAIKH

For the safe administration of anaesthesia, certain core standards of monitoring should be used. These minimum standards should be uniform irrespective of the duration, location or mode of anaesthesia.

SUMMARY OF AAGBI STANDARDS (REVISED AUGUST 2015)

- The anaesthetist must be present throughout.
- Minimum monitoring devices must be attached before induction of anaesthesia and their use continued until the patient has recovered.
- The same standards apply to local/regional anaesthesia or sedation techniques.
- Information provided by all monitoring devices should be recorded on the anaesthetic record.
- All equipment, including monitoring equipment, must be checked before use. Alarm limits must be set appropriately before use. Audible alarms should be enabled.
- Monitoring devices that are essential ('minimal' monitoring) and which must be immediately available during anaesthesia are described. If it is necessary to continue without an essential monitor this must be recorded, with reasons, in the anaesthetic record.
- Additional monitoring may be necessary.
- Minimal monitoring should be used during the transfer of anaesthetised patients.
- Provision, maintenance, calibration and renewal of equipment are the responsibilities of the institution.
- All patient monitoring equipment should be checked before use in accordance with the AAGBI guideline 'Checking Anaesthetic Equipment'.

EQUIPMENT MONITORING

- The use of an oxygen analyser with an audible alarm is essential during anaesthesia. Appropriate oxygen level alarm limits should

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be set and checked. The analyser must be placed in such a position that the composition of the gas mixture delivered to the patient is monitored continuously.

- During spontaneous ventilation, observation of the reservoir bag may reveal a leak, disconnection, high pressure or abnormalities of ventilation. Continuous $ETCO_2$ monitoring will detect most of these problems. During controlled ventilation, low pressure (disconnection) and high pressure (obstruction) alarms within the breathing systems should be used.
- The end tidal concentration of each inhalational agent should be documented on the anaesthetic record.
- Infusion devices must be checked before use when any drug is administered by infusion with appropriate alarm settings and infusion limits with the intravenous cannula visible throughout the procedure. When using a TIVA technique with neuromuscular blockade, a depth of anaesthesia monitor is recommended.
- Alarms should be set to appropriate values. Audible alarms must be enabled before anaesthesia commences. When IPPV is used, airway pressure alarms must also be used to detect high airway pressure and give warning of disconnection or leaks.
- Care should be taken to configure the display setup, with attention to both the size and arrangement of on-screen data with regular updating of displayed values.

PATIENT MONITORING

- The patient's physiological state and adequacy of anaesthesia require continual assessment. Monitoring devices supplement clinical observation.
- Appropriate clinical observations include mucosal colour, pupil size, response to surgical stimuli and movements of the chest wall and/or the reservoir bag. Palpation of the pulse, auscultation of breath sounds and measurement of urine output and blood loss

may also be included. A stethoscope must always be available.

- SpO_2, NIBP, ECG, inspired and expired O_2, CO_2, N_2O and volatile agent, and airway pressure should all be monitored.
- A peripheral nerve stimulator and means of measuring temperature must also be available.
- Minimal monitoring for recovery includes SpO_2, NIBP, ECG, capnography if the patient has a tracheal tube, supraglottic device in situ or is deeply sedated and temperature.
- Some patients require additional monitoring, e.g. intravascular pressures, cardiac output, biochemical or haematological variables depending on patient and surgical factors. The use of additional monitoring is at the discretion of the anaesthetist.
- Use of depth monitors is recommended when patients are anaesthetised with TIVA and neuromuscular blocking drugs to reduce the risk of accidental awareness.
- Monitoring for regional techniques must include SpO_2, NIBP, ECG to which is added $ETCO_2$ if the patient is sedated.
- SpO_2, ECG and BP are mandatory for transfers. Invasive BP should be considered. $ETCO_2$ must be monitored continuously if the patient has a tracheal tube or supraglottic airway device in situ. Airway pressure, tidal volume and respiratory rate must also be monitored in mechanically ventilated patients.
- The same minimum essential standards of monitoring apply outside the operating theatre as inside.

GAS MONITORING

OXYGEN

- Fast response paramagnetic oxygen analysers are used to measure FiO_2 and FeO_2.
- Persisting FiO_2/FeO_2 difference greater than 5% may be an indicator of hypoventilation or low cardiac output state.
- FeO_2 is useful in monitoring nitrogen and nitrous oxide washout.

F_ECO_2

- Infrared absorption is usually used.
- In healthy adults with normal lungs, $ETCO_2$ is 0.3–0.6 kPa less than arterial CO_2.
- Sudden reduction in F_ECO_2 can be due to disconnection, air embolism or fall in cardiac output.
- F_ECO_2 also underestimates $PaCO_2$ in significant intra- or extra-pulmonary shunting, respiratory rates too high for accurate analysis, tidal volumes too low for accurate sampling and dilution of sample by fresh gas as in the Bain's system.
- Capnography monitors respiratory rate and effectiveness of ventilation.

NITROUS OXIDE AND VOLATILE AGENTS

- Infrared absorption is usually used.
- End tidal volatile agent concentrations will overestimate arterial concentration in the presence of high A-a gradient.

NITROGEN

- A sudden rise in the F_EN_2 in the absence of inspired nitrogen is indicative of air embolism.

RESPIRATORY MECHANICS

Combined pneumotachograph and Pitot tube design improves accuracy when measuring the inspired and expired tidal volume, compliance, airway pressures, volume/pressure and flow/volume loops. Modern devices can be used accurately even in neonates and infants.

BODY TEMPERATURE

Oesophageal, tympanic membrane and nasopharyngeal temperature correlates well with the core temperature.

OXYGEN SATURATION BY PULSE OXIMETRY (SpO$_2$)

Inaccuracy may result from excessive ambient light, hypoperfusion, severe peripheral vasoconstriction, movement artefacts and venous pulsations. Carbon monoxide poisoning, coloured nail varnish and certain dyes like methylene blue are other sources of error.

Peripheral SpO_2 may lag 30–45 seconds behind central SpO_2.

SpO_2 does not give any information about adequacy of ventilation.

ELECTROCARDIOGRAPHY

Continuous observation of:

- Rate, rhythm and conduction.
- Lead II is ideal for detecting arrhythmias. CM5 configuration is ideal in detecting ST segment changes due to left ventricular ischaemia.
- Electrolyte status: peaked T waves (e.g. hyperkalemia) and prolonged QT (e.g. hypocalcemia).

SYSTEMIC BLOOD PRESSURE

NONINVASIVE BP

Automated oscillotonometry is used. Accuracy depends on:

- Correct cuff size: width 20% greater than arm diameter, or 1/3 of arm circumference.
- Correct application: loose wrapping results in falsely high readings.

INVASIVE BP

A transducer converts pressure waves transmitted from a vessel via fluid-filled tubing into changes in voltage or resistance. To obtain an optimally damped, accurate waveform:

- Use short connecting tubing (60–120 cm), with 1.5–3.0 mm internal diameter with rigid walls.
- Ensure no air bubbles are in the system.
- Use 4 mL hr^{-1} continuous flush to prevent thrombotic occlusion.

Precautions:

- Use a small cannula to avoid arterial occlusion or damage.
- Avoid accidental intra-arterial injection by using colour-coded taps.

- Display waveform.
- Ischaemia distal to the cannula is rare but should be looked for.

CENTRAL VENOUS PRESSURE (CVP)

Measures the filling pressure of the right atrium. The tip of the catheter is usually positioned in the SVC at the entrance to the right atrium. The internal jugular, subclavian and basilic veins are possible routes for cannulation. The Seldinger technique under real time ultrasound guidance is the most common method used. Complications include air embolism, arrhythmias, bleeding, pneumothorax and sepsis.

PULMONARY ARTERY AND CAPILLARY WEDGE PRESSURES

Measured via balloon-tipped flow guided catheter, it facilitates measurement of cardiac output and left atrial pressure. PCWP reflects LV filling pressure. CO, CI, CVP, RV pressure, PA pressure, stroke volume, SVR, PVR, SvO_2 (mixed venous oximetry) can also be obtained. In certain conditions, the PCWP does not accurately reflect LV filling pressure, e.g. mitral stenosis and regurgitation, massive PE.

Indications:

- Ischaemic heart disease, cardiogenic shock, right ventricular failure.
- Sepsis and septic shock.
- Adult respiratory distress syndrome.
- Oliguria and unexplained hypotension.
- Perioperative monitoring, e.g. coronary artery bypass grafting, vascular surgery.

Complications:

- Dysrhythmias
- Sepsis
- Pulmonary infarction
- Haemorrhage

Newer non-invasive cardiac output monitors are becoming more popular and are replacing the PA catheter in many cases.

OESOPHAGEAL DOPPLER

Cardiac output can be estimated by placing the probe in the distal oesophagus. The CO correlates well with that obtained from the PA catheter. Other parameters obtained include stroke volume, SVR and SV variance. This information can be used to guide fluid challenges and vasopressors.

LIDCORAPID™

Arterial pressure waveform analysis software is used to generate a 'nominal' cardiac output value. As it does not require calibration, it can be quickly set up and the effect of fluids or inotropes assessed.

TRANSOESOPHAGEAL ECHOCARDIOGRAPHY

Multiplane imaging of heart and colour Doppler visualization of blood flow are obtained. Conventional transthoracic echocardiography can be difficult in ventilated patients. It is particularly useful in cardiac surgery, allowing monitoring of left ventricular function, regional wall motion abnormalities (suggestive of ischaemia), detection of air or clot emboli, assessment of valve/septal repairs and prosthetic valve function. This technology may also be useful in high-risk patients and those undergoing surgery where more precise fluid management is essential.

NEUROMUSCULAR FUNCTION

The response to a supramaximal stimulus of a peripheral motor nerve is measured by eye, feel, force transducer or an electromyogram.

Train-of-four (TOF) is commonly used and indicates the degree of neuromuscular blockade. TOF count of 0–2 correlates with acceptable block. The TOF ratio (fourth twitch force as a fraction of the first) is useful for monitoring recovery. A TOF ratio of 0.7 has long been regarded as appropriate for reversal but recent evidence suggests that a higher value may be better.

Double burst stimulation may be easier to use than TOF. Two short bursts of 50 Hz are applied 750 ms apart and the ratio of the second to first response is estimated.

Tetanic stimulation (50 or 100 Hz for 5 s) in the presence of partial nondepolarizing block results in 'fade' (failure to sustain muscular contraction) and 'post-tetanic facilitation' of force from a single stimulus. This is useful for monitoring deeper levels of block in which there is no response to TOF.

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CROSS-REFERENCE

Depth of anaesthesia monitoring, Chapter 27

PRE-USE CHECK PROCEDURES

SINDY LEE AND BAHA AL-SHAIKH

Anaesthetists use a wide variety of equipment on a daily basis and must be adept at carrying out routine checks in order to deliver safe anaesthetic care. Pre-use check procedures for commonly used anaesthetic equipment should take place in accordance with the latest AAGBI recommendations.

SELF-INFLATING BAG

- This must be immediately available in any area where anaesthesia may be administered as it can potentially be life-saving in the event of oxygen supply failure as an alternative means of ventilation.

MANUFACTURER'S AUTOMATIC MACHINE CHECK

- Many modern machines perform automatic checks when switched on. Be familiar with the checks that are included and confirm that they have been performed satisfactorily.

POWER SUPPLY

- Check the anaesthetic machine is connected to mains electricity and switched on at the wall socket. If the anaesthetic machine has an ON/OFF switch, ensure that it is turned on.
- Ensure that the back-up battery is fully charged.
- Be aware of the back-up generators and systems available in your hospital and theatre.

GAS SUPPLIES AND SUCTION

- Once a week, check that the oxygen failure alarm sounds upon disconnecting the oxygen pipeline or gas supply master switch; keep a written record. Avoid more frequent checks as this can damage the Schrader valve connections.
- Perform a tug test to check each gas and vacuum pipeline is correctly inserted into the appropriate gas supply terminal.
- Pressure gauges from pipelines display a pressure of 400–500 kPa with adequate reserve supply of oxygen from a spare cylinder.
- Cylinders are adequately filled and turned off.
- Flowmeter (where present) control valves turn smoothly and the bobbin moves freely through entire range.
- Anti-hypoxia device provides a minimum of 25% oxygen when nitrous oxide is in use and discontinues flow of nitrous oxide upon oxygen failure.
- Emergency oxygen flush operates with no significant drop in pipeline supply pressure when pressed and shuts off when button is released.
- Suction is clean and able to generate a negative pressure of 500 mmHg within 10 seconds of occluding the tubing.

BREATHING SYSTEM

- Inspect breathing system to ensure correct assembly with no holes or foreign bodies.
- Secure connections with 'push and twist'.
- Ensure whole breathing system is patent with gas flowing to the patient interface (e.g. facemask).
- Pressure leak test for breathing system – occlude patient end, set APL valve between 20 and 60 cmH$_2$O and squeeze reservoir bag. A leak may be detected audibly or by the sensation of escaping gas.
- Check colour of carbon dioxide absorber.
- Check alternative breathing systems.
- Select correct gas outlet – recheck this every time a breathing system is changed.

VAPORISER

- Vaporisers are correctly seated on the back bar, not tilted, adequately filled with filling port tightly closed and plugged in where applicable. Control knobs rotate fully throughout the range.
- Leak test at common gas outlet: turn off vaporiser, set oxygen flow to 5 L/min, occlude common gas outlet of anaesthetic machine. There should be no audible leak and flowmeter bobbin (where present) dips. Turn on vaporiser and repeat test. There should be no liquid leaking from the filling port. Turn off vaporiser and flowmeters. *Caution*: this test may damage modern anaesthetic machines and it is recommended that the manufacturer's guidance be consulted.

VENTILATOR

- Configured correctly with appropriate alarm settings.
- Set controls to ensure adequate pressure is generated during inspiratory phase.
- Pressure relief valve is functioning.

TWO-BAG TEST

- Attach patient-end of breathing system to a bag or test lung.
- Turn on oxygen to 5 L/min.
- Manually ventilate to check system is patent and uni-directional valves are moving.
- Squeeze both bags to check the APL valve releases pressure at set limits.
- Switch over to ventilator to ventilate bag or test lung.
- Turn off oxygen or reduce flow to minimum.
- Open and close each vaporiser sequentially and check there is no loss of volume to indicate that there is no leak in the system.

SCAVENGING

- Anaesthetic gas scavenging system (AGSS) is switched on and functioning.
- Tubing connected to correct exhaust port of breathing system.

MONITORS

- All monitoring devices functioning with appropriate alarm settings and configuration. Specifically, check that:
  - *Non-invasive blood pressure* – Appropriate sized cuff available.
  - *Pulse oximeter* – Light emitting diode functioning normally.
  - Electrocardiography available.
  - *Gas monitoring and capnography* – Calibrated with no kinks or cracks in sampling lines.
  - *Oxygen analyser* – Displays 21% oxygen in air and approaching 100% when pure oxygen used.

ANCILLARY AIRWAY AND RESUSCITATION EQUIPMENT

- Ensure full range of airway equipment is available at the point of use, with spares, and functioning.

- Difficult airway equipment must be checked regularly and kept in a known location.
- Check that the patient's trolley, bed or operating table can be tilted head-down rapidly.
- Resuscitation trolley and defibrillator is checked regularly and available.
- Drugs and equipment for anaesthetic emergencies such as local anaesthetic toxicity and malignant hyperthermia are available and checked regularly.

RECORDING

- Record check in anaesthetic machine log book. This documentation should be sufficient to permit audit on a regular basis.
- A clear note must be made in the patient's anaesthetic record that the anaesthetic machine check has been performed, that appropriate monitoring is in place and functional, and that the integrity, patency and safety of the whole breathing system has been assured.

BEFORE EACH CASE

- Recheck the breathing system, ventilator, airway equipment and suction.

TOTAL INTRAVENOUS ANAESTHESIA

See total intravenous anaesthesia section.

SINGLE-USE DEVICES

- Any part of the breathing system, ancillary equipment or other apparatus that is designated 'single-use' must be used for one patient only, and not reused.
- Packaging should not be removed until the point of use, for infection control, identification and safety.

MACHINE FAILURE

- In the event of failure, some modern anaesthetic workstations may default to little or no flow or oxygen only with no vapour. Know the default setting for the machine in use.
- Alternative means of oxygenation, ventilation and anaesthesia must be available.

'SHARED RESPONSIBILITY' EQUIPMENT

- The anaesthetist shares responsibility for the use of other equipment, e.g. diathermy, intermittent compression stockings, warming devices, cell salvage and tourniquets.
- Involvement with this equipment, especially 'trouble shooting' problems that arise intraoperatively, must not be allowed to distract anaesthetists from their primary role.

RECOVERY

There must be clear departmental procedures for the daily and other checks of equipment that is used in recovery. This may also include pre-use checks of patient-controlled analgesia and epidural pumps, etc.

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CROSS-REFERENCE

Total intravenous anaesthesia, Chapter 28

VENTILATORS

SARAH HODGE AND BAHA AL-SHAIKH

A ventilator is an automatic machine designed to provide all or part of the work required to ventilate a patient's lungs. Classification has been updated to describe the physical characteristics of the ventilator and the breathing pattern delivered to the patient.

Power source	1. **Electrically powered** – Mains electrical output to power internal components including those which generate gas flow to the patient. 2. **Pneumatically powered** – High pressure gas is used to power the gas delivery to the patient. 3. **Pneumatically powered microprocessor-controlled** – Both power sources required since electrical power is required to power a microprocessor that adds further control options to the gas flow such as pressure waveform.
Pressure generation	1. **Positive pressure** – Most common. Gas is pushed into the lungs, generating positive pressure to cause chest expansion. Airway pressure is higher than atmospheric pressure so exhalation occurs passively due to pressure gradient and elastic recoil of the chest wall. 2. **Negative pressure** – • *Tank ventilator or 'iron lung'* – Gas is pumped out of the airtight tank to generate a vacuum around the body, decreasing intrapulmonary pressure and leading to chest expansion. As the vacuum is released, elastic recoil of the chest leads to expiration.
	• *Cuirass ventilator* – An upper body shell or cuirass generates negative pressure only around the chest. This technique has been improved by the ability to generate two pressures; biphasic cuirass ventilation. This allows control of expiration as well as inspiration and, therefore, the I:E ratio and respiratory rate.

Control systems	1. **Open and closed loops systems** – In closed loop systems, microprocessors allow feedback loops between the control variable (such as tidal volume) as set by the operator and the measured control variable (exhaled tidal volume). If the two differ, for example due to a leak, the ventilator can adjust to achieve the desired expired tidal volume by increasing the volume delivered. Open loop systems deliver ventilation as set by the operator but do not measure or adjust.
	2. **User interface/control panel**
	3. **Pneumatic circuit** – The means of delivering gas flow from high pressure gas source to the patient.
	  • **Internal:**
	    • **Single** – The gas from the high pressure source flows directly to the patient (e.g. modern ICU ventilators)
	    • **Double** – The power source causes gas flow to compress a chamber such as bellows or 'bag-in-a-chamber'. The gas in the chamber is then delivered to the patient.
	  • **External** – Tubing from the ventilator to the patient.

Drive mechanisms	The internal hardware that converts electrical power or gas pressure into a breath to the patient.
	1. *Flow devices* – Compressors move atmospheric pressure gas into a higher pressure storage chamber which is then delivered as a breath. Blowers generate high flows of gas as the direct ventilator output.
	2. *Volume displacement devices* – The volume of gas to be delivered to the patient is displaced by a moving part such as a piston or spring-loaded bellows.
Output control mechanism	Valves that regulate gas flow to and from the patient.
	1. *Proportional solenoid valve* – Opens in very small increments dependent upon flow required.
	2. *Digital on/off valves* – A collection of valves, each one is either fully open or closed. Each valve produces a certain flow by controlling the opening/ closing of a specifically sized orifice.

COMPONENTS OF BREATH DELIVERY

There are four components in breath delivery: trigger, delivery, cycle and expiration phases.

Trigger phase	The end of expiration and initiation of the breath by the following:
	• *Time* – Ventilator initiates the next breath a set time after the previous breath.
	• *Pressure* – The ventilator detects a drop in pressure as the patient inhales (generating negative pressure) triggering a ventilator delivered breath.

	• *Flow* – The ventilator has a set baseline flow around the patient circuit, if the patient takes a breath this will cause a drop in flow returning to the ventilator compared to that leaving. This drop in flow is detected by the ventilator, initiating the next patient-triggered breath.
	• *Volume* – The ventilator detects a drop in volume within the patient circuit during expiration as the patient inhales. This initiates the next breath.
Delivery phase	The delivery of inspiratory flow (to give a breath) up to a maximum set limit, known as the limit variable. This is set by the operator. Limit variable can be:
	• *Pressure* – Which cannot be exceeded to avoid excessive pressure being applied to the lungs.
	• *Flow* – Not used in spontaneously breathing patients as increased flow is needed to allow spontaneous breaths.
	• *Volume* – Maximum volume limit is set but if patient is spontaneously breathing this will not support their respiratory effort.
Cycle phase	This is the end of inspiration or breath termination, which occurs at the set cycle variable which is measured by the ventilator during the inspiratory phase. Cycle variable can be:
	• *Volume* – Inspiration ends when a set volume has been delivered.
	• *Time* – A predetermined time for inspiration is set, cycling occurs when this is reached.
	• *Flow* – When the inspiratory flow drops to a set percentage of the peak inspiratory flow rate inspiration ends.

	• **Pressure** – Inspiration ends when a set pressure in the upper airways is reached.
Expiration phase	Occurs when the expiratory valve in the ventilator opens and allows passive exhalation which depends upon lung recoil and airway/circuit resistance. Expiration occurs until the set baseline variable, which is usually a pressure.

MODES OF VENTILATION

Sophisticated ventilators may function in a number of modes, some which provide mandatory ventilation and others which provide some form of respiratory support when triggered by the patient. Triggered modes (see below) can be safely used only when there are spontaneous patient breaths.

- IPPV (Intermittent Positive Pressure Ventilation). Generic term used for all forms of positive pressure ventilation.
- CMV (Controlled Mandatory Ventilation). Mandatory mode where a preset minute volume is delivered usually to a paralysed or apnoeic patient as any patient respiratory efforts are ignored.
- Triggering indicates the ability of the ventilator to detect the initiation of a spontaneous breath by the patient, usually by detecting a set negative pressure in the breathing system. The patient triggers the onset of the inspiratory phase.
- PSV (Pressure Support Ventilation) is one commonly used mode in which the patient triggers the inspiratory phase. The adjustable level of pressure support (often 5–20 cm H_2O) determines the degree of respiratory support provided by the ventilator. Often the inspiratory phase during pressure support is terminated when the inspiratory flow-rate decreases below a critical value.
- AC (Assist Control). The ventilator provides a breath with a preset tidal volume (or preset peak pressure) each time the patient initiates a breath. Back up rate of mandatory breaths in case of apnoea.

- SIMV (Synchronised Intermittent Mandatory Ventilation). Similar to AC but ventilator provides a preset mechanical breath (the mandatory breath) every specified number of seconds (e.g. for 12 breaths per minute this is a 5-second cycle). Ventilation is synchronised to the first patient breath in the set cycle. Additional patient breaths after the first in the cycle are not supported, though PSV is commonly added to support these breaths (PSIMV).
- PSIMV is therefore a mode in which a mandatory number of breaths are delivered, breaths are synchronised to the first breath of a set time cycle, and further spontaneous breaths are supplemented by pressure support.
- PRVC (Pressure Regulated Volume Control). Under dual control of pressure and volume. Tidal volume is preset, and then the ventilator delivers pressure-controlled breath until tidal volume is achieved. Designed to optimise peak inspiratory pressures.
- APRV (Airway Pressure Release Ventilation). The ventilator cycles between two pressure levels (upper and lower pressure levels). Baseline airway pressure is the upper level, with pressure intermittently being released to allow removal of waste gases.
- BIPAP (Biphasic Positive Airway Pressure). Single ventilation mode which can be used throughout weaning. Uses the principle of APRV but with spontaneous breathing also. Flow is generated mechanically by alternating between the two pressure levels, and also by patient triggering. Can deliver mandatory ventilation alone, SIMV ventilation, or purely spontaneous ventilation. When the upper level is weaned to the same as the lower one, this is CPAP, and the patient takes over ventilation. Note: Not to be confused with BiPAP (Bilevel Positive Airway Pressure), which is a noninvasive, pressure support ventilatory system.
- PAV (Proportional Assist Ventilation). Form of synchronised ventilation where the ventilator generates pressure in proportion to the patient effort and responds to changes in lung dynamics. No target tidal volume, pressure or flow is

needed. The level of support given is dialed up as the percentage of patient work of breathing to be overcome (usually started at 80%).

- ASV (Adaptive Support Ventilation). Similar to PAV, but different algorithm (Hamilton Medical). Meant to optimize breathing pattern, promote spontaneous breathing and reduce weaning time.
- NAVA (Neurally Adjusted Ventilatory Assist). Novel mode of positive pressure ventilation where the ventilator is controlled by the patient's own neural control of breathing. Electrodes on a nasogastric tube monitor electrical impulses from the phrenic nerve at the level of the diaphragm, and adjust ventilatory parameters accordingly.
- PEEP is positive end expiratory pressure during controlled ventilation.
- CPAP is continuous positive airway pressure during spontaneous respiration and is functionally the same as PEEP. It can be used both in invasive ventilation and noninvasively.

CHECKING AND SETTING

Checking and setting the ventilator and its breathing system before use is vital:

- Connect to electricity supply, high pressure gas, low pressure gas, etc.
- Set tidal volume to 10 mL kg^{-1} or inflation pressure to 15 cmH$_2$O.
- Set respiratory rate to 12 breaths min^{-1}.
- Set I/E ratio to 1:2, with an inspiratory time of not less than 1 s.
- Switch on and see if pressure develops in the system.
- Check that the pressure relief valve is functioning at correct value.
- Check that gas monitoring is present and working.
- Check that airway pressure monitor is working.
- Check manual mode functioning (if fitted).
- Check that the emergency air intake is patent (if appropriate).

Many modern anaesthetic and ITU ventilators now have automated programs for checking the ventilatory breathing system.

HIGH FREQUENCY VENTILATION

Refers to ventilation occurring at rates well in excess of those in normal breathing.

- HFJV (High Frequency Jet Ventilation). Gas is passed by a small bore catheter, placed either sub- or supra-glottically. Ventilation rate can be from 4–11 Hz. Exhalation is passive. Uses include glottic/laryngeal surgery, bronchopleural fistula, and ARDS. Thought to reduce ventilator-induced lung injury (VILI).
- HFOV (High Frequency Oscillatory Ventilation). Pressure wave is generated by a moving diaphragm controlled electromagnetically. Respiratory rates of up to 15 Hz are achieved. Pressure oscillates about the distending pressure (analogous to PEEP). Tidal volumes are less than dead space. Multiple mechanisms of gas transfer have been proposed but exact mechanism is yet to be fully understood. Can produce pressures less than ambient pressure; hence, gas is pushed in during inspiration and actively 'pulled out' during expiration. Uses include severe ARDS and neonatal ventilation. Also thought to reduce VILI.

THE IDEAL VENTILATORY BREATHING SYSTEM

The ideal ventilatory breathing system should have the following characteristics:

- Wide range of tidal volumes and respiratory rates to encompass paediatric and adult use.
- Pressure or volume controlled modes.
- Adjustable inspiratory/expiratory timing (I/E ratio).
- Airway pressure display, with alarm limits.
- Expired minute volume display, with alarm limits.
- Adjustable, monitored inspired oxygen concentration.
- Provision of PEEP/CPAP.
- Sophisticated ventilatory modes.
- Humidification.
- Adjustable inspiratory waveforms.

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- Adjustable pressure relief valve.
- Facility for drug administration into breathing system (nitric oxide, inhaled bronchodilator).

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CROSS-REFERENCES

Techniques: General

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BLIND NASAL INTUBATION

SINDY LEE AND BAHA AL-SHAIKH

First introduced by Rowbotham and Magill in the 1920s, it was originally described in spontaneously breathing patients anaesthetised with an inhalational agent, but has been modified for intubating awake patients – with or without sedation. Since direct vision of the glottis is not necessary, it proved a useful technique in the management of patients with difficult airways. Blind nasal intubation has largely been superseded by fibre-optic intubation but remains a relevant and simple alternative in situations where a fibre-optic scope is not available or fibre-optic intubation has failed.

INDICATIONS AND CONTRAINDICATIONS

The indications and contraindications are essentially the same as those for fibre-optic intubation. The nasal route is indicated particularly where there are structural abnormalities in the mouth or limited mouth opening.

SEDATION FOR AWAKE BLIND NASAL INTUBATION

For awake blind nasal intubation, just as for awake fibre-optic intubation, the patient must be able to cooperate and respond to commands. Sedation, a topical vasoconstrictor and local anaesthetic will facilitate improved tolerance of the procedure and reduce bleeding.

TECHNIQUE

- Check that all necessary equipment are functioning and that emergency drugs are available.
- Gain IV access.
- Have a trained assistant familiar with blind nasal intubation, in addition to a sedationist where sedation is required.

- Ensure that the patient is spontaneously breathing at all times – this is key to success!
- Position the patient in the 'sniffing the morning air' position – with the neck flexed and head extended at the altantoaxial joint. Where there is concern about C-spine stability, carry out the procedure without neck manipulation (ideally with manual inline stabilisation).
- Identify a suitable nostril by assessing size, patency and history of epistaxis or polyps.
- Insert a well-lubricated 6–7 mm ID nasotracheal tube, which has been softened in warm water. The bevel should face the septum to reduce trauma to the inferior turbinate.
- Direct the tube posteriorly along the floor of the nasal cavity. Gentle pressure and twisting may be required to pop into the oropharynx.
- While keeping the other nostril and mouth closed, gently advance the nasotracheal tube into the hypopharynx towards the glottis, taking care to stay in the midline.

- Listen for breath sounds as a guide to the position of the tube tip. As the tube approaches the glottis, breath sounds get louder and misting of the tube may occur. Table 28.1 outlines the expected clinical findings at different positions and provides hints for repositioning to increase chances of a successful intubation.
- Once at the laryngeal inlet, ask the patient to take a deep inspiration to abduct the vocal cords and intubate the trachea in one smooth motion. Transient coughing and a degree of laryngospasm suggests correct tube placement where gag reflex is still intact – this should quickly settle down.
- Confirm correct placement in the trachea with breath sounds through the tube, movement of the reservoir bag when connected to the breathing system, inability to phonate and capnography.
- Inflate cuff and secure tube.
- In awake patients, induce anaesthesia.

Table 28.1 Clinical findings for tube repositioning to increase chances of a successful intubation

Position	Clinical findings	Action
Trachea	Breath sounds through tube maintained as it is advanced, coughing through tube	Confirm ETCO$_2$, bilateral air entry
Anterior	Breath sounds heard through tube but unable to advance further, bulge felt anteriorly at level of hyoid, coughing mostly through tube	Withdraw tube 2 cm, reduce neck extension or externally manipulate larynx before readvancing
Left/right piriform fossa	No breath sounds, unable to advance tube, no coughing, a bulge may be seen or palpated superio-lateral to larynx	Withdraw tube until breath sounds return, rotate tube to midline or turn head towards bulge and readvance
Oesophagus	Tube advances smoothly with loss of breath sounds, no cough, larynx elevated	Withdraw tube until breath sounds return and try the following manoeuvres before readvancing: • Head extension • Cricoid pressure • Inflate cuff with 15 mL of air to direct the tip anteriorly. Advance until resistance met, ensure breath sounds retained (at laryngeal inlet), slowly deflate cuff while maintaining some advancing pressure on tube to enter trachea

COMPLICATIONS

Complications can be classified into general, which are also associated with fibre-optic intubation, and specific, arising from using the nasal route and a blind technique.

General complications are described in the section on fibre-optic intubation.

Relating to *nasal intubation*: epistaxis, turbinate or polyp fracture, retropharyngeal laceration, retropharyngeal abscess and mediastinitis, intracranial placement in basal skull fracture and pneumocephalus.

Relating to the *blind technique*: trauma to the airway from multiple intubation attempts (e.g. crico-arytenoid cartilage subluxation), obstruction caused by the epiglottis being pushed into the glottic opening and oesophageal intubation. Unlike intubating under direct vision, there may be a delay in recognition of these complications.

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CROSS-REFERENCES

Difficult airway, Chapter 26
Fibre-optic tracheal intubation, Chapter 28

CLOSED CIRCLE ANAESTHESIA

SANJAY AGRAWAL AND BAHA AL-SHAIKH

When the current inhalational anaesthetic agents with low blood solubility were introduced, their expense and the increasing awareness of environmental pollution led to a renewed interest in the use of low flow and closed circle anaesthesia.
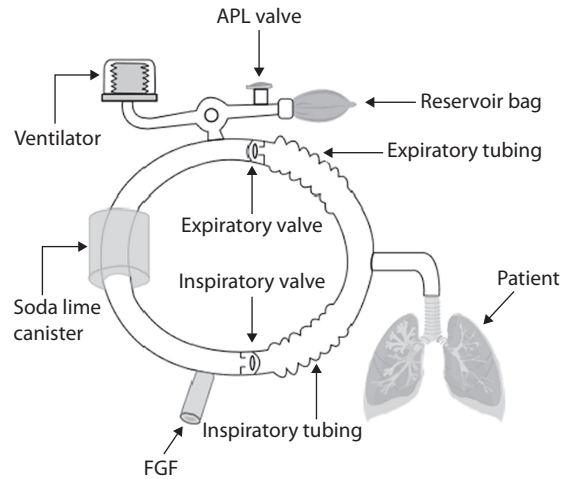
Figure 28.1 Most efficient configuration of the circle system.

The circle system is so named because its components are arranged in a circular manner. While it prevents rebreathing of CO_2, it allows rebreathing of other gases and vapours. It consists of:

- Fresh gas flow (FGF)
- Inspiratory and expiratory unidirectional valves
- Inspiratory and expiratory tubing
- Y piece connector
- APL valve
- Reservoir bag
- CO_2 absorber

The most efficient arrangement of these components (Figure 28.1) is such that:

- FGF enters the system before the inspiratory unidirectional valve.
- APL valve and reservoir are situated between the expiratory valve and the CO_2 absorber.
- CO_2 absorber is situated before the FGF entry point.

DEFINITIONS

- *Closed circle anaesthesia* – FGF is just sufficient to replace the volume of gas and vapour taken up by the patient (i.e. basal oxygen requirements, volatile agent uptake and N_2O

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uptake if used). No gas leaves via APL valve and the exhaled gases are rebreathed after CO_2 is absorbed. Significant leaks and gas losses are eliminated.

- *Low-flow anaesthesia* – FGF is less than the patient's alveolar ventilation (usually below 1.5 L/min). Excess gases leave the system via the APL valve.
- *Ultra-low-flow (minimal flow) anaesthesia* – FGF is less than 0.5 L/min. Excess gases leave the system via the APL valve.

ADMINISTRATION OF ANAESTHETIC AGENT

While it is possible to have vaporisers within the circle (VIC), it is more usual to have the vaporizer outside the circle (VOC). A VIC vaporiser has low resistance to gas flow to minimise the work of breathing required for spontaneously breathing patients. VIC systems allow vaporisation of agent into recirculated gas already containing anaesthetic agent, allowing rapid increases in concentration, especially when IPPV is used, potentially resulting in risk of accidental overdose. The calibration of such vaporisers is impossible.

Direct injection of volatile agent into the circle system has also been described. It is not recommended due to the great fluctuations of volatile concentration that result, and the inability to guarantee even and rapid evaporation of the agent.

VOC systems utilise a conventional plenum vaporizer to add agent to the FGF.

PRINCIPLES OF LOW FLOW ANAESTHESIA

- If oxygen and N_2O are used as the carrier gases, high FGF is needed initially to denitrogenate the circle and the FRC. This is important to avoid build-up of nitrogen in the system. In closed circle anaesthesia, this high FGF is needed for up to 15 minutes but in low-flow anaesthesia only 5 minutes is required. If oxygen and air are used as the carrier gases, denitrogenation is not necessary.

- Wash-in and wash-out curves for changes in vapour concentration within the closed system are exponential. The time constant is the duration needed to reach 63% of the intended FGF concentration in the system.

Time constant for circle = Volume of circle system/FGF

Three time constants are required for 95% equilibration of FGF with circle gases. As a result, it is normal to use an initial period of high flows to 'prime' the system and patient during the period of high initial volatile uptake before lower flows are established. If large changes in anaesthetic agent or carrier gases concentrations are required later it may be necessary to temporarily increase FGF.

- Side-stream gas/vapour monitors remove between 150 and 200 mL min^{-1} of gases from the breathing system for analysis. The sampled gas must be returned to the system for ultra-low flow or closed circle anaesthesia.

- For closed system, ultra-low and low-flow anaesthesia, a ventilator with rising bellows is necessary which will collapse if there is leak in the system. With the descending type of bellows, any leak may lead to entrainment of driving gas.

- FGF should be intermittently increased to aid removal of accumulated nitrogen (if oxygen and N_2O are used as carrier gases) and to remove other undesired gases which can accumulate in the system such as carbon monoxide, methane, acetone, ethanol, hydrogen and substance A.

- At the end of anaesthesia, anaesthetic gases are switched off and oxygen flows increased to rapidly wash out the anaesthetic agents.

VOLATILE REQUIREMENTS

- To calculate the dose of an anaesthetic agent required, we need to know
  - Minimum alveolar concentration (MAC)
  - The amount of vapour needed to achieve this within the system and lungs

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- The amount of vapour required for uptake into the circulation
- The amount required for uptake into the tissues
- The ED_{95} dose of a volatile agent is achieved at concentrations of 1.3 MAC and above.

Agent	MAC (%)	1.3 MAC
Halothane	0.75	>0.95
Enflurane	1.7	>2.26
Isoflurane	1.2	>1.5
Sevoflurane	2.0	>2.6
Desflurane	6.1	>8.1

- The amount of anaesthetic vapour required to achieve the target anaesthetic concentration in the breathing system and lungs depends upon the volume of the system and lungs. This is known as the ventilation-priming dose.

$$\text{Ventilation-priming dose} = \text{Target concentration}$$
$$\times (\text{Volume of system}$$
$$+ \text{Volume of lungs})$$

This volume of system and FRC provides a large reservoir, within which the anaesthetic gases are diluted at the beginning of anaesthesia.

Components	Volume (mL)
Absorber (2 kg)	2000
Breathing tubing (1 m)	1000
FRC	3000

$$\text{Ventilation-priming dose} = 1.3 \text{ MAC}$$
$$\times (\text{Volume of system}$$
$$+ \text{Volume of lungs})/100$$
$$= \text{mL of anaesthetic vapour}$$

DENITROGENATION

- A 70-kg adult has approximately 2500–3000 mL of nitrogen, of which 1300 mL is dissolved in the body (450 mL in the blood and 850 mL

in the fat). The remaining 1200–1700 mL is contained in the FRC.
- With a high initial FGF of 10 L min^{-1}, more than 95% of nitrogen is usually washed out of the FRC and circle in approximately 5 min. Thereafter, nitrogen build up occurs slowly due to release by tissues.

CO$_2$ ABSORPTION

- CO_2 absorbtion is achieved with soda lime (more common in the UK) or baralyme (more common in the US).
- Soda lime consists of 94% calcium hydroxide, 2%–5% sodium hydroxide, 0.2% silica (to prevent disintegration of the granules), an indicator dye and a zeolite which is added to maintain a higher pH for a longer time. Older variants also contained 1% potassium hydroxide, which acted as a catalyst for the reaction.
- Soda lime can absorb 25 L of CO_2 per 100 g. However, in practice, small canisters containing 500 g of soda lime appear exhausted with a CO_2 load of 10–12 L 100 g^{-1} and jumbo absorbers containing 2 kg of soda lime appear exhausted with a CO_2 load of 17 L 100 g^{-1}.
- Soda lime and its additives are made into granules to increase the surface area for absorption and to minimize resistance to gas flow. The granule size is measured in 'mesh'. The usual size is 4–8 mesh.
- Baralyme consists of 80% barium hydroxide and 20% barium octahydrate. It is less efficient than soda lime, but more stable in dry environments, and produces less heat.
- Absorber granules are consumed more rapidly the lower the FGF used because most of the exhaled gases pass through the absorber with very little being discarded through the APL valve.
- CO_2 absorption by soda lime or baralyme is an exothermic reaction resulting in heat and water vapour formation. The humidity generated by the reaction makes the use of a heat and moisture exchanger unnecessary.
- Sevoflurane is partly degraded by soda lime forming compound A, which is nephrotoxic in rats. The amount produced is proportional

to the sevoflurane concentration and the temperature of the absorber. The latter is higher with lower FGF. Sevoflurane use in humans is safe although it is advisable that it should not be used with FGF of less than 1.5 L min^{-1} for more than 3–4 hours.

- Enflurane, isoflurane and desflurane can react with soda lime or baralyme to form carbon monoxide. This is only significant when the water content is less than 1.5% in soda lime or less than 5% in baralyme. Carbon monoxide is produced when the absorber has been flushed with dry fresh gas for a considerable period of time without being attached to a patient before the agent is used (i.e. dry absorber). Humidity prevents its production.
- The CO_2 absorbant, Amsorb, does not contain a strong base (sodium or potassium hydroxide) and does not produce compound A or carbon monoxide when used with sevoflurane or desflurane.

CONTROL OF MOISTURE

Prolonged use of the circle system results in significant condensation of water vapour. This can result in sticking of the one-way valves due to surface tension, potentially turning the entire volume of the system into dead space. This complication can be reduced by using a hydrophobic filter (i.e. HME) at the end of the expiratory limb before the CO_2 absorber, or by changing the breathing tubing between cases.

MONITORING

The following monitoring is recommended for the use of closed circle, ultra-low flow and low-flow anaesthesia, due to the possible variations between FGF composition, and inspired and expired gas compositions.

- Inspired and end tidal oxygen
- Inspired and end tidal CO_2
- Inspired and end tidal N_2O
- Inspired and end tidal agent
- Tidal volume
- Standard monitoring

PRECAUTIONS AND SAFETY FEATURES

- Monitor inspired oxygen, end-tidal carbon dioxide and inhalational agent.
- Prevent the unidirectional valves from sticking because of water vapour condensation.
- Resistance to breathing is increased especially during spontaneous ventilation mainly due to the unidirectional valves.
- Compound A: newer designs of soda lime claim less or no production of compound A; baralyme is worse than soda lime and Amsorb© is the safest.
- Carbon monoxide production can occur when certain agents are used with very dry granules; recent designs of soda lime claim less or no production of carbon monoxide.
- Methane, acetone, ethanol and hydrogen can accumulate but generally do not become clinically significant.
- Uneven filling of the canister with soda lime leads to channelling of gases and reduced efficiency.
- Because of many connections, there is an increased potential for leaks and disconnection.

ADVANTAGES OF LOW-FLOW TECHNIQUES

- Economy of gases and inhalational agents
- Humidification of the inspired gases
- Reduced atmospheric pollution
- Greater understanding of breathing systems and the pharmacokinetics of inhalational anaesthesia is claimed

DISADVANTAGES OF LOW-FLOW TECHNIQUES

- Capital investment for breathing systems and gas monitoring may limit use in poorer countries
- Accumulation of unwanted gases although this is less of a problem with modern low flow systems

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CROSS-REFERENCES

Awareness, Chapter 30
The anaesthetic machine, Chapter 27

FIBRE-OPTIC INTUBATION

SINDY LEE AND BAHA AL-SHAIKH

Fibre-optic intubation is an essential skill in the management of difficult airways. It can be performed either awake, with or without sedation, or asleep. Awake fibre-optic intubation (AFOI) is the gold standard for anticipated difficult airways. Asleep fibre-optic intubation may be suitable in patients with isolated difficult tracheal intubation.

INDICATIONS

- Known difficult intubation or previous awake fibre-optic intubation
- Anticipated difficult intubation
- After failed intubation in unanticipated difficult airway
- Known or suspected difficult mask ventilation
- Unstable C-spine

CONTRAINDICATIONS

ABSOLUTE

- Patient refusal or inability to cooperate

RELATIVE

- Lack of trained personnel
- Risk of impending airway obstruction
- Coaguloapthy or bleeding in airway
- Allergy to local anaesthetic
- Base of skull fracture (for nasal route)

PREOPERATIVE PATIENT ASSESSMENT

Refer to section on difficult airway management.

PREPARATION

- Ensure that all necessary equipment and emergency drugs are available and checked.
- Two anaesthetists – One to perform the intubation, one to provide sedation – and a trained assistant familiar with fibre-optic intubation.
- Intravenous access.
- Full monitoring throughout the procedure, including ECG, pulse oximetry, noninvasive blood pressure, capnography and sedation level.
- Glycopyrronium (400 mcg IM or 200 mcg IV), atropine or hyoscine to reduce airway secretions.

SEDATION

- Conscious sedation improves the level of comfort and cooperation. It is not essential as local anaesthetic alone is adequate. If the airway is already acutely compromised, sedation may lead to complete obstruction and might best be avoided.
- Appropriate methods of sedation include:
  - *Remifentanil* – Target controlled infusion (TCI) at an effect site concentration of 1–5 ng/mL as sole agent or in conjunction with propofol or midazolam. Provides

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excellent analgesia, anxiolysis and obtunds the laryngeal reflexes.

- *Propofol* – As an infusion or intermittent boluses either as sole agent or in combination with fentanyl, remifentanil or midazolam. When used with a second agent by TCI, an effect site concentration of 0.5–1 mcg/mL is usually adequate.
- *Midazolam* – 0.5–1 mg boluses to maximum of 0.05 mg/kg, usually in conjunction with an opioid.
- *Dexmedetomidine* – Loading dose of 0.7–1 mcg/kg over 10–20 minutes and a maintenance infusion of 0.3–0.7 mcg/kg/h, it offers analgesia, amnesia and anxiolysis without respiratory depression.

LOCAL ANAESTHETIC

- Identify the preferred nasal passage based on assessment of patency and history of epistaxis.
- Administer supplemental oxygen at 4 L/min via nasal sponge in opposite nostril. Alternatively nasal cannulae can be used for oral fibre-optic intubation.

Administer local anaesthetic to the airway in a stepwise manner. Do not exceed a maximum dose of topical lidocaine of 7 mg/kg.

NOSE

The aim is to reduce bleeding and oedema with a vasoconstrictor and provide topical anaesthesia:

- Co-phenylcaine (0.5% phenylephrine + 5% lidocaine) given via mucosal atomiser to each nostril.
- Co-phenylcaine, xylocaine (2% lidocaine + adrenaline) or 4% lidocaine soaked cotton buds/ribbon gauze inserted into nasal cavity and left for 3 minutes.
- 2 mL of 4% lidocaine via nebuliser.

OROPHARYNX

- 10 sprays of 10% lidocaine to tongue and posterior pharynx.

LARYNX

- 4% lidocaine sprayed above vocal cords under direct vision during endoscopy. This is achieved by advancing an epidural catheter through the working channel of the fibrescope, or attaching a syringe with lidocaine and 1 mL of air directly onto the working channel (Spray As You Go technique).

TRACHEA

- Either 4% lidocaine sprayed below the vocal cords under vision during endoscopy or cricothyroid injection of 2 mL of 2% lidocaine via 20G cannula or 22G needle (immediately remove needle after injection to avoid trauma caused during coughing).

TECHNIQUE

- Place the patient in a semi-recumbent position.
- Check orientation of the fibrescope and perform white balance.
- Load the scope with a lubricated 6 mm ID endotracheal tube and fix with tape in a way to allow rapid release.
- Pass epidural catheter down suction port if using Spray As You Go technique. Ensure tip of catheter is not protruding out of the distal end of scope when not administering local anaesthetic.
- Insert the fibrescope through the nostril or mouth depending on chosen route. For oral intubation, use a Berman airway or Breathesafe as a bite block and to maintain the scope in the midline.
- Once in the oropharynx, identify the epiglottis and slowly advance towards the glottic opening. Visualisation may be improved by asking the awake patient to stick out their tongue. In asleep patients, ask your assistant to pull the tongue with a swab or provide jaw thrust.
- Keep black air cavity in the centre of the screen to assist with orientation. When the whole screen is pink, the tip of the scope is against mucosa – withdraw slightly until relevant anatomy can be identified before advancing further.

- Spray local anaesthetic onto the laryngeal inlet. Warn the patient that this will cause them to cough.
- Enter the subglottic space to identify the trachea and spray a further dose of local anaesthetic.
- Advance the scope until the tip is just proximal to the carina.
- Release the endotracheal tube and railroad this gently over the fibrescope. In the event of hold-up, do not use force but rotate the tube as it is advanced.
- Confirm that the tip of the endotracheal tube is in the trachea on removal of the fibrescope under vision.
- Connect to breathing system and check for correct placement with capnography.
- Proceed with induction of anaesthesia with inhalational or intravenous induction agents.
- Inflate endotracheal tube cuff.
- Plan for difficult extubation as per Difficult Airway Society guidelines.

COMPLICATIONS

- Oversedation, respiratory depression, airway obstruction, apnoea.
- Trauma, bleeding, airway obstruction, laryngospasm, vomiting in unstarved patient.
- Local anaesthetic allergy, toxicity, risk of aspiration due to loss of laryngeal reflexes.

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CROSS-REFERENCE

ONE-LUNG ANAESTHESIA

DANIEL LAKE, NESSA DOOLEY AND SIMON STACEY

One-lung anaesthesia is the process of complete functional separation of the two lungs. It facilitates certain types of thoracic surgery but causes significant physiological disadvantages in regards to pulmonary gas exchange.

INDICATIONS

- Protective isolation.
  - To prevent contamination or spillage of infectious material – pus or secretions from the contralateral lung.
  - To prevent massive hemorrhage.
- To control the distribution of ventilation between the two lungs in the presence of:
  - Bronchopleural fistula
  - Giant unilateral cyst/bulla
  - Surgical opening of major airway
  - Tracheo-bronchial tree disruption
- Unilateral bronchopulmonary lavage, e.g. for alveolar proteinosis.
- VATS.
- To facilitate surgical exposure for pulmonary surgery (e.g. pneumonectomy, lobectomy or thoracoscopy) or non-pulmonary surgery (e.g. oesophageal surgery, thoracic aneurysm, thoracic spinal surgery). Many thoracic procedures can be accomplished with a normal tracheal tube.

TECHNIQUES OF LUNG SEPARATION

- *Double lumen tube* – Allow rapid transition between one-lung ventilation and two-lung ventilation, permitting either lung to be suctioned and CPAP applied to the non-ventilated lung.
- *Bronchial blockers* – Do not facilitate ventilation or suction distal to the blocker.
- *Uncut tracheal tube* – Can be advanced into the relevant main bronchus. Not ideal, but useful in an emergency situation.

- *Papworth BiVent tube* – A new double lumen tube designed to enable rapid and reliable lung isolation using a bronchus blocker without endoscopic guidance. Allows suctioning of the collapsed lung through a central opening in the bronchial blocker but not ventilation.

PHYSIOLOGICAL EFFECTS OF THE LATERAL DECUBITUS POSITION

Awake, closed-chest: Gravity causes a vertical gradient in the distribution of pulmonary blood flow with the dependent lung better perfused. The dome of the lower diaphragm is pushed up into the chest and hence can contract more efficiently. Thus, the better perfused dependent lung is better ventilated regardless of the side on which the patient is lying.

Anaesthetised closed-chest: There is no difference in the distribution of pulmonary blood flow between dependent and non-dependent lung when compared to an awake patient. Ventilation is now switched from the dependent to the non-dependent lung. This is due to a loss of FRC with induction of anaesthesia which leads to the dependent lung moving to a less favourable portion and the non-dependent lung moving to a more favourable portion on the pressure-volume curve. The high curved lower diaphragm no longer confers any advantage in ventilation. The weight of the mediastinum, abdominal contents and the chest wall all impede lower lung ventilation.

Anaesthetised, open-chest, paralysed: The dependent lung is less compliant, poorly ventilated and overperfused whereas the non-dependent lung is over-ventilated and underperfused resulting in a considerable degree of V/Q mismatch. When one-lung ventilation commences, the preferential distribution of ventilation to the upper lung is completely eliminated. Perfusion to this lung continues and this results in increased shunt. At this stage, the dependent lung receives the entire minute volume and a high proportion (about 60%) of the cardiac output.

Hypoxic pulmonary vasoconstriction increases the pulmonary vascular resistance of the collapsed lung; however, its effects and the effects of anaesthetic agents on it are usually of little clinical importance in routine thoracic practice.

PREOPERATIVE ASSESSMENT AND INVESTIGATIONS

- Full blood count; urea and electrolytes.
- Chest X-ray: assess the anatomy of the airways to see if endobronchial intubation is possible. Often a CT of the thorax is also available.
- Arterial blood gases.
- Pulmonary function tests and CPET.
- ECG.

PERIOPERATIVE MANAGEMENT

MONITORING

- Routine standard monitoring with direct arterial pressure.
- Airway inflation pressure; flow/volume loops are also useful.
- Fluid balance and blood loss.
- Core temperature.
- Central venous pressure (insert ipsilateral to the thoracotomy).
- Nerve stimulator.

ANAESTHETIC TECHNIQUE

- Premedication if necessary.
- General anaesthesia (TIVA or inhalational agent) with muscle relaxation and controlled ventilation.

DOUBLE LUMEN TUBES

Robertshaw double-lumen endobronchial tubes are available in three sizes (small, medium and large). The PVC derivative (Bronchocath) is more malleable but more likely to migrate and suffer cuff damage by teeth during insertion. They are available in a wide range of right and left. Care must be taken not to over-distend the bronchial cuff since bronchial rupture may be catastrophic.

Right-sided endo-bronchial tube placement should be avoided where possible because of the high risk of right upper lobe occlusion and lobar collapse.

The correct positioning of a double-lumen tube is traditionally confirmed by observation of chest expansion and auscultation while each lumen in turn is occluded. However, reliance on clinical signs alone will miss a significant number of malpositioned tubes. NCEPOD 1998 highlighted the morbidity and mortality associated with malpositioned double lumen tubes. The use of a flexible fibre-optic bronchoscope allows direct visual confirmation of tube position and is now the method of choice. The new VivaSight-DL double lumen tube has an integrated high resolution camera for continuous monitoring of tube position, but there is a limited range of sizes and only a left-sided version exists.

ARTERIAL HYPOXAEMIA

Hypoxaemia is one of the most important problems encountered during one-lung anaesthesia. The elimination of carbon dioxide during one-lung ventilation is not a problem if the minute volume is maintained at the amount previously delivered to the two lungs. Two-lung anaesthesia should be maintained for as long as possible.

MANAGEMENT OF HYPOXAEMIA DURING ONE-LUNG VENTILATION

- Increase inspired oxygen to 100%.
- Check position of tube with fibre-optic bronchoscope. Suctioning of secretions may be required.
- Ensure adequate blood pressure and cardiac output.
- PEEP 5–10 cmH_2O to the dependent lung to decrease atelectasis and increase FRC. Excessive PEEP increases pulmonary vascular resistance and may increase shunt.
- CPAP 5–10 cmH_2O with 100% oxygen to the non-ventilated lung to facilitate oxygen uptake in this lung whilst not adversely affecting the surgical conditions.
- Abandon one-lung ventilation and intermittently ventilate the collapsed lung after warning the surgeon.
- Early clamping of the appropriate pulmonary artery will stop the shunt.

POSTOPERATIVE PERIOD

Adequate pain relief, physiotherapy and high dependency care are important factors for reducing the incidence of postoperative complications and hospital stay.

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Breathing systems, Chapter 27
Thoracic surgery, Chapter 15
Pneumonectomy, Chapter 15
Lobectomy, Chapter 15

PROLONGED ANAESTHESIA

GREGORY WAIGHT AND BAHA AL-SHAIKH

Adequate preparation, good management and close attention to details reduce the risks associated with prolonged anaesthesia. It contributes to perioperative complications which in turn may lead to delayed hospital discharge.

PROBLEMS ASSOCIATED WITH PROLONGED ANAESTHESIA

- Accumulation of anaesthetic agents leads to delayed emergence depending on pharmacokinetics of the drugs used:
  - Volatile anaesthetics with a high blood: gas solubility coefficient, e.g. isoflurane (1.4) will have a longer wake-up time compared to agents with a lower value, e.g. desflurane (0.4).
  - Fentanyl duration of action is prolonged with increasing length of infusion. This is in contrast to remifentanil, which displays a relatively constant clearance profile independent of duration of infusion.
- Potential toxicity of administered agents:
  - Degradation of inhalational agents by CO_2 absorber may lead to accumulation of toxins, e.g. sevoflurane to compound A. In the US, the use of sevoflurane with fresh gas flow rates of at least 2 L min^{-1} is recommended for procedures lasting more than 1 hour.
  - Inorganic fluoride production from hepatic metabolism of sevoflurane and enflurane may be nephrotoxic in patients with chronic renal impairment.
  - Prolonged exposure to nitrous oxide may result in acute vitamin B_{12} deficiency with megaloblastic anaemia and neurological deficit. Toxic manifestations may occur in susceptible individuals after shorter exposures. Risk is increased in pernicious anaemia, exposure to DNA synthesis inhibitors (e.g. methotrexate), pre-existing bone marrow depression, folate deficiency, diseases of the ileum and malabsorption. Nitrous oxide also causes expansion of air spaces.
- Impairment of gas exchange and respiratory mechanics, notably development of hypoxaemia and hypercarbia secondary to slowly developing dependent atelectasis.
- The influence of anaesthetic agents on renal function can lead to water and salt retention. Disturbances in intermediary carbohydrate metabolism promotes the development of metabolic acidosis.
- Retention of anaesthetic agents in the body can extend untoward effects into the postoperative period.
- Decreased carbohydrate metabolism results in intraoperative hyperglycaemia.
- Problems exist with accurate management of fluid and electrolyte balance.
- Inadvertent perioperative hypothermia, defined as a core body temperature below 36°C, can lead to:
  - Increased wound infection
  - Surgical bleeding
  - Impaired immune function
  - Increased incidence of myocardial ischaemia and infarction
  - Malignant arrhythmias
  - Postoperative shivering
- Prolonged immobility can lead to:
  - Increase risk of deep vein thrombosis
  - Nerve damage and pressure sores
  - Bilateral compartment syndrome
  - Rhabdomyolysis
  - Corneal damage if eyes are left open
- Postoperative delirium.
- Immunosuppression and increased susceptibility to infections.
- Increased opportunity for human error due to fatigue.

PREPARATION

- Discuss the risks of prolonged procedures with the theatre team prior to the case and plan appropriately.
- Position the patient with meticulous attention to detail, including:
  - Padding of pressure areas
  - Neutral joint positioning and support of the lower back
  - Avoid traction on at-risk nerves, e.g. brachial plexus
  - Ensure documentation of these interventions

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- Adequate scavenging to protect the theatre team: in the UK, recommended maximum accepted concentrations over an 8-hour time-weighted period are
  - 100 parts per million (ppm) for N_2O
  - 50 ppm for enflurane and isoflurane
  - 10 ppm for halothane
- Use a low-flow circle system to reduce consumption of gases and vapours.
- Maintain body temperature (theatre temperature, forced air warmer, warming blanket, fluid warmers, humidification of inspired gases, clothing/limb wrapping, hat).

MONITORING

- Minimal monitoring with direct invasive BP
- Core and skin temperature
- Blood loss
- Bispectral index analysis
- Tracheal tube cuff pressure
- Peripheral nerve stimulator
- Blood gases, electrolytes, glucose, coagulation
- Pressure-volume loop and lung compliance
- Inspiratory and expired concentration of oxygen, nitrous oxide, anaesthetic vapour and CO_2 concentration
- Fluid balance (central venous pressure, hourly urine output via urinary catheter)
- Cardiac output

ANAESTHETIC TECHNIQUE

The technique chosen is dependent on the surgery proposed, anticipated blood loss and the chronic health status of the patient. Controlled ventilation provides the ability to manipulate oxygenation and carbon dioxide. There have been case reports of healthy patients having prolonged surgery on the extremities breathing spontaneously via a laryngeal mask for 8 hours with no adverse effect. Regional anaesthesia may be used for surgery on the lower extremities, but prolonged immobilisation often necessitates light general anaesthesia or sedation for patient comfort.

- Consider using oxygen/air technique, omitting nitrous oxide with minimum FiO_2 to achieve an acceptable oxygen saturation or tension.
- Consider TIVA/TCI.
- Use high-volume low-pressure cuff with regular tracheal tube suctioning.
- Pay careful attention to body positioning with frequent repositioning of the head.
- Passive movement of joints through their range of motion periodically may reduce incidence of arthralgia postoperatively.
- Cover exposed body surfaces.
- Eye protection (lubrication, tapes, padding or eye shields).
- DVT prophylaxis.
- Be aware of the risks of fatigue amongst the theatre staff, and the increased likelihood of errors.

POSTOPERATIVE CARE

Consider:

- ITU/HDU stay for continued ventilation until warm and stable; awaken slowly.
- Regular physiotherapy.
- Maintenance of DVT prophylaxis.
- Ensure adequate fluid input and output.

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CROSS-REFERENCES

Monitoring, Chapter 27
Awareness, Chapter 30

TOTAL INTRAVENOUS ANAESTHESIA (TIVA)

MAUREEN BEZZINA AND BAHA AL-SHAIKH

TIVA is a technique where intravenous drugs are used to induce and maintain general anaesthesia, avoiding the use of inhalational anaesthetics. A continuous intravenous infusion is used commonly in the form of a target controlled infusion (TCI) pump.

CHARACTERISTICS OF THE IDEAL AGENT

Properties	Characteristics
Physical	• Long shelf life
	• No reconstitution before use
	• Stable in solution
	• Does not corrode metal or rubber
	• Stable in the presence of light and air
	• Non-flammable
	• No additives
	• No interaction with other drugs
Pharmacokinetic	• Rapid onset
	• Rapid metabolism and elimination, through different pathways to ameliorate accumulation of drug or metabolites
	• Minimal context sensitive half life
	• Non-cumulative over time
	• Rapid and predictable offset

Properties	Characteristics
Pharmacodynamic	• No cardiovascular or respiratory depression
	• No epileptiform movements, excitation or emergence phenomena
	• No pain on injection and not irritant if injected extravascularly
	• Safe following inadvertent intra-arterial injection
	• No histamine release or hypersensitivity reactions
	• No adverse drug reactions
	• Analgesic properties at sub-anaesthetic concentrations
	• Antiemetic
	• Safe in pregnancy and paediatrics

Both hypnosis and analgesia can be achieved using an intravenous technique. At the present time, propofol is the hypnotic of choice for TIVA. Analgesia can be achieved using the short acting opioids such as alfentanil and remifentanil. More recently, TCI has been widely used, although TIVA can also be achieved using intermittent bolus injection or manual infusion techniques.

TIVA must achieve the following goals: smooth induction, reliable and titratable maintenance and rapid emergence. Achieving these has been possible with TCI.

DOSE–RESPONSE RELATIONSHIP

In order to compare TIVA with inhalational anaesthesia, $CP50_m$ has been used. This is the plasma concentration required to prevent 50% of patients from responding to painful stimuli – similar to MAC for volatile anaesthetics. It is important to note, however, that at present, no method for measuring drug concentration directly in the plasma is commercially available and so $CP50_m$ values are based on calculated data only.

TARGET CONTROLLED INFUSION (TCI)

As there is no method that allows real-time measurement of plasma concentration of a drug, sophisticated TCI syringe drivers are used. These incorporate real-time pharmacokinetic models that deliver the appropriate dose of the drug to achieve and maintain the requested target concentration. For this to be achieved, the appropriate infusion rates needed to produce a required target concentration are continuously calculated by the microprocessor within the syringe driver.

A bolus/elimination/transfer principle is used by TCI pumps to maintain an appropriate plasma level of drug. Anaesthesia is induced by the syringe driver infusing propofol rapidly, giving a bolus calculated to achieve the required plasma concentration. This is followed by a progressively decreasing infusion rate calculated to match the *transfer* of drug in and out of the peripheral compartments and *elimination* of drug from the body (and therefore maintain the required plasma concentration). Once the compartments reach steady state concentration, the infusion rate slows to match elimination only.

To increase the target plasma concentration further, the syringe driver delivers another bolus to achieve the desired concentration and then maintains this higher concentration with a higher infusion rate. To decrease the target plasma concentration, the syringe driver stops infusing until the microprocessor calculates that the new target has been achieved. The new, lower level is then maintained.

The predicted concentration in the brain, which is displayed as the effect site concentration, increases slower than that of the plasma (plasma site concentration). Modern TCI pumps have software allowing titration to a choice of either plasma site or effect site concentrations, depending on the mathematical pharmacological model used.

As there is considerable variation in uptake and effect of drugs between individual patients, it has been proposed by many that depth of anaesthesia monitoring (such as bispectral index [BIS]) be used during TIVA used for hypnotic purposes on paralysed patients.

PROPOFOL

Propofol (a phenolic derivative – 2,6 diisopropyl-phenol) is highly lipid-soluble and is available as either a 1% or 2% lipid–water emulsion (with soya bean oil and egg phosphatide), as its water solubility is low. It is cleared from the body mainly by hepatic metabolism. It has, however, been successfully used in patients with liver cirrhosis, and the pharmacokinetics are not significantly affected.

The pharmacokinetics of propofol may be influenced by age. Values for clearance and plasma concentration on awakening are higher in children than adults. Children require significantly higher doses of propofol than do adults.

The rapid recovery from propofol is due to its short distribution phase, high clearance rate and short elimination half-life.

PROPOFOL TCI MODELS

Currently there are four common propofol TCI models in use. These are Marsh and Schnider for adults and Kataria and Paedfusor for paediatrics.

The Marsh model bases its calculations on the assumption that the central compartment volume is proportional to the weight of the patient, ignoring the age. Therefore, it administers the same bolus dose to all patients of a given body mass for any chosen plasma concentration regardless of their age (and hence their pharmacodynamic response). The Marsh model requires age to be inputted into the pump but only to ensure that the age of the patient is >16 years. It assumes that the central compartment volume is 19.4 L for an 85 kg patient.

The Schnider model was developed later and requires age, height and body weight to be inputted. The pump then calculates a sex-specific lean body mass and works out the dosages accordingly. It assumes that the fixed central compartment volume is 4.27 L for an 85 kg person and therefore there is a fourfold difference in calculated peak plasma concentration when compared to the Marsh model. Lower doses of propofol are required to achieve a given plasma concentration and therefore this model is used more commonly in the elderly population.

The Schnider model allows for effect site concentration to be targeted specifically, whereas the Marsh model was designed primarily for targeting plasma site concentration (although effect site concentration is also calculated).

The Paedfusor model is a variant of Marsh kinetics for patients between 1 and 16 years of age. It uses the weight to calculate target plasma concentration and it features a nonlinear scaling of central compartment volume as age exceeds 12 years.

The Kataria model can be used for patients 3–16 years of age with a minimum weight of 15 kg. It also uses weight to calculate target plasma concentration.

Propofol TCI allows easy control and rapid change of the target propofol concentration. In unpremedicated adult patients under the age of 55 years, a target propofol concentration of 4–8 μg mL^{-1} is usually adequate for the induction of anaesthesia. Induction is smooth and usually takes 60 to 120 seconds. When co-administering an analgesic, anaesthesia can be maintained using propofol concentrations of approximately 3–6 μg mL^{-1}. Lower target concentrations are used in elderly patients reducing the risks of side effects. Due to the differing pharmacokinetic profile in children, propofol TCI has not been used extensively in the paediatric population.

MANUAL INFUSION AND MANUAL INTERMITTENT BOLUS TECHNIQUES

Manual infusion can be achieved by a bolus dose for induction using a syringe driver with a rapid bolus facility. Anaesthesia is maintained using a step-down sequence of infusion rates. Before the advent of TCI, a popular method was to administer a 1 mg/kg propofol bolus followed by 10 mg kg^{-1} h^{-1} for 10 min, 8 mg kg^{-1} h^{-1} for 10 min, and 6 mg kg^{-1} h^{-1} thereafter – the Bristol model. This model was originally described, however, to include premedication with fentanyl and co-administration of nitrous oxide.

A manual intermittent bolus technique has also been used but leads to wide variations and fluctuations in the plasma concentrations and the anaesthetic effects. The majority of anaesthetists now prefer to use a TCI system due to its simplicity and reliability.

ANALGESIA

Since propofol has no analgesic properties, TIVA is generally achieved by combining a propofol infusion with a regional local anaesthetic block or supplemental opioids. The shorter-acting opioids are often suggested as an ideal complement to propofol. Of these, remifentanil, alfentanil and sufentanil are widely used.

REMIFENTANIL

Remifentanil (a fentanyl derivative and pure μ agonist) has a unique metabolic and pharmacokinetic profile. It undergoes rapid methyl esterase hydrolysis by tissue and plasma esterases to relatively inactive metabolites. Its effect is terminated by rapid metabolic clearance (elimination half-time is 3–10 minutes) rather than redistribution, unlike fentanyl and alfentanil, resulting in rapid reduction in plasma concentration even after prolonged infusion. It does not accumulate in either hepatic or renal failure.

The time required for the drug concentration to fall by 50% (context sensitive half time) is always the same at about 3 minutes, leading it to be described as context *insensitive*. This is independent of age, weight, sex or hepatic and renal function, making it ideal for a continuous intravenous technique since it does not accumulate even after prolonged infusions.

Remifentanil can be given via TCI using the Minto pharmacokinetic model. This is easy to use and allows easy titration based on patient age, gender, weight and height. Remifentanil has become the opioid of choice in TIVA for many anaesthetists, certainly in longer or more stimulating procedures. A target plasma concentration of 3–8 ng/mL is usually required for induction and intubation and this can be increased to up to 15 ng/mL in stimulating procedures. Care must be taken, however, to ensure adequate analgesia after remifentanil has worn off. This can be ensured either by local/regional techniques or by judicious administration of an alternative opioid toward the end of the case.

ALFENTANIL

Alfentanil (a μ agonist) has a short onset of action of 90 seconds and can be used effectively in TCI for

analgesic purposes. The Maitre model is mostly used for alfentanil TCI and it bases its calculations on age, gender and weight.

SUFENTANIL

Sufentanil is a much more potent opioid than remifentanil but it has a longer duration of action and therefore tends to accumulate if infused for a prolonged time. Two TCI models were developed for sufentanil: Gepts and Bovill. The Gepts model uses a fixed compartment volume which is not dependent on weight. The Bovill model bases its calculations on the assumption that the central compartment volume is proportional to the body weight.

USE OF TCI IN THE ELDERLY AND PAEDIATRIC POPULATION

Patients at the extremes of age have different physiological factors that may affect the pharmacokinetics of drugs.

Elderly patients have a smaller central compartment volume, decreased clearance, increased volume of distribution and altered metabolism. Lower concentration of drugs is required to induce and maintain anaesthesia, and they are more sensitive to the effects of drugs on the cardiorespiratory system.

TCI use in the paediatric population is less popular. Children tend to have a larger central compartment volume and a higher clearance rate than adults.

SAFE PRACTISE DURING USE OF TIVA

The NAP5 (2014) highlighted problems during the use of TIVA which led to accidental awareness with the following recommendations:

- Making sure the cannula is visible and accessible at all times and checked regularly to prevent disconnection or tissuing.
- Checking pump setup regularly to make sure tubing disconnection accidents do not occur, clamps are open/closed accordingly, pump alarms are rectified and no backtracking of drug occurs.

- Ensuring that the concentration of drug matches that programmed.
- Ensuring correct drug syringe is placed in the correct syringe driver programmed for it.

The Safe Anaesthesia Liaison Group in 2007 also recommended the use of anti-reflux and anti-siphon valves with clamps in a multilumen tubing for safe delivery of a continuous intravenous infusion.

INDICATIONS FOR THE USE OF TIVA

1. Malignant hyperthermia susceptibility
2. Long QT syndrome
3. History of severe PONV
4. Surgery requiring neurophysiological monitoring
5. Anaesthesia in non-theatre environments
6. Transfer of anaesthetised patients between different locations
7. Sedation in intensive care
8. Tubeless ENT procedures and rigid bronchoscopy
9. Thoracic surgery
10. Intracranial surgery
11. Procedures requiring sedation, e.g. endoscopy, cardioversion

ADVANTAGES OF TIVA

- Avoiding the use of nitrous oxide with its effect on air emboli and pneumothoraces, bone marrow suppression.
- Elimination of volatile agents along with their possible toxicity to the liver and kidney, potential rise in intracranial pressure, their effect on the uterus and possible environmental effects.
- Elimination of the need for accurately calibrated vaporizers.
- Superior quality recovery with less hangover.
- Propofol is a powerful anti-emetic.

DISADVANTAGES OF TIVA

- Pharmacokinetic and pharmacodynamic variability of response to the injected drug.
- Lack of ability to accurately assess actual blood levels.
- Variations in the haemodynamic state of the patient.

- Requirement for dedicated IV access, and risk of disconnection.
- Risk of accidental awareness.

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CROSS-REFERENCE

Awareness, Chapter 30

Neuraxial anaesthesia and analgesia continues to be useful in a wide range of subspecialties such as general, urological, gynaecological and orthopaedic surgery. In addition, it is a mainstay of obstetric anaesthetic practice. Benefits of neuraxial anaesthesia are shown in Table 29.1.

INDICATIONS

- *Surgery* – Central neuraxial blockade (CNB) can be used as the sole anaesthetic technique with or without sedation or to augment general anaesthesia for surgery below the level of T6.
- *Analgesia* – CNB has been shown to result in superior postoperative analgesia.
- *Obstetrics* – CNB is used in almost 90% of caesarean sections and in around 25% of nonoperative deliveries.
- Chronic and cancer pain.

CONTRAINDICATIONS

RELATIVE

- Condition causing fixed cardiac output
- Hypovolaemia
- Disorder of coagulation
- Systemic sepsis
- Raised intercranial pressure

ABSOLUTE

- Patient refusal
- Allergy to drugs used
- Local sepsis

DIFFERENTIAL BLOCKADE

Small diameter fibres (sensory and autonomic) are more readily blocked than large motor fibres as local anaesthetic penetrates them more easily. Thus, autonomic function, temperature and pain sensation are

Table 29.1 General potential benefits

- Superior pain relief when compared to systemic opioids.
- Reduction in thromboembolic complications after surgery.
- Reduced respiratory complications after surgery.
- Reduced cardiovascular complications after surgery.
- Enhanced recovery and early return of gastrointestinal function.
- Attenuation of perioperative stress response.
- Reduced surgical blood loss and blood transfusion requirement.

lost before motor function. The concentration of local anaesthetic agent used similarly influences the mode of block produced. Low concentrations produce analgesia with minimum motor blockade. High concentrations produce motor blockade in addition to analgesia. Sympathetic block is usually around two levels above the sensory block, which in turn is two levels higher than the motor block.

CENTRAL NEURAXIAL OPIOIDS

Opioids appear to have very little benefit when administered alone via the epidural route. When combined with low concentration local anaesthetic, however, there is a synergistic effect in terms of efficacy and duration of block. Highly lipid soluble opioids such as fentanyl or sufentanil may also have some systemic action. Opioids containing preservatives are neurotoxic and should be avoided. Commonly used opioids include fentanyl, sufentanil, diamorphine, morphine and pethidine, although the risk of late respiratory depression is increased with less lipid soluble drugs.

When administered either in combination with a local anaesthetic or alone, intrathecal opioids can produce prolonged postoperative analgesia. Morphine, diamorphine and fentanyl are the most frequently used. Intrathecal morphine has been shown to be efficacious at treating pain and reducing systemic opiate requirements in a wide variety of surgical specialties. It does, however, come with the risk of opioid induced ventilator impairment (OIVI). This peaks at 3.5–7.5 hours but can be seen up to 19.5 hours following administration, limiting its use.

Due to their more predictable ventilatory effects, fentanyl and diamorphine are more often used.

Systemic side effects can include pruritis, nausea, vomiting, urinary retention, sedation and respiratory depression and may occur with administration of opioids via either route but tend to be more common following morphine and intrathecal administration. Naloxone is an effective treatment.

OTHER ADJUNCTS TO LOCAL ANAESTHESIA

Epidural racemic ketamine combined with local anaesthetic may reduce overall opioid requirements without increasing the incidence of adverse effects. The efficacy of epidural clonidine is unclear.

Intrathecal clonidine and magnesium have been shown to increase duration of analgesia when combined with opiates with or without local anaesthetic.

Intrathecal and epidural epinephrine has been shown to improve the quality of analgesia while dexmedetomidine administered via either route has been shown to prolong analgesia when combined with local anaesthesic drugs.

CONTROVERSIES

DISORDERS OF COAGULATION

A disorder of coagulation can be caused by anticoagulant drugs, haematological disease, trauma, sepsis, uraemia, liver failure or massive transfusion. The risk of performing CNB on any patient with a disorder of coagulation is a continuum that runs from normal to very high risk. The presence of a coagulation disorder should always be considered a relative contraindication, but it may be that the performance of such a technique places the patient in less danger when compared to a general anaesthetic. Clinicians should be involved in multidisciplinary conversations and involve the patient in these decisions.

PRE-EXISTING NEUROLOGICAL DEFICIT

The 'double crush' theory suggests that patients with any pre-existing neurological injury may be at

increased risk for subsequent injury. Pre-existing neurological injury has historically been considered a contraindication to CNB, but evidence on the issue is limited. Decisions will be made on a case by case basis and if not specifically contraindicated, a documented detailed history, clinical examination and full and frank discussion of the risks and benefits are essential prior to CNB.

SYSTEMIC INFECTION

There may be an increased risk of bacterial meningitis or vertebral canal abscess in the presence of systemic infection. However, of all cases of bacterial meningitis identified in NAP3, none had evidence of pre-existing or local infection. Prolonged epidural catheterisation and compromised immunity would seem to be more prominent risk factors in the development of vertebral canal abscess.

PERFORMANCE OF BLOCK IN ANAESTHETISED OR SEDATED PATIENTS

The American Society of Regional Anaesthesia (ASRA) recommends that the use of ultrasound offers no reduction in risk when administering neuraxial anaesthesia to a deeply sedated or anaesthetised patient. General anaesthesia or deep sedation removes any ability for the patient to recognise and report warning signs that may indicate potential neurological injury and should be avoided unless specific conditions lead to a reconsideration of the likely risk and benefit. In paediatric patients, however, ensuring an immobile and cooperative child likely outweighs the risks posed by performing the procedure anaesthetised or deeply sedated.

EPIDURAL ANAESTHESIA

ANATOMY

- The epidural space lies between the dural sac and the periosteum lining the vertebral bodies extending from the foramen magnum to the sacral hiatus.
- Anteriorly, it is bounded by the posterior longitudinal ligament, posteriorly by the ligamentum flavum and laterally by the vertebral pedicles and intervertebral foramina.
- It communicates freely with the paravertebral space via the intervertebral foramina.
- It contains nerve roots, venous plexuses, fat and lymphatics.

TECHNIQUE

- Access can be gained through the ligamentum flavum via a median or paramedian approach with a loss of resistance technique using saline or air or through the sacrococcygeal membrane as a caudal block.
- Lumbar or thoracic regions are commonly used in adults while the caudal route is most common in children.
- As epidural local anaesthetic provides segmental analgesia, it should be sited at the level where anaesthesia is required, typically at the midpoint of the vertical incision.

ADVANTAGES

- Level of block can be controlled with titrated doses of local anaesthetic and site of epidural cannulation.
- Greater cardiovascular stability due to slow onset of block and the ability to titrate local anaesthetic via an epidural catheter.
- Block can be maintained and extended via an epidural catheter.

DISADVANTAGES

- Slow onset.
- Use of large volume of local anaesthetic increases the risk of toxicity.
- Risk of catheter migration, knotting or fragmentation.
- Increased risk of infection or haematoma formation with use of catheter.
- Subjective end-point may result in higher failure rate.

COMPLICATIONS

- Failure of intended technique (up to 22%).
- Hypotension (3%–10%).

- Respiratory depression (1%–15%).
- Dural tap (1%–3%. Operator dependent).
- Postdural puncture headache (75% after puncture with 16G needle).
- Vertebral canal abscess with CNB (1 in 115,000 in obstetric population, 1:24,000 in perioperative population and 1:47,000 overall). The use of epidural catheters and patients with compromised immunity seem to be at particular risk.
- Vertebral canal haematoma. (1:117,000. Risk appears much higher when catheter left in for postoperative analgesia and if anticoagulants are given.)
- Permanent neurological injury (between 1:6,000 and 1:12,000 in perioperative population and 1:160,000 in the obstetric population).

SPINAL ANAESTHESIA

ANATOMY

- Local anaesthetic is introduced into the cerebrospinal fluid (CSF), which surrounds the spinal cord within the subarachnoid space.
- In adults, the spinal cord typically ends at around L1/2 and so to reduce the risk of spinal cord trauma lumbar puncture should occur below this level.

TECHNIQUE

- Tuffier's line (intercristal line) corresponds roughly to the body of L4 in men and L5 in women and serves as a useful landmark.
- Dural puncture may be done via a median or paramedian approach. The spinal needle passes through skin, subcutaneous tissue, supraspinous ligament, interspinous ligament, ligamentum flavum, epidural space, dura and into the subarachnoid space where CSF is obtained.
- The extent of the block obtained is mainly determined by the baricity of the local anaesthetic agent, position of the patient and the concentration and volume of local anaesthetic injected.
- Hyperbaric solutions such as heavy bupivacaine, which contain 8% glucose, have a higher specific gravity in comparison to CSF and therefore tend to move downwards following injection.
- Hypobaric solutions, such as plain L-bupivacaine, have a lower specific gravity to CSF and tend to rise following injection.
- If a bilateral block is required, a predictable block is achieved by using a hyperbaric solution and positioning the patient seated or supine to alter the level.
- A unilateral block may be produced with hypo/hyperbaric solution with the patient in the lateral position.

ADVANTAGES

- Clear end point of technique, thus low failure rate.
- Rapid onset of block.
- Relatively predictable.
- Use of a small non-toxic dose of local anaesthetic.
- Rapid sacral block.
- Good muscle relaxation.

DISADVANTAGES

- Risk of haemodynamic instability resulting from higher denser block.
- Risk of postdural puncture headache.
- Single shot technique limits the ability to extend anaesthesia or analgesia intraoperatively.

COMPLICATIONS

- Postdural puncture headache (<1% following the use of small gauge Whitacre needle).
- Cardiovascular collapse.
- Pruritis, ventilatory deficiency, urinary retention, nausea and vomiting if opioid used as mentioned above.
- Vertebral canal abscess with CNB (1 in 115,000 in obstetric population, 1:24,000 in perioperative population and 1:47,000 overall) The use of epidural catheters and patients with compromised immunity seem to be at particular risk.

- No incidence of vertebral canal haematoma in 360,000 spinals recorded in NAP3.
- Permanent neurological injury (between 1:38,000 and 1:63,000 in perioperative population and 1:67,000 in the obstetric population).
- No incidence of vertebral canal haematoma in 360,000 spinals recorded in NAP3.

COMBINED SPINAL EPIDURAL

- Greater flexibility; the spinal block providing rapid onset while the epidural catheter can be used to extend the block.
- They have been found to have a disproportionately high complication rate.
- Possibly due to the fact that:
  - Combination of complication risks of spinals and epidurals.
  - Drugs infused into epidural space in presence of dural puncture.

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CROSS-REFERENCES

Regional blocks general principles, Chapter 29
Total spinal, Chapter 30

GENERAL PRINCIPLES OF PERIPHERAL REGIONAL ANAESTHESIA

Regional anaesthesia is the practice of using a local anaesthetic (LA) to block individual nerves, plexuses or the neuraxis. Indications are shown in Table 29.2, with contraindications in Table 29.3. Peripheral regional anaesthesia can be provided using a single injection peripheral nerve block (PNB) or continuous peripheral nerve blockade (CPNB) using an indwelling catheter. With the proliferation of enhanced recovery after surgery (ERAS) programs and the desire to allow early ambulation and avoid opioid analgesia, the use of peripheral regional analgesia is increasing. Other benefits are listed in Table 29.4. Simultaneously, ultrasound, needle, catheter and infusion pump technology required to perform these techniques is improving, making techniques more practical.

PREOPERATIVE PREPARATION

Prior to undertaking any regional anaesthetic technique, a full preoperative assessment needs to be undertaken with a full and frank discussion of the

Table 29.2 Indications

- Anaesthetic technique ± sedation.
- Analgesic technique in combination with general anaesthesia.
- Treatment of vascular insufficiency.
- Treatment of chronic and cancer related pain.

Table 29.3 Contraindications

Absolute	Relative
• Patient refusal.	• Coagulopathy (depends on degree of coagulopathy, technique and potential benefit).
• Infection at site of needle insertion.	
• Allergy to LA.	• Pre-existing neuropathy.

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Table 29.4 Benefits of regional anaesthesia

- Improved analgesia.
- Reduction in opioid use and associated adverse effects.
  - Nausea
  - Vomiting
  - Pruritus
  - Sedation
- Potentially decreased recurrence of some cancers.
- When compared to single shot PNB, CPNB results in improved pain control, decreased need for opioids, reduced nausea and improved patient satisfaction.

Table 29.5 Risks of regional anaesthesia (depending on technique and method used)

- Failure (10%–20%)
- Transient paraesthesia (8%–17%)
- Vascular puncture (2%–9%)
- Local infection from catheter techniques (0%–3%)
- Abscess from catheter techniques (0%–0.9%)
- Permanent motor or sensory nerve damage (1–5/10,000)
- Local anaesthetic systemic toxicity (LAST)
- Damage to adjacent structures, e.g. pneumothorax
- Unwanted nerve block, e.g. phrenic or recurrent laryngeal nerves

process of the block as well as the potential risks and benefits to the patient. Potential risks of peripheral nerve blockade are listed in Table 29.5. Alternative methods of anaesthesia/analgesia should be discussed in order to make a fully informed decision. This information should ideally be presented before the day of surgery. Consent should ideally be obtained by the person performing the block, and can be written or verbal (documented in patient notes).

BLOCK PERFORMANCE

- Trained assistance, a fully checked anaesthetic machine (as per AAGBI guidelines) and resuscitation equipment with 20% lipid emulsion are mandatory due to risk of complications such as LAST.
- Patient must be starved, consented and prepared for GA conversion due to possibility of failure of technique.
- IV access and full monitoring as per AAGBI standards.
- Block can be performed awake, sedated or anaesthetised.
- A sterile technique is used with 0.5% chlorhexidine in 70% alcohol used for skin antisepsis.
- Ergonomic positioning of patient, ultrasound, needle and practitioner to allow for ease of technique.

PERIPHERAL NERVE BLOCK NEEDLES

- Short bevelled blunt needles with side port for injection of LA and adjuncts. This design gives feedback as the needle passes through tissue planes and reduces the chance of neural and fascicular puncture.
- Insulated needles are Teflon-coated with an exposed tip. These use an electric current to locate plexus/nerve(s).
- Usually 20–22G and various lengths available (25–150 mm).
- Novel 'echogenic' needles are available which allow for improved needle visualisation at steeper angles of insertion.

ELECTRICAL NERVE LOCATORS

These are battery-operated devices designed to produce muscular contraction by stimulating motor fibres in a nerve or plexus. This reduces the need to seek paraesthesia as an end point to injection and therefore may reduce complications.

- Positive lead is connected to patient via an ECG electrode and negative lead is connected to needle. This arrangement results in a lower current being required.
- A complete circuit is created when the needle is in contact with the patient.
- The rheobase is the minimum current required.

- Chronaxie is the current duration required to stimulate a nerve at twice the rheobase.

$$Current\ required = Rheobase \times (1 + Chronaxie/Stimulus\ Duration)$$

- To propagate a nerve impulse, a threshold stimulus must be reached. This depends on the duration of the stimulus.
- Different nerves have differing chronaxie. In large Aα motor fibres (0.05–0.1 ms) this is much shorter than Aδ (0.15 ms) or C fibres (0.4 ms). A pulse width of 50–100 μs therefore allows for selective stimulation of motor fibres.
- Current is applied with a frequency of 1–2 Hz. Lower frequencies can be used if muscular contraction will cause pain (in trauma, for example) but the needle will need to be moved more slowly.
- The closer the needle tip is to the nerve, the lower the current required to stimulate the nerve.

$$Threshold\ current = Constant \times (Minimal\ current \times Distance\ from\ nerve^2)$$

- An initial high current (1–2 mA) is used until nerve stimulation is noticed. The current is then reduced until a maximal stimulation is obtained with minimal output (<0.4 mA) indicating that the tip of the needle is very close to the nerve.
- Persistence of stimulation at currents <0.2 mA could indicate intraneural placement, and so no injection should take place and the needle should be withdrawn.

STIMULATING CATHETERS

- These function like insulated needles but can be inserted to provide a continuous block.
- Made from insulating plastic material with a metallic wire inside which conducts the current to its exposed tip electrode. They should not be cut as then some of the catheter can become separated during insertion or removal.

- Usually, a nerve block needle is placed close to the nerve and then the stimulating catheter is introduced through it and the nerve stimulator is connected to the catheter. Stimulation through the catheter should reconfirm the catheter tip position in close proximity to the target nerve(s). The threshold currents needed with stimulating catheters may be considerably higher.

ULTRASOUND (US)

Transducer probes have various shapes and sizes that determine their field of view. The frequency of emitted sound waves determines how deep the sound waves penetrate and the resolution of the image. Higher frequency waves generate greater resolution but poorer penetration and so are used for superficial imaging. Lower frequency waves generate poorer resolution but with greater depth. These are used for deeper blocks. Linear probes are used for the majority of peripheral blocks. Curved lower frequency arrays are used for deeper nerve structures. Smaller footprint probes are useful for children and for certain very superficial blocks.

PNBs performed by US guidance alone, or in combination with electrical nerve locators, are superior in terms of improved sensory and motor block, reduced need for supplementation and less incidence of paraesthesia and vascular puncture. Using US alone shortens block performance time when compared with electronic nerve location, but when used in combination it increases performance time.

LA DRUGS

The choice of local anaesthetic drugs depends on:
- Speed of onset
- Potency
- Duration of action
- Sensory-motor differentiation
- Potential toxicity

As shown in Table 29.6.

Lidocaine and bupivacaine are widely used for regional blocks although due to its decreased cardio-toxicity levo-bupivaine is gaining popularity. Ropivacaine has a better sensory-motor differentiation than bupivacaine mainly affecting the

Table 29.6 Characteristics of local anaesthetic agents

Local anaesthetic	Onset	Duration	Relative potency	Toxicity
Lidocaine	fast	++	++	++
Prilocaine	fast	+	++	+
Bupivacaine	moderate	+++	++++	+++
L-Bupivacaine	moderate	+++	++++	++
Ropivacaine	moderate	+++	++++	++

sensory fibres. This makes it ideal for postoperative analgesia. Prilocaine is the agent of choice for intravenous regional anaesthesia.

ADJUVANTS

EPINEPHRINE

When 1 in 200,000 is used, the systemic side effects are reduced. In combination with lidocaine causes a 50% decrease in the peak plasma concentration when used for subcutaneous infiltration, but a 20%–30% decrease when used for intercostal or brachial plexus blocks. The blood concentration of bupivacaine and levo-bupicaine is reduced significantly less than that of lidocaine, and so is not normally combined with either of these two agents.

DEXAMETHASONE

In doses of 4–8 mg perineural dexamethasone has been shown to increase duration of long-acting LA agents by 7 hours. Recent studies have suggested, however, that IV dexamethasone administered at the same time is equally effective at extending block duration. As dexamethasone is licensed for IV and not perineural use, it is recommended that the IV route is preferred.

CLONIDINE

Can be used to prolong block duration and enhance analgesic action. Centrally it can cause sedation and hypotension; however, doses of up to 150 μg can be used peripherally with no side effects.

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CROSS-REFERENCE

Local anaesthetic toxicity, Chapter 30

HEAD AND NECK BLOCKS

ANATOMICAL OVERVIEW

Cutaneous supply to the head and neck is provided by the trigeminal nerve and the nerves of the cervical plexus. The cervical plexus consists of the ventral rami of C1-4. In the neck, it is deep to sternocleidomastoid muscle.

BLOCKS OF THE CERVICAL PLEXUS

ANATOMY

Anaesthesia can be provided for neck surgery by performing either superficial or deep cervical plexus block. Deep cervical plexus block is performed by injecting local anaesthetic at the transverse processes of C2-4. Superficial block is performed by infiltrating

local anaesthetic in a subcutaneous plane posterior to the sternocleidomastoid muscle.

INDICATIONS

- Carotid endarterectomy
- Thyroid surgery
- Clavicular surgery

TECHNIQUES

- US guided or landmark techniques.

SPECIFIC COMPLICATIONS

- Phrenic nerve block
- High/total spinal anaesthesia

OCCIPITAL NERVE BLOCK

ANATOMY

- Blockade of lesser occipital (branch of cervical plexus) and greater occipital nerve (branch of dorsal ramus of C2).

INDICATIONS

- Treatment of post-dural puncture headache.

TECHNIQUES

- Subcutaneous injection of local anaesthetic with landmark or US guidance.

SCALP BLOCK

ANATOMY

The scalp block aims to block seven nerves on each side of the head. These nerves contain branches of the trigeminal nerve, cervical plexus and cervical spinal nerve. They are supraorbital nerve, supratrochlear nerve, zygomaticotemporal nerve, auriculotemporal nerve, lesser occipital nerve, greater occipital nerve and greater auricular nerve.

INDICATIONS

- Craniotomy (awake and asleep).

TECHNIQUES

- Individual nerves are blocked with landmark-based infiltration of small volumes of local anaesthetic.

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CROSS-REFERENCE

Local anaesthetic toxicity, Chapter 30

LOWER LIMB BLOCKS

With the advent of enhanced recovery after surgery (ERAS) techniques for orthopaedic surgery, the motor block and consequent immobility and falls risk has led to lower limb blocks falling out of favour. They are still used in vascular surgery and some specific orthopaedic indications, and may be useful when a general anaesthetic and neuraxial technique are both contraindicated.

ANATOMICAL OVERVIEW

The lumbosacral plexus (T12-S4) provides sensory and motor supply to the lower limb.

LUMBAR PLEXUS BLOCKS

Otherwise known as the psoas compartment block or lumbar paravertebral block. The posterior lumbar

plexus block is the most reliable peripheral regional anaesthetic technique for providing anaesthesia to the lateral femoral cutaneous nerve, femoral nerve and obturator nerve.

ANATOMY

The lumbar plexus consists of the ventral rami of the first four lumber nerves (L1-4) as well as a contribution from the subcostal nerve (T12). It often sits within the body of the psoas major muscle at the level L4/5, but can be found completely posterior to it. The lumbar plexus gives rise to the following terminal branches: Ilioguinal nerve (T12-L1), iliohypogastric nerve (T12-L1), genitofemoral nerve (L1-2), obturator nerve (L2-4), lateral femoral cutaneous nerve (L2-3) and femoral nerve (L2-4).

INDICATIONS

- Hip/femoral trauma.
- *Hip/femoral surgery* – May be able to be used as sole technique if combined with proximal (i.e. Parasacral/Mansour) sciatic nerve block.

TECHNIQUES

Winnie, Chayen, Dekrey and Capdevila have all described different anatomical approaches. There is no difference in efficacy between the four approaches in adults; in children, Chayen's approach increases the risk of epidural spread.

- *Winnie approach* – Needle entry point at junction of the intercristal line and a line parallel to the midline passing thorough posterior superior iliac spine. Results in medial needle direction.
- *Chayen approach* – Needle entry point 3 cm caudal and 5 cm lateral to spinous process of LA (point at which intercristal line intersects with midline). Perpendicular needle insertion should make contact with transverse process of L5. Needle redirected cephalad and inserted 1–2 cm to locate plexus.

- *Dekrey approach* – Needle insertion is 3 cm lateral to spinous process of L3. Perpendicular needle insertion identifies transverse process of L3. Needle redirected caudally and inserted 1–2 cm to locate plexus.
- *Capdevila approach* – L4 identified and line drawn laterally to intersect with line parallel to midline passing through posterior superior iliac spine. Needle insertion is 2/3 away from midline on this lateral line. Perpendicular needle insertion locates transverse process of L4. Needle redirected caudally and inserted 1–2 cm to locate plexus.

Nowadays ultrasound guidance is commonly used.

SPECIFIC COMPLICATIONS

- Epidural spread of LA (9% > 90%)

FEMORAL/FI/3 IN 1 BLOCK

While the femoral nerve block aims to block just the femoral nerve, the '3 in 1' block and fascia iliaca compartment block both aim to block the three main terminal braches of the lumbar plexus via an anterior approach.

ANATOMY

The femoral nerve is deep to the fascia iliaca and lateral to the femoral artery.

INDICATIONS

- Analgesia for hip/femoral trauma.
- Analgesia for hip/femoral surgery.

TECHNIQUES

The '3 in 1' block was originally described by Winnie et al. in 1973 but has been shown to unreliably block the obturator nerve.

The fascia iliaca compartment block was described in paediatric patients by Dalens et al. in 1989. It has been shown to more reliably block the lateral femoral cutaneous nerve when compared to the '3 in 1' block but is equally as unreliable at blocking the obturator nerve.

SAPHENOUS/ADDUCTOR CANAL BLOCK

ANATOMY

The saphenous nerve lies anterior to the femoral artery deep to the sartorius muscle in the proximal thigh, and is the largest sensory branch of the femoral nerve.

INDICATIONS

Analgesia for knee surgery. Combined with a sciatic block it will provide complete anaesthesia below the knee.

TECHNIQUES

- Field infiltration technique
- Trans-sartorial approach
- Femoral paracondylar approach
- Paravenous approach
- Ultrasound guided adductor canal block

SCIATIC NERVE BLOCKS

ANATOMY

The sacral plexus has contributions from the lumbar plexus (L4-5) as well as the ventral rami of S1-4. The sacral plexus gives rise to the following terminal branches: Superior gluteal nerve (L4-S1), inferior gluteal nerve (L5-S2), sciatic nerve (L4-S1), pudendal nerve (S2-4).

The sciatic nerve leaves the pelvis via the greater sciatic foramen and enters the gluteal region where it passes roughly halfway between the greater trochanter and the ischeal tuberosity.

INDICATIONS

The sciatic nerve can be blocked for any lower limb surgery as it provides innervation to the hip, knee and ankle.

TECHNIQUES

- Parasacral approach as described by Mansour. A line is drawn between posterior superior iliac spine and ischial trochanter. Needle insertion 6 cm along this line from posterior superior iliac spine.
- Transgluteal (Labat approach or modified Winnie approach): This is the most widely used and probably the most reliable.
- Subgluteal (Raj approach).
- Anterior approaches.
- Transgluteal and subgluteal approaches can be conducted with ultrasound guidance.

POPLITEAL BLOCKS

ANATOMY

As the sciatic nerve descends in the thigh it bifurcates. This can occur at a variable distance above the popliteal fossa. This forms the common peroneal and the tibial nerves.

INDICATIONS

Anaesthesia for analgesia for foot/ankle surgery.

TECHNIQUES

Lateral or posterior approach with or without ultrasound guidance.

ANKLE BLOCK

ANATOMY

At the ankle the following five nerves provide the cutaneous supply to the foot: Saphenous nerve, superficial peroneal nerve, deep peroneal nerve, sural nerve and posterior tibial nerve.

INDICATIONS

Anaesthesia and analgesia for foot and ankle surgery.

TECHNIQUES

Ultrasound or landmark technique.

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CROSS-REFERENCES

Regional techniques general principles, Chapter 29
Trauma, Chapter 22

TRUNCAL BLOCKS

PARAVERTEBRAL BLOCK

ANATOMY

After leaving the intervertebral foramen, the thoracic spinal nerves enter the paravertebral space. This is a continuous space extending from T1-T12 bordered anterio-laterally by the parietal pleura, medially by the vertebral bodies and intervertebral discs and posteriorly by the transverse processes, ribs and costo-transverse ligament.

INDICATIONS

- Breast surgery
- Thoracic surgery
- Renal surgery
- Open cholecystectomy
- Rib fractures

TECHNIQUES

- Can be inserted using either a landmark or US guided technique.
- US guided approach can be either in plane or out of plane with the US probe in sagittal or transverse plane.
- Whichever technique is used, a volume of 15 mL local anaesthetic will provide sensory block over three dermatomal levels. If more extensive anaesthesia is required, multiple injections will be required.

SPECIFIC COMPLICATIONS

- *Hypotension* – secondary to epidural or intrathecal spread (4.6%)
- Vascular puncture (3.8%)
- Pleural puncture (1.1%)
- Pneumothorax (0.5%)

INTERCOSTAL NERVE BLOCKS

ANATOMY

After passing through the paravertebral space the ventral rami of T1-T12 continue as the intercostal nerves. They lie in the subcostal groove and distal to the angle of the rib run between the inner and innermost intercostal muscles. The lateral cutaneous branch originates at the mid-axillary line and so to provide sufficient anaesthesia local anaesthetic must be deposited proximal to this point.

INDICATIONS

- Breast surgery
- Thoracic surgery
- Renal surgery
- Open cholecystectomy
- Rib fractures

TECHNIQUES

A landmark or US guided technique can be used. In either case, needle insertion should be as close to the posterior border of the rib as possible to allow local anaesthetic spread to the subcostal groove.

SPECIFIC COMPLICATIONS

- Vascular puncture
- Pleural puncture
- Pneumothorax
- Local anaesthetic systemic toxicity due to high levels of systemic absorption from this particular block

PECS 1/PECS 2/SERRATUS PLANE BLOCK

ANATOMY

The medial pectoral nerve (C8-T1) is a branch of the medial cord of the brachial plexus. It runs deep to pectoralis minor to supply pectoralis minor and major. The lateral pectoral nerve (C5-7) is a branch of the lateral cord of the brachial plexus and runs in the plane in between pectoralis minor and major to supply pectoralis major.

INDICATIONS AND TECHNIQUES

The original description was of the Pecs 1 block. This involved US guided deposition of local anaesthesia in the plane in between pectoralis minor and major to block the lateral ± medial pectoral nerve(s). This was used for any surgery affecting pectoralis major (i.e. subpectoral implant, mastectomy)

The Pecs 2 was a modification that aimed to also block the axilla. It involves a Pecs 1 block and a further volume of local anaesthesia infiltrated either between the pectoralis minor and serratus muscle or between the serratus muscle and external intercostal muscle. It is also performed using ultrasound guidance. It was developed to extend Pecs 1 blocks to be able to be used for operations involving axillary clearances, by blocking the long thoracic nerve and intercostal nerves.

The serratus plane block is a further development on the above. It aims to deposit local anaesthetic superficial or deep underneath the serratus anterior.

SPECIFIC COMPLICATIONS

- Pleural puncture
- Pneumothorax

TAP BLOCK

ANATOMY

At the costal margin T7-T11 emerge in the plane between transversus abdominis and internal oblique. T12 and L1 also run in this muscular plane. At around half way along their course these nerves give off lateral cutaneous branches and so a posterior deposition of local anaesthetic maximises the chances of adequate anaesthesia.

Any regional technique to the nerves of the abdominal wall provides cutaneous anaesthesia only and no visceral analgesia.

INDICATIONS

- Sub-umbilical (below T10) surgery. Can be unilateral or bilateral.
- Subcostal TAP block has been described for anaesthesia T6-T10.

TECHNIQUES

- Landmark or US guided technique can be used.

SPECIFIC COMPLICATIONS

- Visceral trauma

ILIOHYPOGASTRIC/ILIOINGUINAL BLOCK

ANATOMY

The iliohypogastric nerve (T12-L1) runs in the TAP plane before piercing the internal oblique. The Ilioinguinal (L1) nerve perforates the tranversus abdominis at the level of the iliac crest.

INDICATIONS

- Inguinal hernia repair.

TECHNIQUES

- Ultrasound guided or landmark approach.

COMPLICATIONS

- Femoral nerve block and associated quadriceps weakness.

RECTUS SHEATH BLOCK

ANATOMY

Around the midline the anterior cutaneous branches (T7-T11) pierce the posterior rectus sheath. The space behind each rectus muscle and this posterior sheath is continuous and local anaesthetic can spread from the subcostal margin to the pelvic brim.

INDICATIONS

- Midline surgery of the abdomen.

TECHNIQUES

- Ultrasound guided or landmark approach. Continuous infusions are often used to provide prolonged postoperative analgesia.

COMPLICATIONS

- Visceral trauma.

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CROSS-REFERENCE

Local anaesthetic toxicity, Chapter 30

UPPER LIMB BLOCKS

Regional anaesthesia for the upper limb is predominantly used for orthopaedic and trauma surgery although knowledge of the anatomy and techniques can also be beneficial for plastic and even breast surgical procedures due to the brachial plexus supplying the pectoral muscles.

ANATOMICAL OVERVIEW

- Sensory and motor innervation to the upper limb is via the brachial plexus with the following exceptions:
  - Supraclavicular nerves (C3-4) provide cutaneous innervation to the 'cape' of the shoulder
  - Intercostobrachial nerve (T2) innervates proximal posterior and medial cutaneous innervation of the arm
- It originates from the ventral primary rami of C5-T1 and extends from the neck to the apex of the axilla.
- Can be described in terms of roots, trunks, divisions, cords or peripheral branches.

INTERSCALENE BLOCK

ANATOMY

- The interscalene grove is deep to the sternocleidomastoid muscle and in between the middle and anterior scalene muscles and contains the roots of the brachial plexus.
- The ventral rami of C5 and C6 unite to form the upper trunk near to the middle scalene muscle, C7 continues to become the middle trunk and the lower trunk is formed by the union of the ventral rami of C8 and T1.
- The phrenic nerve lies in between the sternocleidomastoid and anterior scalene muscle and is often affected by interscalene brachial plexus block. It can be identified and avoided if using ultrasound.
- Block should be avoided if patient has contralateral diaphragmatic pathology.

INDICATIONS

- Shoulder or humeral surgery.

TECHNIQUES

- US guided
  - In plane lateral to medial
  - Out of plane lateral to medial
- Landmark
  - Classic Winnie
  - Modified Winnie

SPECIFIC COMPLICATIONS

- Persistent neurological complications (0%–4.4%)
- Transient neurological symptoms (up to 18%)
- Hoarse voice (recurrent laryngeal nerve blockade) (31%)
- Dyspnoea (phrenic nerve blockade) (12%)
- Reduction in spirometric measure of respiratory function of 25%–32%
- Hemidiaphragmatic paresis reported in 100%

PERICLAVICULAR BLOCKS

ANATOMY

- After exiting the interscalene groove the trunks of the brachial plexus run over the first rib posterolateral to the subclavian artery. Here the brachial plexus is at its most compact.
- At this level the pleura is found within a few centimetres of the brachial plexus, hence the historically high incidence of pneumothorax associated with supraclavicular brachial plexus blockade.
- At the lateral border of the first rib the trunks become anterior and posterior divisions and lay posterolateral to the axillary artery as they emerge from under the clavicle.
- Anterior divisions and the superior and middle trunks become the lateral cord.
- Posterior divisions become the posterior cord.
- Anterior division of inferior trunk forms the medial cord.

- The cords of the brachial plexus are arranged around the second part of the axillary artery just medial to the coracoid process.

INDICATIONS

- Arm, elbow, forearm, wrist or hand surgery.

TECHNIQUES

- US guided
  - Supraclavicular
    - In plane lateral to medial
    - In plane medial to lateral
    - In plane oblique posterior to anterior
  - Infraclavicular
    - In plane cephalad to caudad approach
    - Vertical out of plane approach
- Landmark
  - Supraclavicular
    - Plumb-bob' approach
    - Subclavian perivascular approach
  - Infraclavicular
    - Coracoid approach
    - Lateral sagittal approach
    - Vertical approach
    - Raj technique

SPECIFIC COMPLICATIONS

- Vascular puncture 6.6%.
- Horner syndrome 12%.
- Hemidiaphragmatic paresis in 50% not associated with dyspnoea or decrease in respiratory function.
- Pneumothrax 0.5%–6%. Redcued to 0.1% or less with US guided techniques.

AXILLARY BLOCK

ANATOMY

- The terminal branches of the brachial plexus are derived from the three cords and are arranged around the axillary artery in the axilla, surrounded by the biceps, coracobrachialis and triceps muscles.

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- The musculocutaneous nerve branches of the lateral cord proximal to this point is often located within the body of coracobrachialis.

INDICATIONS

- Wrist or hand surgery

TECHNIQUES

- US guided
  - In plane approach
  - Out of plane approach
- Landmark
  - Perivascular technique
  - Transarterial technique

SPECIFIC COMPLICATIONS

- Infection in 3%–4% if catheter placed

The branches of the brachial plexus can also be blocked at the mid-humeral, elbow, forearm or wrist level. These blocks may be useful to augment a partially effective block but have limited use as they do not provide cover for a tourniquet and multiple blocks are required for hand or wrist surgery.

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CROSS-REFERENCES

30

Management problems

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ALLERGIC REACTIONS

Allergic reactions are unpredictable and may be potentially life-threatening. Prompt recognition and treatment are essential. The incidence is between 1:10,000 and 1:20,000 anaesthetics. The estimated mortality has declined in recent years, and is now thought to be less than 2%.

When associated with anaesthesia, the causative agents are commonly:

- Neuromuscular blockers, especially suxamethonium and rocuronium 50%–70%
- Latex (notably risen in recent years) >10%
- Antibiotics
- Induction agents
- NSAIDS
- Colloids
- Opioids
- Radiocontrast media and dyes

PATHOPHYSIOLOGY

Allergic and non-allergic anaphylactic reactions are clinically indistinguishable (Figure 30.1). The terms refer to the triggering pathway responsible for the final common end-point, i.e. the release of potent circulating inflammatory mediators from degranulated mast cells and basophils.

Anaphylactic reactions classically involve the cross-linking of two adjacent IgE antigen-specific antibodies to the mast-cell surface causing a type-I hypersensitivity reaction; however, IgG and complement can also be implicated. Non-IgE mediated anaphylaxis is typically less rapid in onset than IgE-mediated anaphylaxis.

Reactions caused by mast cell degranulation due to other triggering pathways are termed 'non-allergic'. The majority of the reactions caused by anaesthetic drug mixtures occur in this fashion, causing 'aggregate anaphylaxis'.
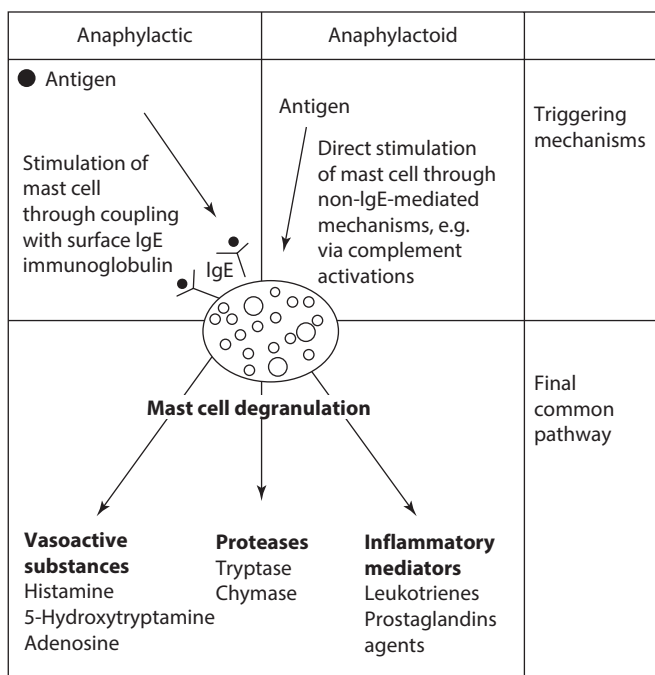
Figure 30.1 Immune mechanisms of allergic and non-allergic anaphylactic reactions. The 'antigen' refers to the drug or substance responsible for activating the triggering mechanism. In some cases, for example, penicillin, binding to a hapten (carrier protein) is necessary prior to coupling with surface membrane IgE antibody.

In many cases, previous sensitisation to the antigen or similar compound cannot be demonstrated.

SIGNIFICANT CLINICAL FEATURES

When associated with anaesthesia symptoms often occur rapidly but may not manifest until after 30 minutes in the case of anaphylaxis to latex. Profound hypotension or cardiac arrest has been reported in up to 80% of cases; however, this figure may be unreliable due to reporting bias. Hypoxia may be present due to inability to ventilate, bronchospasm or laryngeal oedema. Urticarial or erythematous rashes feature more prominently outside the hospital.

PRIMARY TREATMENT OF SUSPECTED ANAPHYLAXIS (AAGBI GUIDELINES 2009)

- Discontinue administration of suspected agent.
- Assess and maintain airway, check breathing and circulation.
- Give 100% oxygen.
- Call for help.
- Elevate legs to increase venous return.
- If necessary, begin cardiopulmonary resuscitation.
- Give epinephrine:
  - either IV 0.5 mL of 1:10,000, repeat as necessary
  - or IM 0.5 mL of 1:1000 (500 mcg) repeat as necessary
- Start intravenous fluid resuscitation with colloid or crystalloid.

SECONDARY TREATMENT

- Chlorpheniramine 10 mg IV
- Hydrocortisone 200 mg IV
- Epinephrine infusion (0.05–0.1 mcg/kg/min)
- 10–20 mL/h of 1 mg in 50 mL solution for a 70 kg patient
- Consider sodium bicarbonate

- Consider bronchodilators (aminophylline, salbutamol)
- Airway evaluation (before extubation)
- Arrange transfer to ICU if necessary

FURTHER INVESTIGATION

- Investigations should not be undertaken at the expense of resuscitation.
- Maintain a detailed written record of events.
- Send blood for mast cell tryptase:
  - As soon as possible after the onset.
  - 1–2 hours later
  - 24 hours later
- In anaphylaxis, mast cell tryptase peaks 30–90 minutes after the onset and returns to baseline after 6 hours.
- The anaesthetist is responsible for providing advice for the patient and for ensuring appropriate follow-up takes place.
- Seek advice from a specialist in allergy and clinical immunology as further investigation is required.
- See useful addresses for committee on safety of medicines, medic-alert and Immunology Society websites.
- Commonly skin-prick tests are performed with a range of drugs 4–6 weeks after the acute episode and 5 days after discontinuation of antihistamines in order to minimise the risk of false-negative testing. If clinical suspicion persists despite negative skin-prick testing, intradermal testing may be conducted.

CLASSIFICATION OF ANAPHYLAXIS

Anaphylactic reactions are classified according to their severity:

- *Grade 1* – Rash, erythema or swelling.
- *Grade 2* – Unexpected hypotension (not severe) and/or bronchospasm ± Grade 1 features.
- *Grade 3* – Severe hypotension, and/or severe bronchospasm, and/or airway compromise due to swelling ± Grade 1 features.
- *Grade 4* – Ardiac arrest.
- *Grade 5* – Death.

Grade 1 and 2 anaphylaxis are not life-threatening, whereas Grade 3 and above are termed 'severe anaphylaxis'.

NAP-6

At the time of writing, data is being collected for the sixth UK National Audit Project, which aims to prospectively record all aspects of severe perioperative anaphylaxis in all hospitals in the UK. This should provide more accurate information regarding the incidence and outcome of severe anaphylactic reactions.

LATEX ALLERGY

There is an increasing incidence of latex allergy especially in population groups with previous repeated exposure to latex, e.g. patients with a history of numerous surgical procedures or urethral catheterization. In spina bifida patients, the incidence may be in excess of 60%. The incidence in healthcare workers is about 10% and in the general population about 0.7%.

Other important associations:

- Reactivity to party balloons, barrier contraceptives, dentists' gloves
- Cross-reactivity to certain foods (banana, kiwi, chestnut, avocado)
- Positive investigation (serological tests, skin prick, intradermal testing)

Sensitivity to latex may vary from contact dermatitis to anaphylaxis. Anaphylaxis may occur via any route but is more likely after intravenous or direct mucosal exposure.

MANAGEMENT OF THE PATIENT WITH LATEX ALLERGY

- Provide a latex-free environment.
- Preoperative immunological testing may be helpful in selected cases but false negatives occur.
- Consider premedication with chlorpheniramine, ranitidine or hydrocortisone.

- Put the patient first on the morning operating list.
- Communicate risks to other anaesthetic, nursing and surgical staff.
- Check *all* equipment is latex-free, especially gloves (all staff), facemasks, reservoir bag, syringes and drip injection ports.
- Departments should have a management protocol and a designated box of latex-free equipment available.
- Monitor postoperatively for delayed reactions for up to 1 hour.
- Vigilance and full resuscitation resources are essential.

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Mills ATD, Sice PJA, Ford SM. (2013). Anaesthesia-related anaphylaxis: Investigation and follow-up. *Contin Educ Anaesth Crit Care Pain* **14**: 57–62.

USEFUL WEBSITES

Association of Anaesthetists of Great Britain and Ireland guidelines: aagbi.org/publications/guidelines.htm

Medicines and Healthcare Products Regulatory Authority (MHRA) yellow card reporting scheme: yellowcard.mhra.gov.uk

British Society of Allergy and Clinical Immunology: www.bsaci.org

Medic-Alert Foundation: www.medicalert.org.uk
NAP 6: www.nationalauditprojects.org.uk/NAP6home

AMNIOTIC FLUID EMBOLISM (AFE)

Amniotic fluid embolis (AFE) is a syndrome characterised by hypoxia, hypotension and coagulopathy, which typically occurs during or shortly after labour. The pathological process remains unclear, as the entry of amniotic fluid into the circulation occurs almost universally in labour, and animal models suggest that this is innocuous. It may be the composition of the amniotic fluid, or an abnormal immunological reaction to an otherwise normal event which determines the severity of the pathological events comprising the syndrome. There are similarities to anaphylaxis and septic shock and the same mediators may be involved. Disruption of the foetal–maternal barrier during delivery allowing foetal and infectious tissue to enter the maternal circulation may be the trigger resulting in activation of pro-inflammatory mediators, coagulation cascade and disseminated intravascular coagulation. There is also transient hypertension (systemic and pulmonary) and reduced uterine tome followed by myocardial depression, pulmonary injury and CNS injury.

AFE has an incidence of about 2:100,000 pregnancies and 20%–60% of women who develop AFE do not survive. Most commonly, AFE occurs antenatally, but may present several hours after delivery.

The proposed risk factors (Table 30.1) remain controversial due to conflicting evidence. It has also been suggested that amniotic fluid containing meconium, the presence of a male foetus and ethnicity influence the severity of the response to AFE.

Table 30.1 Risk factors for AFE

- Age over 35 years
- Multiple pregnancy
- Placenta praevia and abruption
- Induction of labour
- Cervical trauma
- Operative delivery

CLINICAL PRESENTATION

The commonly described acute features of AFE are

1. Cardiovascular collapse
2. Respiratory compromise
3. Depressed conscious level
4. Convulsions
5. Coagulopathy
6. Uterine atony
7. Fetal distress

The classical presentation is at or around delivery with symptoms and signs of cardiorespiratory collapse (Table 30.2). From case reports where patients have survived long enough to have a pulmonary flotation catheter inserted, left ventricular failure, elevated left ventricular end diastolic pressure and elevated pulmonary capillary wedge pressure were seen. These concur with clinical and X-ray pictures of pulmonary oedema, which occurs in many of the patients who survive the initial cardiopulmonary collapse. Whether intrapulmonary shunting, pulmonary hypertension and right heart failure precede or even precipitate, the documented left heart failure is unproven. Evidence exists for release of a potent vasoconstrictor (possibly endothelin) in AFE, which may be responsible for pulmonary, coronary and even systemic vasoconstriction.

Convulsions due to cerebral hypoxaemia or hypotension may delay the diagnosis by confusion with eclampsia. Coagulopathy is the other main feature of AFE. It may be obvious as severe vaginal haemorrhage or as severe haemorrhage during caesarean section and may be compounded by uterine atony. Activation of the clotting cascade by foetal antigens or trophoblastic tissue exerting a thromboplastin-like effect may possibly be the trigger. Occasionally,

Table 30.2 Cardiorespiratory symptoms and signs

- Dyspnoea
- Hypoxia
- Hypotension
- Convulsions
- Bronchospasm
- Arrhyhmias
- Cardiac arrest
- Fetal bradycardia

AFE has to be included in the differential diagnosis when abnormal bleeding occurs from wounds or intravenous access sites without preceding evidence of AFE. In up to 80% of cases of AFE there is evidence of coagulopathy, and this should be anticipated if the diagnosis of AFE has been made.

DIFFERENTIAL DIAGNOSIS

- Local anaesthetic toxicity
- Transfusion reaction
- Other emboli
- Septic shock
- Myocardial infarction
- Anaphylaxis
- Eclampsia
- Placental abruption
- Uterine rupture

TREATMENT

RESUSCITATION

- Avoid aorto-caval compression by lateral tilt.
- Administer 100% oxygen; where appropriate intubate and ventilate.
- Obtain large-bore intravenous access. Commence fluid, vasoconstictors and/or inotropes as indicated.
- Full monitoring including an arterial line.
- Site a central venous cannula.
- Manipulate fluids and cardiovascular drugs according to haemodynamic measurements; diuretics if evidence of pulmonary oedema; an oesophageal Doppler probe may be useful.
- Deliver the fetus if still in utero. Approximately 79% fetal survival has been reported but survival of a neurologically intact infant is around 39%.
- Transfer to the ITU for further management.
- Commence cardiac arrest algorithm with lateral tilt where appropriate.

CORRECTION OF COAGULOPATHY

- Take blood for a full coagulation screen.
- Near patient testing such as ROTEM or TEG will help to direct blood product

administration, and should be used if available.
- Inform the haematology laboratory.
- Give fresh frozen plasma, platelets, cryoprecipitate and concentrated red cells, depending on clotting results and blood loss. Haematological advice should be sought before the use of serine protease inhibitors or heparin.
- Anticipate uterine atony and give oxytocics as indicated.

ADDITIONAL CONSIDERATIONS

- Cardiopulmonary bypass and pulmonary thrombectomy may be required.
- Nitric oxide and inhaled prostacyclin may be needed to treat refractory hypoxaemia.
- Extracorporeal membrane oxygenation and intra-aortic balloon counterpulsation have been used.

CONFIRMATION OF DIAGNOSIS

To date, no definitive laboratory test exists for either the diagnosis or exclusion of AFE. It therefore remains a clinical diagnosis of exclusion.

The presence of amniotic fluid debris, squames, lanugo hair and mucin on sectioning of lung specimens postmortem confirms the diagnosis of AFE.

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CROSS-REFERENCES

Cardiopulmonary resuscitation, Chapter 30
Caesarean section, Chapter 12

AWARENESS

Accidental awareness under general anaesthesia is a situation in which the patient remembers part or all of the anaesthetic or surgical procedure despite the intention to administer general anaesthesia. Recall of specific words or sounds in the operating room may distinguish awareness from hallucination or dreaming.

A clinically relevant proportion of awareness is accompanied by pain, and may give rise to postoperative psychological trauma and litigation.

Awareness may be explicit, when the patient recalls intraoperative events spontaneously or implicit, where subconscious learning during anaesthesia surfaces as a behavioral change, sometimes after a considerable time delay. Implicit recall has been studied using postoperative hypnosis or recall of key words or phrases. A number of events may be recalled (Table 30.3). The sensation of paralysis and auditory stimuli are most frequently recalled, especially negative comments regarding the patient's condition or appearance.

INCIDENCE

The 5th UK National Audit Project (NAP 5), which relied on patient-reporting, calculated an overall incidence of 1:19,000 general anaesthetics. Amongst cases where neuromuscular blocking drugs (NMBDs) were used, the incidence was 1:8000, and where NMBDs were not used the incidence was 1:136,000. The two clinical contexts of particularly increased incidence were cardiac surgery (1:8600) and caesarean section (1:670). The patient-reported incidence in NAP 5 is substantially lower than the incidence reported in studies using the Brice protocol (1:600), suggesting that many cases of awareness go unreported.

IDENTIFICATION

- Increased BP, heart rate, sweating, and/or lacrimation during the procedure.
- Depth of anaesthesia monitoring may indicate light anaesthesia (e.g. BIS™ >60 indicates increased probability of awareness).
- Structured postoperative interview (e.g. Brice protocol), avoiding leading questions, may actively identify cases of awareness.
- The hand-written anaesthetic record is limited as a method of determining why awareness and recall have occurred.

SEQUELAE

- Sleep disturbances, e.g. nightmares
- Flashbacks
- Anxiety
- Increased fear of anaesthesia

CAUSES

INDUCTION

- Intubation immediately after injection of intravenous agent (i.e. too early) may lead to awareness of intubation.

Table 30.3 Events recalled following awareness

Event	%
Inability to move	42
Inability to communicate	41
Hearing noises/voices	37
Awareness of tracheal intubation	21
Pain	18
Inability to breathe or suffocation	11
Movement or being moved	9
Visual sensations	3
Dream-like experiences	5

Source: Adapted From Pandit JJ, Cook TM (eds), NAP 5, 5th National Audit Project of the Royal College of Anaesthetists and the Association of Anaesthetists of Great Britain and Ireland, Accidental Awareness during General Anaesthesia in the United Kingdom and Ireland. London: RCoA and AAGBI, 2014.

- Undue delay before intubation (e.g. waiting for the relaxant to take effect, problems with intubation) may result in the intravenous induction agent wearing off.
- Rapid sequence induction and the use of thiopental were associated with an increased risk in NAP 5.

BETWEEN INDUCTION AND SURGERY

- Any delay in transfer to the operating room may allow the blood concentration of the intravenous agent to decay before an inhalational agent has reached anaesthetic levels, leading to awareness of incision.

DURING SURGERY

- Anaesthetic machine or circuit faulty or damaged.
- Low minute-volume settings on some electrically driven ventilators may lead to entrainment of air.
- Oxygen bypass tap left on.
- Exhausted vaporizer.
- Failure to eliminate air from a closed circuit.
- Exhausted, disconnected or malfunctioning TIVA syringe pump.
- Intentional administration of low doses of anaesthetic agent (e.g. to minimize cardiovascular instability or in obstetric anaesthetic practice).

DURING EMERGENCE

- Inadequate reversal of relaxant.
- Discontinuing anaesthetic agent too early.

PATIENT FACTORS

- Patient resistance to anaesthetic agents (e.g. due to concomitant medications or possibly genetic predisposition) may occur. Up to 11% of patients with awareness have a previous history.
- Younger adults, females and obese patients are of increased risk.

PREVENTION

- Know exactly how your anaesthetic machine and associated equipment work and check it all before use.
- Flush circle with a high fresh gas flow for the first 5 min.
- Periodically check vaporizer levels.
- Monitor end-tidal volatile concentrations. A concentration of >0.7 MAC (age adjusted) is often proposed as the threshold above which awareness is less likely to occur.
- Enable the low end-tidal volatile concentration alarms.
- The AAGBI recommends the use of a depth-of-anaesthesia monitor when relaxants and/or TIVA techniques are used.
- Remember that patients differ considerably in their anaesthetic requirements.
- Ensure that relaxants have been adequately reversed prior to extubation.

MANAGEMENT

- If awareness is suspected intraoperatively, increase dose of anaesthetic agent, administer analgesia and reassure the patient that they are safe.
- Postoperative interview.
- Referral to psychologist for counseling may be required.
- Incident reporting as per institutional policy.

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BLOOD TRANSFUSION

Concerns about disease transmission and potential shortages in a safe supply have led to a more critical approach to the use of blood and blood products. Two-thirds of all transfusions are given during the perioperative period. Commonly used blood products are listed in Table 30.4.

MASSIVE TRANSFUSION

Blood transfusion remains the treatment of choice where there is severe acute blood loss, e.g. major trauma, ruptured aortic aneurysm, major obstetric haemorrhage. In an emergency, group O Rhesus-negative or type-specific packed cells may be given. Haemolysis will occur in 3% of these 'transfusion episodes'. The use of FFP (15 mL/kg) and platelets (target platelet count 75×10^9/L) should be considered early in the management of a patient with massive haemorrhage. Cryoprecipitate or fibrinogen concentrate should be administered to maintain a fibrinogen level of >1.5 g L^{-1} (>2 g L^{-1} in obstetrics). Optimal use of blood products is guided by a combination of laboratory tests and the use of near patient testing devices such as the thromboelastography (TEG or ROTEM). Continuing blood loss will be exacerbated by hypocalcaemia, acidosis and hypothermia.

When bleeding is controlled and the patient stable, consideration should be given to starting thromboprophylaxis, as these patients subsequently become hypercoagulable, and are therefore at risk of subsequent thromboembolism.

RISKS OF TRANSFUSION

There are risks associated with all homologous transfusions. Mild reactions are relatively common (up to 3% of cases), but life-threatening complications are rare.

ACUTE LIFE THREATENING COMPLICATIONS ARE DUE TO

- Red cell incompatibility (1:40,000)
- Transfusion related acute lung injury (TRALI) (1:1,200–190,000)
- Anaphylaxis (1:20,000–50,000)

Red cell incompatibility occurs when the recipient has antibodies to antigens on the donor red cells. The most important antigens are ABO and Rhesus.

Table 30.4 Commonly used blood products

Product	Use
Packed red cells	Increase oxygen delivering capacity of the blood – acute or chronic anaemia
Platelets	Prevention or treatment of haemorrhage in patients with thrombocytopenia
Fresh frozen plasma (FFP)	Replacement of coagulation factors
	Thrombotic thrombocytopenia
	Reversal of warfarin when Prothrombin Complex Concentrate not available
Cryoprecipitate	Hypofibrinogenaemia (as can occur in massive transfusions)
	Haemophilia – when factor concentrates not available
Fibrinogen concentrate	Hypofibrinogenaemia – fibrinogen can be replaced more rapidly and reliably using fibrinogen concentrate,
Prothrombin complex concentrate (PCC)	Product of choice for reversal of the effect of warfarin (Contains Factors II, VII, IX and X)
Recombinant factors VIIa, VIII and IX	Haemophilia

If a second transfusion is required more than 3 days after the first, a further cross-match should be carried out in case antibodies have developed. A severe ABO incompatibility reaction may be provoked by as little as 30 mL of transfused blood. This is usually due to clerical error. Death from DIC occurs in 1 in 1000 cases.

While other blood components do not have antigens, those containing plasma may have a small amount of antibodies so group specific is preferable.

TRALI is non-cardiogenic pulmonary oedema following the administration of blood products and is clinically indistinguishable from acute respiratory distress syndrome. The aetiology is not fully understood but is thought to be related to leucocyte and neutrophil antibodies in donor plasma. Therefore, 'plasma rich' blood products (FFP and platelets) represent a much higher risk than 'plasma poor' blood products (red cells and cryoprecipitate).

INFECTION

- Viral hepatitis
- Human immunodeficiency virus (HIV)
- Cytomegalovirus (CMV)
- Human T lymphocytic virus (HLTV)
- Parvovirus
- Malaria
- Bacterial contamination
- Variant Creutzfeldt-Jakob disease (vCJD)

All blood in the UK is leucodepleted in an attempt to decrease the potential risk from transfusion-transmitted CMV, HTLV (causes T-cell leukaemia and T-cell lymphoma in adults) and vCJD. For patients at particularly high risk from CMV (Table 30.5), CMV negative blood is available.

Other measures to reduce the risks of transmission of vCJD include importing plasma for patients born after 1996 (when public health measures are assumed to have halted dietary transmission of vCJD), reducing the plasma content of red cells and platelets and exclusion of donors who may be at increased risk of carrying the disease (e.g. have received blood transfusions).

Table 30.5 Indications for CMV-negative blood and irradiated blood

- Patients who should have CMV-negative blood
  - CMV antibody-negative pregnant women
  - Intrauterine transfusions
  - CMV antibody-negative patients with HIV
  - CMV negative recipients of allogenic stem cell grafts
  - Neonates and children under 1 year
- Patients who should have irradiated blood
  - Congenital immune deficiency with defective cell mediated immunity (e.g. DiGeorge syndrome, Wiskott Aldrich syndrome, purine nucleoside deficiency)
  - Transfusions from first- or second-degree relatives
  - Intrauterine transfusions
  - Neonatal exchange transfusions
  - Stem cell transplant recipients
  - Patients receiving purine analogues
  - Any granulocyte transfusion
  - Hodgkin disease
  - HLA selected platelet units

IMMUNOMODULATION

There is a weak association between perioperative transfusion and an increased incidence of recurrence of colorectal carcinoma. There may also be an increased risk of infection due to immunosuppression following transfusion.

TRANSFUSION-ASSOCIATED GRAFT-VERSUS-HOST DISEASE (TA-GVHD)

This very rare complication is associated with white blood cells in donor blood. Even if blood is leucodepleted, the small residual amount of white blood cells left can trigger a reaction which can be fatal in susceptible individuals (Table 30.5). These patients should know about their condition and carry a warning card. Gamma irradiation of the blood inactivates lymphocytes and prevents TA-GVHD.

PROBLEMS ASSOCIATED WITH LARGE VOLUME TRANSFUSION

- Transfusion-associated circulatory overload (TACO)
- Dilutional thrombocytopenia
- Dilution of coagulation factors
- Hyperkalaemia
- Hypothermia
- Hypocalcaemia

These complications, if uncorrected, can contribute to impaired haemostasis and further blood loss.

METHODS FOR REDUCTION OF EXPOSURE TO HOMOLOGOUS BLOOD

- Intraoperative haemodilution
- Intraoperative controlled hypotension
- Autologous transfusion
- Intraoperative red cell salvage
- Use of synthetic oxygen transport media
- Positioning of patient to reduce surgical bleeding

PHARMACOLOGICAL APPROACHES

- Recombinant erythropoeitin
- Antifibrinolytics
- Recombinant factor VIIa (not recommended except in haemophilia)

Intraoperative haemodilution is widely available for avoiding homologous blood transfusion and its attendant problems. It is based on the premise that, whilst maintaining normovolaemia, the maximum oxygen delivery (DO_2) rises to 110% of normal at a haematocrit of 30%. This effect relies on a reduction in blood viscosity. The increase in DO_2 occurs without an increase in energy consumption. There may be an increased incidence of myocardial ischaemia in patients with coronary artery disease.

Erythropoeitin may be used to increase preoperative haemoglobin concentration, and possibly increase the yield of autologous blood donation. Stroma-free haemoglobin and perfluorochemicals remain a potential (but currently unavailable) method for avoiding homologous blood.

Tranexamic acid, an anti-fibrinolytic agent, may reduce bleeding and transfusion requirements in a variety of surgical specialties including cardiac surgery and major trauma. Aprotinin was primarily used in cardiac surgery and shown to be very effective in reducing transfusion requirements, but had its license voluntarily withdrawn because of concerns about outcomes in patients undergoing coronary artery bypass grafts. It is still available on a named patient basis for procedures where there is a high risk of bleeding.

A recent Cochrane review examining the role of recombinant factor VIIa as a haemostatic agent in patients without haemophilia concluded that it should not be recommended as either prophylaxis or therapy for major haemorrhage due to a lack of evidence of benefit and incidence of arterial thrombosis.

Moderate or severe preoperative anaemia is present in approximately 10% of surgical patients, and is predictive of poor clinical outcomes. However, it is not yet known whether correction of anaemia mitigates this effect, and by what methods if any this should be accomplished.

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CARDIOPULMONARY RESUSCITATION

Cardiorespiratory arrest describes the cessation of cardiac output and normal breathing. Cardiac arrest may be primary – due to dysrhythmia or severe myocardial failure, or secondary – due to hypoxaemia (respiratory arrest), electrolyte imbalance, etc. There are four fundamental 'rhythms' of cardiac arrest:

- 'Shockable' rhythms:
  - Ventricular fibrillation (VF)
  - Pulseless ventricular tachycardia (VT)
- 'Non-shockable' rhythms:
  - Asystole
  - Pulseless electrical activity (PEA)

FACTORS AFFECTING SURVIVAL

Survival is most likely when:

- The rhythm is VF or VT.
- The arrest is witnessed.
- Basic life support is started immediately.
- Defibrillation, if indicated, is given promptly.

BASIC LIFE SUPPORT

All medical, nursing and other hospital staff, as well as trained members of the general public, should be able to perform basic life support (BLS). The BLS algorithm is shown in Figure 30.2.

Unresponsive and not breathing normally

↓

Call 999 and ask for an ambulance

↓

30 Chest compressions

↓

2 Rescue breaths

↓

Continue CPR 30:2

↓

As soon as AED arrives switch it on and follow instructions
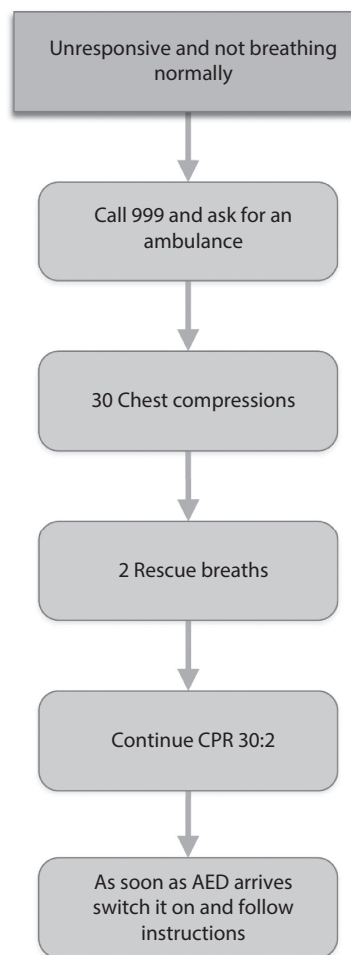
Figure 30.2 Basic life support algorithm. (From Resuscitation Council [UK]. *Adult Basic Life Support*. Resusitation Guidelines 2010. London: Resuscitation Council [UK], 2010.)

IMPORTANT POINTS

- Check that there is no danger to you or to the casualty before starting resuscitation.
- If the casualty is unresponsive first shout for help, then open the airway using a head tilt, chin lift, and then use the look, listen and feel approach for a maximum of 10 seconds to open the airway and assess for signs of life (presence of a central pulse and respiratory effort). Note that in the unconscious patient with suspected cervical spine injuries a jaw thrust should be

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employed instead of a head tilt, chin lift to prevent further injury.

- In unconscious patients foreign bodies may be removed from the airway under direct vision using Magills forceps or suction. Finger sweeps should be avoided as they may promote trauma, laryngeal spasm, worsen the obstruction or the patient may bite! In the conscious patient who has a foreign body airway obstruction, use the choking algorithm shown in Figure 30.3.

- If signs of life are absent, commence BLS immediately, starting with chest compressions. Shout for help and ask for a defibrillator if one is available. Request the attendance of the cardiac arrest team (all UK hospitals use the 2222 emergency number). Survival is very unlikely without advanced life support (ALS).

- The ratio of chest compressions to ventilations for adult patients should be 30:2 and provided at a rate of 100–120 chest compressions per minute.

- Good quality chest compressions are paramount and interruptions in chest compressions should be avoided if possible.

- Ventilation is ideally performed with a bag-valve-mask with reservoir bag and supplemental oxygen, but pocket mask or mouth-to-mouth techniques may be used whilst a bag-valve-mask is obtained. High concentration oxygen should be used as soon as it is available.

- Ventilation by mouth to mouth may offer unknown risks to the rescuer; consider continuous chest compressions until a suitable barrier device becomes available.

- The techniques of CPR cannot be learnt from a book but only from a properly supervised training session.
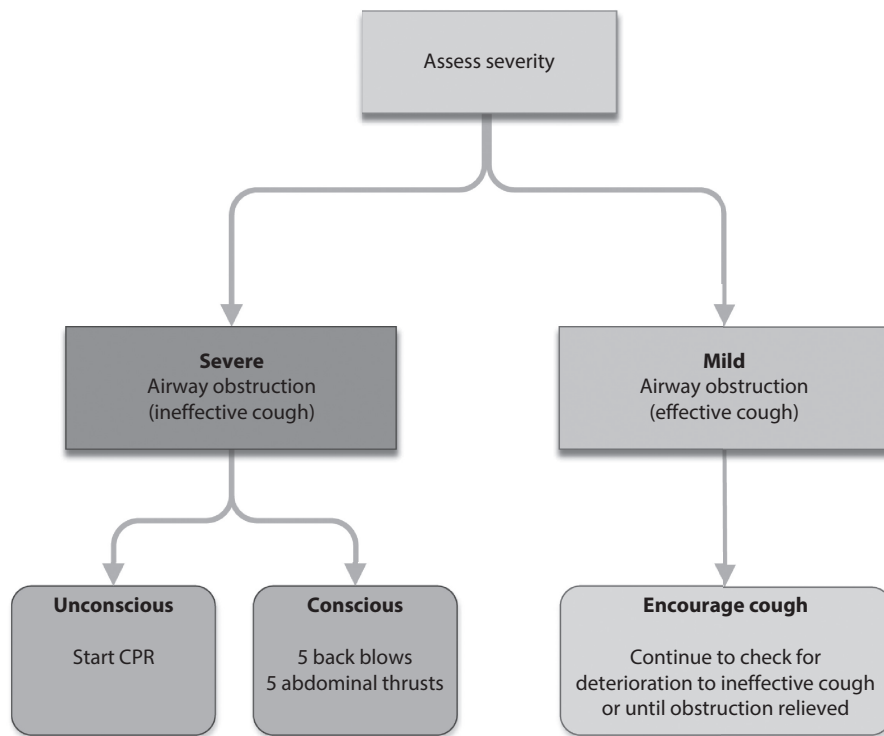
Figure 30.3 Adult choking algorithm. (From Resuscitation Council [UK]. *Adult Basic Life Support*. Resusitation Guidelines 2010. London: Resuscitation Council [UK], 2010.)

ADVANCED LIFE SUPPORT

All medical staff and appropriate senior nursing and paramedical staff should be capable of performing ALS. The ALS algorithm is shown in Figure 30.4. All persons who may be called upon to perform ALS should be familiar with this algorithm. Regular refresher courses are essential to maintain competency.

GENERAL POINTS

- Defibrillation is the definitive treatment for VF or pulseless VT and must be given as soon as possible. CPR should be paused and the rhythm assessed as soon as the defibrillator is activated and the pads are placed on the patient's chest.
- Advanced airway interventions should be considered. However, only intubate the patient's trachea if competent and trained to do so. Prolonged attempts at tracheal intubation may adversely affect the quality of chest compressions and will compromise coronary and cerebral perfusion. Supraglottic airway devices are easier to use and can be inserted without interrupting chest compressions. Once the airway is secure chest compressions may be continued uninterrupted at a rate of 100–120 min⁻¹. Ventilations should continue at 10–12 min⁻¹.
- If intravenous access is not in-situ or cannot be immediately established, the Intraosseous route should be used. Intracardiac injection, attempts at central venous access and endotracheal administration of drugs are not recommended.
- Fine VF may masquerade as asystole on the ECG. If there is any uncertainty, do not delay chest compressions. Fine VF is very unlikely to be defibrillated into a perfusing rhythm and will increase myocardial injury directly via the electrical current and through interruptions in coronary blood flow. The treatment is CPR for another 2 minutes to reoxygenate the myocardium.

- Sodium bicarbonate is no longer routinely recommended, as it may worsen intracellular acidosis. It may be given for specific indications, such as hyperkalaemia or cardiac arrest associated with tricyclic antidepressant overdose.
- In shockable rhythms, administer a shock (typically 150–200 Joules biphasic, but check manufacturer guidelines) without delay followed immediately by 2 minutes of CPR *without* stopping to assess the rhythm. Myocardial stunning may delay any subsequent rhythm from being a perfusing one and the coronary and cerebral perfusion pressure must be preserved until the rhythm is associated with a palpable circulation. Furthermore, a cycle of CPR should not be interrupted to analyse rhythms or to undertake pulse checks, unless the patient shows an obvious sign of life.
- Following 2 minutes of CPR pause briefly (<5 seconds) to assess the rhythm, palpating for a pulse if an ECG rhythm is observed that could be compatible with a cardiac output, and administer a further single defibrillation if a shockable rhythm persists.
- Epinephrine 1 mg (1:10,000 concentration) should be administered after the third shock, and thereafter given every alternate cycle of CPR (every 3 to 5 minutes).
- Amiodarone 300 mg should be administered after the third shock, and an additional 150 mg may be considered after the fifth shock.
- In non-shockable rhythms, epinephrine 1 mg (1:10,000 concentration) is indicated immediately after identification of the rhythm then every alternative cycle (3 to 5 minutes).
- PEA is usually secondary, and CPR is unlikely to be successful unless the underlying cause is treated.
- During CPR, the eight 'reversible causes' of cardiac arrest should be considered and treated, if appropriate. This includes consideration of the history, examination and investigations obtained prior to arrest, and focused examination (including echocardiography if appropriately trained) during arrest:
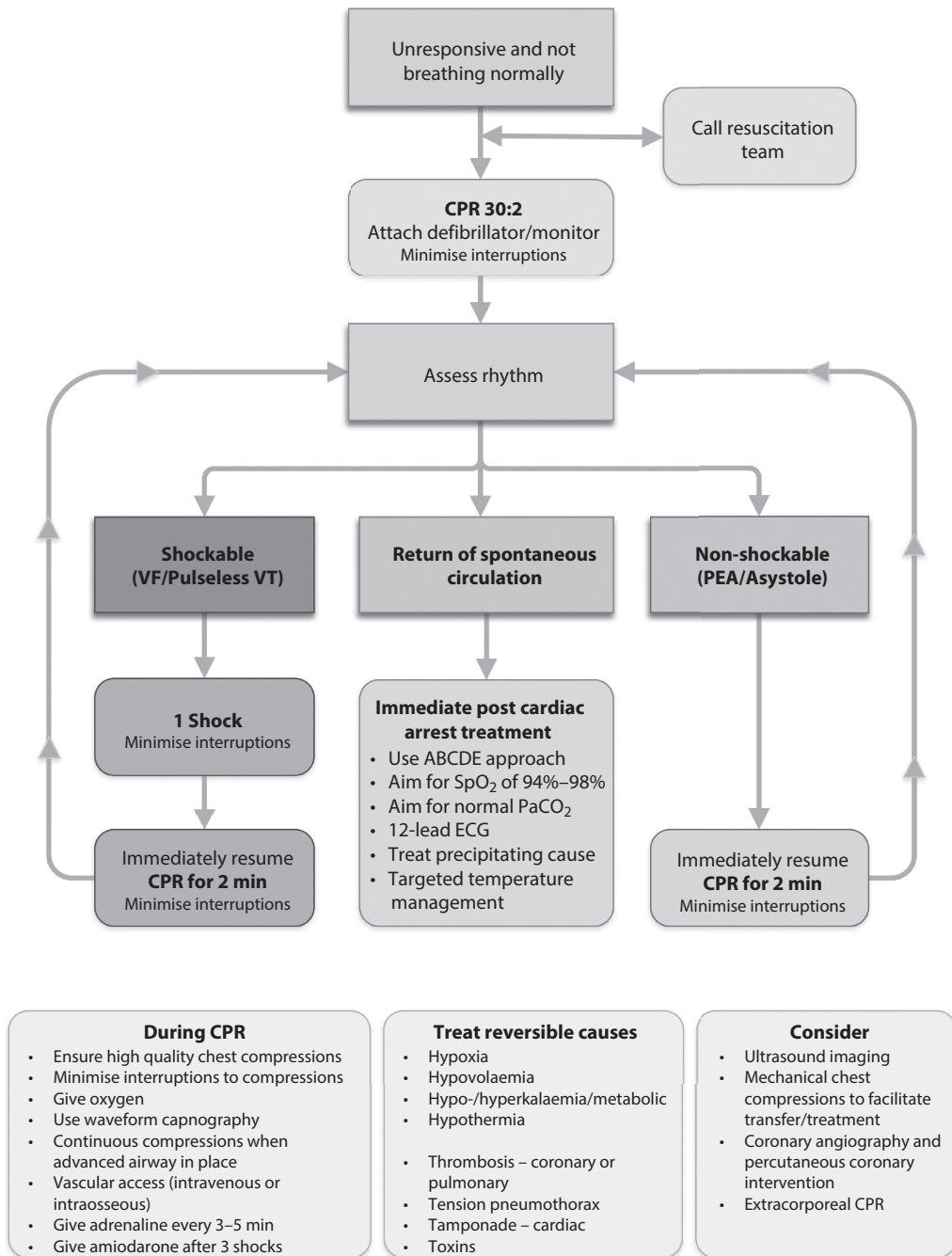
Figure 30.4 Adult life support algorithm. (From Resuscitation Council [UK]. *Adult Basic Life Support.* Resusitation Guidelines 2010. London: Resuscitation Council [UK], 2010.)

- *Hypoxia* – Ensure airway patency and administer high flow supplemental oxygen.
- *Hypovolaemia* – Examine for bleeding/fluid losses. Administer IV fluid resuscitation.
- *Hypothermia* – Measure core temperature, rewarm aggressively if hypothermic.
- *Hypo- and hyper-kalaemia (and other electrolytes and glucose)* – Consider venous blood-gas analysis to assess rapidly.
- *Thrombosis* – Consider thrombolysis if ECG features of STEMI prior to arrest, or clinical suspicion of pulmonary embolism.
- *Toxin* – Consider self-poisoning or drug reaction/overdose.
- *Tension pneumothorax* – Presents as absence of breath sounds, increased ventilatory pressure, and deviation of trachea away from the side of the pneumothorax. Treat with needle thoracocentesis (place large bore cannula in the second intercostal space in the mid-clavicular line on the affected side).
- *Tamponade, cardiac* – Becks triad (hypotension, distended neck veins and muffled heart sounds) is not observable in cardiac arrest. Diagnosis is therefore based on clinical signs observed prior to arrest or focused echocardiography. Treat with pericardiocentesis.

POSTRESUSCITATION CARE

The patient should be managed in ICU following successful resuscitation. The return of spontaneous circulation is just the first step in recovery where ALS aims to stabilise the patient, minimise any cerebral and cardiac injury which has occurred and protect against systemic ischemic reperfusion injury. A full history and examination should be performed. The ABCDE approach is recommended followed by appropriate management according to organ function.

PAEDIATRIC RESUSCITATION

The principles of CPR in children are similar to those in adults, but the following differences apply.

CAUSES OF ARREST

- Asystole or severe bradycardia are the most common dysrhythmias. They may be secondary to hypoxaemia or circulatory failure, and may be reversed by BLS and oxygenation alone.
- Cardiac arrest may be due to sudden infant death syndrome, airway obstruction (foreign bodies, epiglottitis or croup), near-drowning, asthma, trauma or severe infections.

ASSESSING FOR SIGNS OF LIFE

Because of the preponderance of respiratory causes of cardiac arrest in children, a two-step approach to assessing for signs of life is adopted. Initially, the airway is opened and breathing is assessed. If respiratory effort is absent or abnormal, five rescue breaths are administered. A central pulse is then checked (brachial or femoral for infants, carotid or femoral for children). If the pulse is absent or slow (<60 per minute) chest compressions are commenced.

AIRWAY AND VENTILATION

- Mouth to nose-and-mouth ventilation may be needed (remember the smaller tidal volumes required to prevent barotrauma).
- Abdominal thrusts and incisional cricothyrotomy are contraindicated in infants, chest thrusts and needle cricothyrotomy are alternatives.
- Insert an appropriately sized endotracheal tube. Uncuffed or paediatric-specific cuffed tracheal tubes should be used in children. Over the age of about 12 years, standard cuffed tubes may be used, depending upon the size and maturity of the child.

EXTERNAL CHEST COMPRESSIONS

The ratio of compressions to ventilations is 15:2. As per adult practice, the compression rate should be 100–120 min^{-1}. The depth of compressions varies depending on the size of the child: aim to compress 1/3 of the antero-posterior chest diameter.

DEFIBRILLATION

The energy is 4 J kg^{-1}.

DRUG DOSES

In children, drugs may be given by either the intravenous or the intraosseous routes.

- Epinephrine: 10 mcg kg^{-1} (0.1 mL kg^{-1} of 1:10,000).
- Amiodarone: 5 mg kg^{-1} after the third and fifth shock.
- Calcium: 0.2 mL kg^{-1} of 10% calcium chloride, only if specifically indicated such as in hyperkalaemia or hypocalcaemia.

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CROSS-REFERENCES

Infants and children, Chapter 25
Paediatrics – overview, Chapter 24

COMPLICATIONS OF POSITION

Failing to position the anaesthetised patient correctly can result in serious morbidity and even fatality.

Knowledge of the potential complications together with a high standard of care and patient monitoring are necessary.

The most common causes of complications are

- Accidental trauma
- External pressure
- Inappropriate passive movement
- Physiological trespass

PREOPERATIVE ASSESSMENT

Check for:

- Head and neck mobility in patients where neck rotation is anticipated, e.g. ENT or neurosurgery and in all cases of rheumatoid arthritis or ankylosing spondylitis.
- Intraocular lens prostheses.
- Brachial neurovascular symptoms if the patient's arms are to be positioned above their head.
- Symptoms of ulnar nerve entrapment syndrome.
- Active and passive range of limb movement in elderly or arthritic patients.
- Blood pressure changes or symptoms of supine hypotension in pregnant patients.

PATIENT FACTORS

Patients at increased risk of complications due to positioning include:

- Elderly patients
- Children
- Obese and cachectic patients
- Patients with poor tissue perfusion (e.g. peripheral vascular disease, diabetes mellitus)
- Patients with peripheral oedema
- Patients taking steroids

SURGICAL FACTORS

All patients should be assessed thoroughly and positioned with care and attention. Prolonged procedures (>2 h) confer an additional risk and

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should be managed meticulously. Certain surgical procedures require specific patient-positioning, as described next.

COMPLICATIONS AND DANGERS OF THE POSITIONING PROCESS

FAILURE TO SUPPORT THE HEAD ADEQUATELY

- A pillow unsupported by a stretcher canvas or the top part of the bed or trolley may fall away and cause whiplash injury to the neck.
- Uncoordinated movement of team members when rolling the patient may damage the neck.

FAILURE TO SUPPORT THE WHOLE PATIENT

- Accidents where an operating table transfer top fails to 'dock' correctly on its pedestal.
- Failure to apply brakes on transfer trolley or bed may allow the patient to fall between the operating table and the trolley/bed.
- Inappropriate number of team members relative to the weight of the patient when moving and handling.
- Failure to raise the side guard-rails on narrow transfer trolleys.

FAILURE TO PREVENT LOCALIZED DAMAGE

- Fingers can be injured or even amputated by hinged sections of the operating table or by being caught between the carrier/table and pedestal sections.
- Careless removal of the draw sheet or transfer board can cause skin abrasions.
- Accidental traction on infusion lines, drainage tubes and urethral catheters can cause internal damage.
- The 'perineal post' of the orthopaedic traction table can cause genital damage and pudendal nerve trauma.

WELL-RECOGNIZED PROBLEMS ASSOCIATED WITH SPECIFIC POSITIONS

THE SUPINE POSITION

Pressure necrosis of the skin

- Occipital – May be associated with alopecia.
- Sacral – Coincident thermal damage is also a risk with the use of under-patient warming blankets.
- Heels – Of particular relevance in diabetic patients.

Postoperative backache

- Use of a lumbar support is beneficial.

Nerve compression problems

- The supraorbital nerve may be compressed by airway tubing.
- The facial nerve can be compressed by pressure from a face-mask.
- Brachial plexus neurapraxia can result from faulty arm board positioning.
- The radial nerve can be trapped against a head-screen support.
- The ulnar nerve may be damaged against the edge of the mattress.

THE LITHOTOMY POSITION

Nervous system complications

- Straight leg sling systems may cause sciatic and femoral nerve palsies.
- The common peroneal nerve may be trapped against the head of the fibula.
- The saphenous nerve may be trapped against the supporting posts.
- In extreme flexion of the thighs, the femoral nerve can be kinked around the inguinal ligament.

Compartment syndrome

This can result from undue pressure on calf muscles and has been specifically implicated with the use of the Bierhof knee-crutch leg supports.

Joint damage

- Ligamentous damage of the hip and knee joints ensues if passive movement exceeds the normal active range.
- Sacroiliac joint strain is a risk if the legs are not raised symmetrically.

Physiological risks

- The extent of surgically induced hypovolaemia may be masked by autotransfusion from the legs.
- Increased intra-abdominal pressure enhances the possibility of gastric regurgitation – general anaesthesia should never be induced in this position.

THE LATERAL POSITION

Stability of position is essential. Evacuatable mattresses greatly enhance this.

Pressure problems

- The skin overlying the iliac crest is at risk.
- The dependent deltoid can suffer 'crush syndrome'.
- The dependent sciatic nerve is at risk in emaciated patients.
- The dependent common peroneal nerve is vulnerable around the neck of the fibula.
- Axillary support is essential to protect the dependent brachial plexus.

THE SITTING POSITION

Physiological problems

- Venous air embolism – the classical risk.
- Postural hypotension – can be exacerbated by bradycardia or hypovolaemia.

- Oedema and swelling of the tongue if the neck has been too greatly flexed and rotated – can produce airway complications postoperatively.

Nervous system complications

- Quadriplegia has been reported following head flexion and cervical spine rotation.
- Brachial plexus damage can occur if the arms are not properly supported.
- The ulnar nerve can be compressed by misplaced arm supports.
- The sciatic nerve can be stretched if the thighs are flexed and the knees extended.
- Bilateral foot drop can result from compression of the common peroneal nerves.

Pressure problems

- Sacral skin necrosis.
- Haemarthrosis and dislocation of the elbow have been reported.

THE PRONE POSITION

- Straightforward surgical procedures can be performed with the patient supported by pillows beneath the chest and the pelvis. More complex procedures, e.g. spinal axis surgery, usually require that the groins and abdomen are completely free of external pressure.
- The 'kneeling prone' and 'Mohammedan prayer' positions should never be used; lower limb congestion can cause myoglobinuria and acute renal failure (Figure 30.5).
- Methods used to support the pelvis under the iliac crests are only satisfactory if the props are positioned so that the patient is unable to slip sideways (Figure 30.6). If this occurs, unilateral femoral vascular compression is unavoidable, and congestion transmitted to the

Figure 30.5 The Mohammedan prayer position. This position should never be used.

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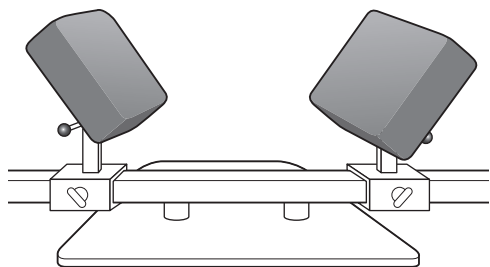
Figure 30.6 Positioning of props to support the pelvis under the iliac crests.
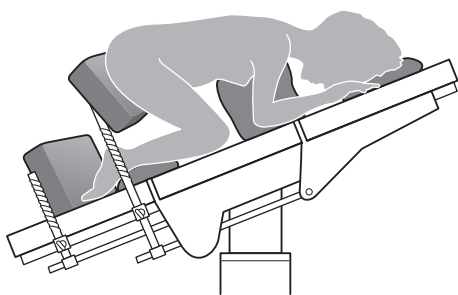
Figure 30.7 The safest position (Tarlov) for all lower thoracic and lumbar disk surgery.

epidural veins will cause troublesome surgical haemorrhage.

- The position originally described by Tarlov is the safest and most satisfactory for all lower thoracic and lumbar disc surgery (Figure 30.7). In some patients it can be used for operations on the cervical and upper thoracic spine. Although inferior vena cava pressures are known to be around 0–3 cm H_2O, venous air embolism is a very rare complication of surgery in this position.

Dangers resulting from the method of prone positioning

- Pressure necrosis of weight-bearing skin areas.
- Patients with previous coronary artery bypass graft surgery are at risk of graft occlusion.
- Neurapraxia, both brachial and axillary, can occur if the arms are positioned above the head.
- Meralgia paraesthetica due to injury of the lateral cutaneous nerve of the thigh.

- Blindness from external orbital pressure.
- Damage to male genitalia if trapped between the patients thighs.

DANGERS ARISING FROM THE SURGERY BEING PERFORMED

Damage to underlying abdominal major vessels or bowel perforation are well recorded when discectomy is being performed. Serious, hidden, intraperitoneal haemorrhage can rapidly cause acute hypovolaemic shock. Abandoning surgery and resuscitation in the supine position is the only action likely to avert disaster.

COMPLICATIONS OF ANAESTHESIA

A reinforced tracheal tube is indicated in positions where the airway may be manipulated or is difficult to access. This must be secured meticulously to avoid inadvertent malposition or removal. Appropriate invasive monitoring, arterial line, central venous line, trans-oesophageal probe, etc. need to be placed before positioning the patient for surgery and securely fixed as replacement once positioned will be challenging or impossible.

VENOUS AIR EMBOLISM

This can be caused by any open vein or sinus above heart level.

Potentiating factors:

- Inability of the vein to collapse:
  - Intracranial venous sinuses and emissary veins
  - Self-retaining surgical retractors
  - Tracks formed around indwelling catheters, e.g. central venous pressure catheter
- The level of venous pressure at the site:
  - Posture and positioning are important.
  - Raising the adult patient's head reduces intracranial venous pressure, and may even create negative pressures.
  - Spontaneous ventilation causing negative intrathoracic pressure potentiates negative pressure at the operating site.
  - In the prone position, the suction effect of a pendulous abdomen can potentiate negative venous pressure within the epidural veins.

RISK SITUATIONS

- Posterior fossa surgery in the sitting position
- Head and neck surgery in the supine head-up position
- Caesarean section
- Intrauterine manipulations in the lithotomy position
- Major reconstructive spinal surgery in the prone position

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ELECTROCONVULSIVE THERAPY

Seizure therapy, first introduced for the treatment of schizophrenia in 1934, employed pro-convulsant drugs such as metrazol. Electroconvulsive therapy (ECT) was introduced in 1938. Early ECT seizures were unmodified, i.e. no sedation, anaesthesia, relaxation, ventilation or supplemental oxygen. In 1963, the treatment was modified by the use of intravenous anaesthetic agents, neuromuscular blockade and ventilation with oxygen.

The mechanism of action of ECT is still uncertain. What is not in doubt is the efficacy and dramatic improvement in the treatment of endogenous depression and acute schizophrenic states in some patients.

PROCEDURE

Modern ECT devices deliver brief electrical stimuli via two electrodes and are equipped with controls to adjust the duration and frequency of the stimulus. The electrodes can either be attached on both sides of the head, typically bitemporally, for bilateral ECT or on the dominant hemisphere for unilateral ECT. A typical setting would be a pulse of 60 Hz and duration of 0.75 milliseconds with total stimulus duration of 1.25 seconds.

As ECT is typically undertaken at a remote site, an experienced anaesthetist (ideally a consultant) should provide anaesthesia for ECT. In cases where the patient is ASA III or above, a consultant anaesthetist must be involved in deciding the appropriate location to perform the procedure.

PHYSIOLOGICAL EFFECTS OF ECT

One of the major effects of ECT is on the autonomic nervous system. There is first stimulation of the parasympathetic system which accompanies the tonic phase of the seizure, followed by intense stimulation of the sympathetic system which accompanies the clonic phase. This can result in bradycardia followed by tachycardia, hypertension and increased myocardial oxygen demands and in susceptible patients, myocardial ischaemia. These changes are transient and rarely require treatment. ECT also raises the intracranial pressure, the intra-ocular pressure and the intragastric pressure.

PREANAESTHETIC ASSESSMENT

Patients for ECT are all adult and many are in the older age group. History can be difficult and is often unreliable. Comorbidities are common and patients may be receiving many different medications in addition to drugs for their psychiatric condition.

Drug interactions are not normally a problem. Monoamine oxidase inhibitors (MAOIs) can be withheld or substituted with the reversible and short acting MAOI moclobemide which can be withheld on the day of the procedure. Lithium is likely to cause problems and should therefore be discontinued prior to treatment. The use of propofol in patients on long-acting benzodiazepines (e.g. diazepam) may result in delayed recovery and hence long-acting benzodiazepines should be withheld prior to ECT.

Contraindications to ECT include recent cerebrovascular accident, intracranial mass lesion, recent intracranial surgery, recent (less than 3 months) myocardial infarction and aortic aneurysm.

ANAESTHETIC MANAGEMENT

Full anaesthetic and resuscitation facilities are essential and preferably a specially designated room with a fully monitored recovery area. Patients should be prepared, starved and investigated as for any other anaesthetic procedure.

PREMEDICATION

Sedative premedication is not usually required. Anticholinergics are not given routinely but should be readily available in case of an exaggerated vagal response.

MONITORING

ECG, NIBP, SaO$_2$, duration of seizure, capnography.

INDUCTION

Propofol has a direct shortening effect on seizure duration but this appears to have minimal effect on the therapeutic effectiveness. Etomidate is an alternative but the increased muscle tone and pain on injection limit its use.

Muscle relaxation is used to reduce the incidence of fractures. A small dose of succinylcholine (0.5 mg kg^{-1}) is the drug of choice. Following modified ECT, only 2% of patients suffer muscle pain.

Intubation is not normally necessary and the airway can be maintained by simple airway manoeuvres and/or airway adjuncts. Assisted ventilation with 100% oxygen is mandatory until spontaneous respiration returns. Because of the masseter contraction that occurs with ECT, it is advisable to insert a mouth guard to prevent damage to the teeth and injury to lips and the tongue.

Following ECT, bradycardia and even asystole may occur, and atropine or glycopyrrolate may be required. These drugs may accentuate the sympathetic response that invariably follows, putting undue stress on the myocardium. The use of a short-acting beta blocker such as esmolol has been advocated to attenuate the sympathetic response and should be available. Prolonged seizure following ECT is uncommon and can usually be aborted with benzodiazepines.

POST-ECT MANAGEMENT

Patients should be closely monitored until fully recovered.

OUTCOME

Mortality following ECT is 0.02%–0.04%. Arrhythmias, myocardial infarction, congestive cardiac failure and sudden cardiac arrest are the most common causes of death, nearly always occurring during the recovery period.

Morbidity following ECT includes memory loss, confusion, drowsiness, muscular aches, weakness, anorexia and amenorrhoea.

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FAILURE TO BREATHE OR WAKE UP POSTOPERATIVELY

All anaesthetic agents and opioids are respiratory depressants and may cause apnoea. Failure to breathe postoperatively may also follow the use of

intraoperative muscular relaxation. Remember that there may be more than one cause operating simultaneously and causes may be cumulative.

FAILURE TO BREATHE POSTOPERATIVELY

NONDEPOLARIZING AGENTS

- Inadequate reversal of neuromuscular block has historically been defined as a train of four ratio of less than 0.7. However, recent studies suggest that a ratio of greater than 0.9 is required to minimise respiratory complications. A dose of neostigmine should have been given before 'failure to breathe' is noted. Sugammadex will only antagonize a block resulting from rocuronium and, to a lesser extent, vecuronium.
- Look for concomitant drugs which themselves depress neuromuscular function or potentiate a neuromuscular blocking agent, e.g.
  - Aminoglycoside antibiotics
  - Magnesium
  - Phenytoin
  - Cyclosporin A
- Renal or hepatic failure may reduce the metabolism or excretion of nondepolarising agents. Cholinergic crisis secondary to excessive neostigmine can present with flaccid paralysis but is rare, and should be prevented by the concomitant administration of an anticholinergic.

DEPOLARIZING AGENTS

- Plasma cholinesterase deficiency (genetic or acquired)
- Dual block (type-II block)

OTHER FACTORS

- Respiratory depressants (opioids, anaesthetic agents, benzodiazepines)
- Pain, especially if thoracic or abdominal in origin
- Hypocapnia

- Severe hypercapnia
- Hypothermia, especially in children
- Metabolic disturbance
- Acidosis
- Hypokalaemia
- Hypomagnesaemia
- Coexisting neuromuscular disease
- Intraoperative cerebral event

MANAGEMENT

- Ensure the patient's safety.
- Ensure that the airway is protected and that adequate ventilation is maintained, by hand if necessary.
- If the patient is conscious, reassure them and consider sedation.
- Monitor vital signs.
- Determine cause:
  - Assess neuromuscular function.
  - Review dose of muscle relaxant.
  - Review dose of reversal.
  - Check if other agents which may affect a block have been given.
  - Verify that all anaesthetic agents are turned off.
  - Consider the patient's metabolic status.
  - Measure the patient's temperature.
- If residual paralysis is present and the train of four count is two or more, a further dose of neostigmine and an anticholinergic (up to 100 mcg kg^{-1} total dose of neostigmine) may help.
- If excessive opiate is present, give increments of 100 mcg naloxone.
- If excessive benzodiazepines have been used, consider flumazenil (200 mcg over 15 s, then 100 mcg every 60 s, as required).
- If central respiratory depression is present, consider titrated doses of doxapram (1–1.5 mg kg^{-1} over 30 s).
- Control pain with local techniques or carefully titrated doses of opioids.
- Optimize the patient's metabolic condition.
- Wait.
- If ventilation is still not adequate, transfer the patient to ICU.

NORMAL AWAKENING

Awakening occurs when the effect-site concentration of anaesthetic agent falls to a level insufficient to maintain unconsciousness. This occurs due to the redistribution, metabolism or elimination. It is therefore a passive process, with specific antagonists existing only for opioids and benzodiazepines.

FAILURE TO WAKE UP

CAUSES

There may be more than one reason present, and effects are cumulative:

- Overdose (absolute or relative) of anaesthetic agent, premedication (including benzodiazepines), or opioid
- Hypothermia, particularly in children
- Hypercapnia
- Hypothyroidism
- Hypoglycaemia
- Severe liver disease, in which anaesthesia may precipitate encephalopathy
- Cerebral hypoxia

MANAGEMENT

- Ensure the patient's safety.
- Ensure that the airway is protected and that ventilation is adequate.
- Continue monitoring.
- Assess cause:
  - Arterial blood gas analysis
  - Core temperature
  - Consider coexisting disease.
  - Blood glucose measurement
- Treat any treatable causes:
  - Correct any abnormalities in acid–base status.
  - Rewarm if necessary.
  - Administer a cautious dose of pharmacological antagonists, if appropriate.
- Wait.
- Consider transfer to ICU if awakening continues to be delayed.

The only cause of persistent, irreversible failure to recover from anaesthesia is a cardiorespiratory or cerebrovascular disaster with resultant cerebral hypoxia. Thankfully, this is rare.

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FAT EMBOLISM

Fat embolism is defined as the presence of fat globules within the lung parenchyma or peripheral microcirculation. The fat embolism syndrome (FES) is defined as fat in the circulation associated with a clinical pattern of symptoms and signs; classically a triad of petechial rash, respiratory and neurological abnormalities. It is associated with:

- Fractures of long bones and pelvis
- Prosthetic joint replacement
- Liposuction
- Bone marrow harvest or transplant
- Bone tumour lysis
- Acute pancreatitis
- Hepatic necrosis and fatty liver
- Acute sickle cell crisis (bone marrow necrosis)
- Following extra-corporeal circulation
- Major soft tissue injury
- Severe burns

The most common group is patients with long-bone fractures and in this situation fat embolism can be demonstrated to be present in over 90% of

patients. FES is only seen in 1%–10%. Those at highest risk include young adult males and those with closed or multiple fractures.

SUBCLINICAL FES

Frequently seen in patients with long-bone fractures. Mild hypoxaemia and minor haematological abnormalities develop up to 3 days after injury.

NONFULMINANT (SUBACUTE) FES

Seen in up to 5% of major trauma patients. Onset may be delayed by 12–36 hours after injury. Classical syndrome of hypoxaemia and respiratory failure, petechial rash, fever, tachycardia, neurological symptoms and associated haematological abnormalities.

FULMINANT FES

Sudden onset, within a few hours of injury. Pulmonary and systemic embolisation of fat, right ventricular failure and cardiovascular collapse. This can occur intraoperatively. Progresses rapidly, often with a fatal outcome.

PATHOPHYSIOLOGY

Two theories have gained acceptance – a mechanical and a biochemical model. Fat emboli are thought to originate from exposed marrow at the site of injury. Fat globules enter the circulation, facilitated by movement of the fracture site. Subsequent pulmonary and neurological damage is thought to be partly due to vascular occlusion (mechanical theory), and partly due to local effects of free fatty acids released from fat emboli (biochemical theory).

Respiratory insufficiency results from emboli entering the venous circulation and lodging in the pulmonary circulation. The free fatty acids released are toxic to pneumocytes and capillary endothelium causing intra-alveolar haemorrhage, oedema and chemical pneumonitis. Cerebral involvement and petechial rash result from fat emboli entering the arterial circulation, either via the pulmonary alveolar capillaries or via precapillary pulmonary shunts which have opened as a result of pulmonary hypertension.

DIAGNOSIS

The most established diagnostic scoring system is Gurd and Wilson's criteria. Major criteria are petechial rash, respiratory insufficiency and cerebral involvement. Minor criteria are tachycardia, pyrexia, retinal changes (fat or petechiae), fall in haemoglobin, thrombocytopaenia, raised erythrocyte sedimentation rate and fat globules in the sputum. One major and four minor criteria, plus fat macroglobulaemia, must be present for the diagnosis of FES to be made. However, this and other scoring systems are frequently criticized and clinical suspicion remains an important component of diagnosis.

LABORATORY FINDINGS

- A decrease in haematocrit at 24 to 48 hours (thought to be secondary to intra-alveolar haemorrhage).
- Thrombocytopaenia (platelet count <150 × 109) and other coagulation abnormalities.
- Fat globules in blood and urine.
- Fat macroglobulinaemia, raised free fatty acids (hypocalcaemia may result due to their affinity for calcium) and triglycerides in serum.

OTHER INVESTIGATIONS

Chest radiograph may be normal initially. Evolving changes may include bilateral diffuse pulmonary infiltrates and fleck-like shadows described as a 'snowstorm' appearance.

A CT brain scan may show generalized cerebral oedema or diffuse white-matter petechial haemorrhages but is often nonspecific. Its utility usually lies in ruling out other diagnoses for reduced conscious level. MRI may detect specific lesions in the presence of a normal CT scan.

Bronchoscopy and bronchoalveolar lavage have been used to provide samples containing macrophages, which act as lung scavengers and may contain fat in FES. This may aid diagnosis but is not an adequately specific test in isolation for FES.

Echocardiography may detect increased right-heart pressures, and intraoperative transesophageal echocardiography has been shown to demonstrate showers of emboli during intramedullary nailing.

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A pulmonary artery catheter may be useful in detecting a rise in mean pulmonary artery pressure or in sampling pulmonary artery blood for fat.

MANAGEMENT

- Treatment is supportive with early resuscitation and stabilization.
- Maintain adequate oxygenation and ventilation.
- Indications for respiratory support:
  - Sustained SaO_2 <90% and PaO_2 <8 kPa on oxygen
  - Respiratory rate of >35 breaths min^{-1}
- Avoid hypovolaemia
- Systolic blood pressure <90 mmHg is associated with adverse outcomes.
- Fluid resuscitation with balanced electrolyte solutions and albumin is recommended.
- Albumin use is considered potentially therapeutic in its ability to bind free fatty acids.

SURGICAL MANAGEMENT

- Early (within 24 hours) surgical stabilization of long bone fractures reduces risk of the pulmonary complications.
- Intramedullary nail insertion and reaming causes increased fat embolization when compared to plate and screw fixation. The method of fracture stabilisation chosen should take into account the physiological state of the patient and concomitant injuries.
- Adequate analgesia should be maintained to limit the sympathetic response to injury.

ADDITIONAL TREATMENT

Corticosteroids (methylprednisolone) have been shown to reduce the risk of FES and hypoxaemia in patients with long bone fractures of the lower limbs. Optimal timings and doses are unclear.

Heparin clears lipaemic serum by stimulating lipase activity and in experimental models has been shown to reduce pulmonary complications. Despite its associated bleeding risks, it has been used clinically but has not shown consistent benefits in reducing lung injury.

Alcohol decreases serum lipase activity and dextrose decreases free fatty acid mobilisation have both been used empirically but there is little supportive evidence.

Aspirin blocks the production of thromboxane, which occurs in animal models of FES. Current evidence does not support its use.

OUTCOME

The overall mortality of FES is 5%–15%. The condition is usually self-limiting with adequate supportive therapy. Long-term morbidity is related to focal neurological lesions.

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CROSS-REFERENCE

Anaesthesia for orthopaedic surgery – general principles, Chapter 22

FLUID AND ELECTROLYTE BALANCE

Body water content varies with age and gender as a percentage of body weight (Table 30.6). Approximately

Table 30.6 Body water composition in health as a percentage of body weight

	TBW (%)	ICF (%)	ECF (%)
Neonate	75	40	35
Infant	70	40	30
Adult male	60	40	20
Adult female	55	35	20
Elderly female	45	30	15

Table 30.7 Distribution of water (L) and electrolytes (mmol L^{-1}) in a normal 70-kg man

	ICF	ECF Interstitial fluid	ECF Plasma
Water (L)	28	11	3
% of TBW	40	16	4
Na$^+$	10	140	140
K$^+$	150	4	4
Ca^{2+}	–	2.5	2.5
Mg^{2+}	26	1.5	1.5
Cl$^-$	–	114	114
HCO$_3^-$	10	25	25
Cl$^-$	–	114	114
HCO$_3^-$	10	25	25
HPO$_4^{2-}$	38	1	1
SO$_4^{2-}$	–	0.5	0.5
Prot$^-$	74	2	16

two-thirds of the total body water (TBW) is intracellular fluid (ICF) and one-third is extracellular fluid (ECF). The ECF is further subdivided into interstitial fluid (ISF) and plasma.

TBW and electrolytes in a 70-kg man are distributed between the various compartments as shown in Table 30.7.

OSMOTIC ACTIVITY

Water moves between compartments from areas of low solute concentration to areas of high concentration by osmosis. The number of osmotically active particles in solution is expressed in osmoles (Osm). Osmolarity is the number of particles per litre of solvent (Osm L^{-1}), and osmolality is the number of particles per kilogram of solvent (Osm

Table 30.8 Approximate fractional distribution of infusions within compartments[a]

	ICF (%)	ECF Interstitial fluid (%)	ECF Plasma (%)
Saline (0.9%)	0	79	21
Dextrose (5%)	67	26	7

[a] These figures demonstrate why large volumes of crystalloids are required to expand plasma volume.

kg^{-1}). Therefore, at 0°C, at which the density of water is 1 kg L^{-1}, osmolarity and osmolality are equivalent.

Osmolality can be estimated by adding the concentrations of osmotically active particles within compartments. ECF osmolality is usually calculated by adding the plasma concentrations (mmol L^{-1}) of sodium, potassium, chloride, urea and glucose. An alternative commonly used rule of thumb is to add twice the sodium plus urea plus glucose.

Osmotic pressure is calculated by multiplying osmolalities by 19.3 to give pressures (mmHg). Thus, ICF has an osmolality of 281 mOsm L^{-1} and an osmotic pressure of 5430 mmHg, but plasma has an osmolality of 281 mOsm L^{-1} and an osmotic pressure of 5453 mmHg. This difference of 23 mmHg is due to the presence of plasma proteins.

The redistribution of infused fluid within the body will depend on its composition relative to that of each compartment, as shown in Table 30.8. Salt solutions are excluded from the ICF by the cell membrane Na$^+$/K$^+$ pump. Dextrose (5%) behaves like water and is distributed throughout the TBW.

NORMAL HOMEOSTASIS

Humans have evolved to retain water and sodium efficiently. Even healthy individuals are slow to excrete a sodium load. Sodium excretion is not active and probably occurs by suppression of the renin-angiotensin-aldosterone system.

Blood volume is maintained at the expense of serum osmolality in hypovolaemia. If hypotonic solutions are administered, this may exacerbate hyponatraemia and water overload.

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WATER

A normothermic 70-kg man with a normal metabolic rate loses approximately 2500 mL of water per day or 35 mL/kg/h (urine 1500 mL, faeces 100 mL, sweat 500 mL, lungs 400 mL). Insensible loss increases by 10% for each 1°C rise in body temperature. Water is gained from ingested fluid (1300 mL), food (800 mL) and metabolism (400 mL). Maintenance require-ments are therefore approximately 25–30 mL kg^{-1} day^{-1} for adult surgical patients (ideal body weight should be used for obese patients).

SODIUM

Loss in faeces and sweat is about 10 mmol day^{-1}, renal excretion being mainly dependent on dietary intake. Average requirements are 1 mmol kg^{-1} day^{-1}.

This could be provided by:

- 2500 mL of 4% dextrose/0.18% saline over 24 h
- 2000 mL of 5% dextrose and 500 mL of 0.9% saline over 24 h

POTASSIUM

Loss is via the same routes as sodium, but renal reten-tion is less efficient. The average requirement is 1 mmol kg^{-1} day^{-1}. This should be added to the infusion regime.

PERIOPERATIVE FLUID MANAGEMENT

ASSESSMENT

The fluid status of a patient undergoing surgery will depend on the presenting complaint (including the severity of physiological derangement), the previous health of the individual and whether surgery is elec-tive or urgent.

Fluid and electrolyte balance should be determined by repeated clinical assessment throughout the periop-erative period and should include an estimate of total balance and intravascular volume (vascular filling).

Total balance

The history will point to decreased intake or increased loss:

- Note the duration of preoperative fasting.
- Review the ability of the patient to maintain fluid balance during illness.
- Consider normal variations in fluid balance at extremes of age.
- Identify increased losses – pyrexia, diarrhoea, vomiting and haemorrhage.
- An acute abdomen may result in large volumes of fluid sequestered in the abdominal compartment.

Examination should include mucous membranes, skin turgor, sensorium, heart rate, blood pressure, respiratory rate and urine output:

- Body weight should be monitored regularly following admission.
- Charts should be regularly reviewed to identify urine output and losses via nasogastric tubes and drains.
- Fluid prescription charts should be analysed to identify the volume and type of fluid administered.

Investigations including simple blood tests supply additional information:

- Elevated urea or creatinine.
- Elevated haematocrit.
- Check plasma levels of sodium and potassium.

Intravascular volume

Assessment is by pulse, blood pressure, capillary refill and jugular venous pressure.

Invasive and noninvasive measures of intravascu-lar volume can include:

- Arterial catheters (arterial pressure and pulse pressure variation).
- Trans-oesophageal Doppler monitoring (stroke volume, flow time corrected).
- Devices utilising pulse contour analysis to derive haemodynamic parameters (stroke volume, stroke volume variation).
- Echocardiography (e.g. inferior vena cava diameter and collapsibility, left ventricular end diastolic area).
- Pulmonary artery catheters (cardiac output, central and pulmonary capillary wedge pressures).

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- Central venous pressure does not correlate with volume status.

The goal of clinical assessment is to determine the fluid and electrolyte balance of each compartment.

PERIOPERATIVE FLUID THERAPY

Patients should commence surgery in a state of normal and stable fluid and electrolyte balance.

In general, healthy patients undergoing elective minor surgery do not need perioperative fluid replacement unless they are unable to drink normally in the early postoperative period.

In other patients, the following should be considered:

Preventing and replacing preoperative deficits

- Patients should be encouraged to maintain their oral intake of clear fluids until 2 hours prior to the induction of anaesthesia (carbohydrate-rich clear fluids may confer additional benefits).
- Correction of a deficit in maintenance (normal daily intake) fluids should be with the equivalent of 1.25 mL kg^{-1} h^{-1} of balanced crystalloid (e.g. Hartmann's solution) in proportion to the oral intake of the patient.
- Patients presenting as an emergency may have had significantly reduced intake over several days. This may continue whilst they are an inpatient and is exacerbated by repeated periods of fasting whilst awaiting surgery.

Abnormal losses and redistribution is common in surgical patients and includes:

- Bowel preparation
- Sequestration (bowel obstruction or ileus)

- Vomiting and diarrhoea
- Enterocutaneous fistulae
- Stoma
- Wounds
- Haemorrhage

Significant hypovolaemia may occur with inflammatory conditions, including severe infections (sepsis), peritonitis and pancreatitis.

All routes by which fluid and electrolytes can be lost from the appropriate compartments should be considered.

Replacement is based on an estimate of the composition and volume of loss. Losses from the gut are particularly important, and compositions of the various gastrointestinal secretions differ. Attention should be paid to replacing electrolytes appropriately, and serum biochemistry should be monitored.

In severe hypovolaemia with signs of shock give isotonic crystalloid (e.g. Hartmann's solution) 10 mL kg^{-1} as a bolus, then reassess, with additional fluid given according to the patient's response.

The properties of intravenous fluids are shown in Table 30.9.

Intraoperative management

The goal is to maintain ideal tissue perfusion (oxygen delivery) whilst avoiding fluid compartments becoming overloaded. Inappropriate administration of unbalanced colloids can result in salt overload (hypernatraemia and hyperchloraemic metabolic acidosis) and can precipitate a hyperoncotic state.

Fluid management is complicated by the dynamic interaction between anaesthesia and surgical insult (including the stress response). Intravascular hypovolaemia can be relative (vasodilatation) and absolute (reduction in blood volume).

Table 30.9 Properties of intravenous fluids in comparison with plasma

	Sodium (mmol L^{-1})	Potassium (mmol L^{-1})	Chloride (mmol L^{-1})	Duration of plasma volume expansion
Plasma	136–145	3.5–5.0	98–105	–
5% dextrose	0	0	0	Not applicable
0.9% saline	154	0	154	<30 min
Hartmann's	131	5	111	<30 min
4% gelatine	145	0	145	1–2 h
6% HES 130/0.4	154	0	154	4–8 h

Table 30.10 Estimated blood volumes

	Volume (mL kg⁻¹)
Infant	90
Child	80
Adult male	70
Adult female	60
Pregnant female at term	100

Maintenance fluid can be provided with balanced crystalloid or dextrose (4%) with saline (0.18%) at 1.25 mL/kg/h.

Additional fluids should be administered based on continuous clinical assessment and the physiological goals for the individual patient. This should be judged according to the patient response, including heart rate, blood pressure, tissue perfusion and urine output (not less than 0.5 mL kg⁻¹ h⁻¹).

Goal directed haemodynamic therapy (GDHT) has demonstrated benefits in high-risk patients undergoing major surgery. Vascular filling can be guided by measures of stroke volume and cardiac output. Flow can be measured continuously using trans-oesophageal Doppler and pulse contour analysis devices. Applying the Frank-Starling curve, small (250 mL) boluses of crystalloid or colloid are infused to maximise stroke volume early in the intraoperative period, thereby maintaining 'optimal' tissue perfusion throughout surgery. Cardiac output is also dependent on contractility which can be increased with cautious use of inotropes.

The evidence base for GDHT targets oxygen delivery in excess of 600 mL/min/m⁻² using intravenous fluids and, if necessary, inotropes and blood transfusion. This strategy has been shown to reduce morbidity and mortality in high-risk surgical patients. However, it has been suggested that targeting the threshold of 600 mL/min/m⁻² may not be achievable or desirable in all cases, and inotropes have been shown not to improve outcomes when compared to fluids alone and are therefore rarely used. It may therefore be appropriate to adopt a more pragmatic goal of maintaining the oxygen delivery above the patient's baseline values.

Postoperative requirements

Opinions about the electrolyte requirements of postoperative patients differ widely in clinical practice. Though the stress response to surgery increases renal excretion of potassium, tissue trauma and catabolism release intracellular potassium which helps to maintain plasma levels. Potassium is not required for the first 24–48 h in most patients. Intravenous fluids should be avoided if possible, in circumstances where oral fluids are not contraindicated.

Postoperative fluids should be tailored to the specific patient; however, a reasonable starting point would be to prescribe a conservative maintenance fluid regimen and titrate as appropriate:

- Maintenance: 25–30 mL kg⁻¹ day⁻¹ of 4% dextrose with 0.18% saline.
- Estimate and replace other fluid and electrolyte losses.

All fluid replacement should be regularly reviewed according to the patient's response: heart rate, blood pressure, respiratory rate, tissue perfusion, plasma electrolytes, urine output (at least 0.5 mL kg⁻¹ h⁻¹) and body weight.

Blood loss

Blood should be given to maintain a reasonable haemoglobin level for the individual patient. Transfusion will be guided by clinical assessment of the patient, the pattern of bleeding and measures of haemoglobin and haematocrit, and consideration of their usual circulating volume (Table 30.10). The adoption of 'liberal' and 'restrictive' transfusion triggers based on haemoglobin remains controversial in the perioperative population.

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CROSS-REFERENCES

Blood transfusion, Chapter 30
Water and electrolyte disturbances, Chapter 25

INTRAOPERATIVE ARRHYTHMIAS

Over 60% of patients may experience perioperative arrythmias. The majority are benign but rhythm disturbance can be associated with potentially serious adverse outcomes. The significance should be evaluated in the context of:

- Coexisting medical problems and their treatment
- The surgical condition
- The operative procedure
- Anaesthetic drugs and technique
- Haemodynamic effect of the arrhythmia and the risk of progression to a more serious arrhythmia

Conditions associated with arrhythmias are given in Table 30.11. Physical examination and a 12-lead ECG with rhythm strip may not help. Further evaluation requires 24-h ECG monitoring. The diagnosis and management can be complex, particularly with the proarrhythmic potential of certain antiarrhythmic drugs. A cardiological opinion may be required.

V1 is the lead of choice for rhythm monitoring. In a three-lead system, MCL1 or II is best. Depending on the context, direct blood pressure monitoring may be indicated.

During anaesthesia (Table 30.12) and surgery (Table 30.13) the development of arrhythmias may indicate an adverse change in myocardial oxygen balance. The relationship between volatile agents and arrhythmias involves effects on cardiac ion channels, sensitisation to adrenaline and myocardial oxygen balance.

Table 30.11 Preoperative conditions associated with arrhythmias

- Pre-existing arrhythmias
- Ischaemic heart disease
- Hypertension
- Congestive heart failure
- Electrolyte disorders (especially potassium, magnesium and calcium)
- Valvular heart disease
- Medications:
  - β_2 agonists
  - Theophylline
  - Tricyclic antidepressants
- Less common causes:
  - Thyrotoxicosis
  - Cardiomyopathies (including alcoholic)
  - Myocarditis
  - Trauma (myocardial or intracranial)
  - Connective tissue disorders
- Drug and solvent abuse

Table 30.12 Anaesthetic factors which may induce arrhythmias

- Hypotension or hypertension (e.g. inadequate anaesthesia)
- Hypoxia
- Hypercarbia
- Laryngoscopy
- Central venous pressure lines (irritation by line tip; microshock hazard)
- Drugs:
  - Volatile anaesthetic agents
  - Suxamethonium
  - Pancuronium
  - Multiple drugs prolong the QT interval; the most common include volatile agents, macrolide antibiotics, butyrophenone antipsychotics, amiodarone and ondansetron.

MANAGEMENT

Intraoperatively, more than one factor is likely to contribute to the occurrence of an arrhythmia. Identify the rhythm, evaluate its significance and identify any precipitating factors.

Table 30.13 Surgical factors which may induce arrhythmias

- Catecholamines
  - Endogenous, from any surgical stimulus
  - Exogenous – topical or infiltrated epinephrine
- Autonomic stimulation
  - Peritoneal and visceral traction
  - Peritoneal insufflation
  - Trigeminovagal reflexes (most notably oculocardiac but seen throughout the trigeminal nerve distribution)
  - Laryngoscopy, bronchoscopy, oesophagoscopy
  - Carotid artery and thyroid surgery
- Direct stimulation of the heart during cardiac or thoracic surgery
- Embolism
  - Thrombus
  - Fat
  - Bone cement
  - Air
  - Carbon dioxide
  - Amniotic fluid
- Other
  - Aortic cross-clamping
  - Limb reperfusion
  - Glycine intoxication

Table 30.14 Distinguishing broad complex tachycardias[a]

- Supraventricular tachycardia (SVT)
  - No axis change and same pattern of bundle branch block as pre-SVT ECG
  - Conforms to right or left bundle branch block pattern
  - Dominant R in V1 and Q in V6.
- Ventricular tachycardia (VT)
  - Axis change from previous ECG
  - Deep S wave in V6
  - Fusion beats
  - Capture beats

[a] Haemodynamic stability is not a distinguishing factor. VT is much more common than broad complex SVT, especially if a previous ECG shows narrow complexes.

- Take an ABC approach.
- Correct precipitating factors (Tables 30.11–30.13).
- Treat the whole patient not just the ECG.

TACHYARRHYTHMIAS

Synchronised DC cardioversion is often a more attractive treatment option than pharmacological therapy in the already anaesthetised patient, especially in the presence of haemodynamic instability. Consider an anti-arrhythmic drug, depending on the arrhythmia and patient factors. Pharmacological options include:

- Ventricular tachycardia (VT): amiodarone 300 mg over 10–60 minutes, followed by 900 mg over 24 hours. Ideally via central venous access.
- Polymorphic VT (torsades): magnesium 2 g (8 mmol) over 10 minutes.
- Atrial fibrillation: beta blocker (e.g. esmolol 50–200 mcg kg^{-1} min^{-1} infusion), digoxin 500 mcg over 1 hour (± repeat), or amiodarone.
- Supraventricular tachycardia (SVT): adenosine rapid boluses of 6 mg, 12 mg, 12 mg via a large-bore proximal cannula.
- Atrial flutter: beta blocker.

The negative inotropic effect of many anti-arrhythmic drugs should be remembered. In patients with existing conduction abnormalities, e.g. bundle branch block, it can be difficult to distinguish SVT from VT. Some distinguishing features are detailed in Table 30.14. If in doubt, treat as VT.

BRADYARRHYTHMIAS

Glycopyrrolate or atropine can be given to antagonize excessive vagal tone (e.g. from peritoneal or ocular traction). Complete heart block is unlikely to respond to anticholinergics, and is likely to require pacing. Positively chronotropic catecholamines, e.g. isoprenaline can be used as a bridging measure.

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CROSS-REFERENCES

Conduction defects, Chapter 2
Coronary artery disease, Chapter 2
Water and electrolyte disturbances, Chapter 25
Fluid and electrolyte balance, Chapter 30
Pacing and anaesthesia, Chapter 2

INTRAOPERATIVE BRONCHOSPASM

Bronchospasm is caused by constriction of bronchiolar smooth muscle within the airways. It is characterized by an expiratory wheeze and elevated airway pressures. Many patients suffer from mild bronchospasm as a part of the conditions of asthma, COPD and allergic reactions. It is usually benign, can be severe, is occasionally life-threatening and on rare occasions can be fatal. During anaesthesia it is rare in comparison to upper airway obstruction and occlusion of the breathing circuit, both of which should be excluded before the diagnosis of bronchospasm is made.

Table 30.15 Incidence of bronchospasm

Age group (years)	Studied variable	Incidence (per 1000 cases)
0–9	Organic heart disease	15.3
	Abnormal ECG	24.3
	Respiratory infection	41.1
	Obstructive lung disease	21.9
	Tracheal intubation	9.1
50–69	Previous myocardial infarction	5.4
	Obstructive lung disease	7.7

Asthma, coronary artery disease, smoking and respiratory infection are recognized risk factors. A significant number of cases, however, including those leading to adverse outcomes (brain damage, death), will occur in those without such factors.

The overall incidence is about 1.7 per 1000 anaesthetics, higher in ages 0–9 years (4.0 per 1000) and 50–69 years (1.8 per 1000) with variations in certain subgroups (Table 30.15).

CAUSES OF BRONCHOSPASM

AIRWAY INSTRUMENTATION

- Instrumentation and irritation of the airway
- Tracheal intubation (the most common trigger)
- Carinal stimulation
- More likely under light anaesthesia

SURGICAL STIMULATION

- Any surgical stimulation
- Upper abdominal, intra-oral, anal and cervical procedures are more prone
- Inadequate anaesthesia

BRONCHIAL ASPIRATION

- May present with unilateral bronchospasm
- Could account for the higher incidence in children and during abdominal surgery

ANAPHYLACTIC REACTIONS

- The first sign in 25% of reactions
- The sole feature in 8% of reactions

DRUGS

- Beta adrenergic blockers
- Neostigmine
- NSAIDs – about 10% of asthmatics have symptoms exacerbated by NSAIDS (beware of Samter's triad of asthma, nasal polyps and aspirin intolerance)
- Histamine-releasing drugs (e.g. morphine, atracurium)

REGIONAL TECHNIQUES

- Bronchospasm has been reported during solely regional techniques.
- Reports of bronchospasm occurring during spinal and epidural anaesthesia exist.
- Psychological factors may trigger bronchospasm in asthmatics.

CLINICAL FEATURES

- Tachypnoea or laboured respiration
- Intercostal recession
- Expiratory wheeze
- Increased airway pressure
- Decreased compliance
- Delayed rise in CO_2 trace on capnography
- Ventilation and oxygenation become increasingly difficult
- Hypoxia and cyanosis
- Air trapping with hyperinflation of the chest
- Acute pulmonary hypertension and reduced venous return due to elevated intrathoracic pressure result with fall in cardiac output in the most severe cases
- Pneumothorax due to barotrauma (suspect pneumothorax if there is a sudden deterioration)

MANAGEMENT

The majority of cases occur as a reflex response to airway instrumentation or surgery and are relatively

Table 30.16 Differential diagnosis of bronchospasm

Diagnosis	Notes
Upper airway obstruction	Chin lift, jaw thrust, insert airway
Obstructed laryngeal mask	Reposition – if in doubt, remove
Obstructed tracheal tube	Verify patency – pass suction catheter, pass bronchoscopeIf in doubt, remove
Breathing circuit malfunction	Check equipment
Breathing circuit obstruction	Check equipment
Tension pneumothorax	Airway pressure, auscultate chest
Oesophageal intubation	Observe capnograph trace

mild. Temporarily interrupt surgery and deepen anaesthesia. If there is an inadequate response:

- Give 100% oxygen and summon help if required
- Stop surgery/stimulation
- Exclude upper airway and breathing and circuit problems
- Exclude other important differential diagnoses (Table 30.16)

VOLATILE AGENTS

- All are effective bronchodilators.
- Many cases respond to an increase in the inspired concentration.
- Isoflurane is the least arrhythmogenic and is the agent of choice if using epinephrine.

BETA-2 ADRENORECEPTOR AGONISTS

- Salbutamol
  - 250 mcg IV, diluted to 50 mcg mL^{-1}
  - Intravenous infusion (5–20 mcg min^{-1})
  - Metered-dose inhaler, in-circuit, via adapter (8–10 puffs)
  - Nebulized in circuit (2.5–5 mg)
- Terbutaline (250–500 mcg SLOW IV)

AMINOPHYLLINE

- May not give additional bronchodilatation if volatile agents are being used
- Inferior to beta$_2$ adrenoreceptor agonists
- Administer 5 mg kg^{-1} slowly intravenously (over 20 minutes)

CORTICOSTEROIDS

- Of benefit in acute bronchospasm although mechanism of action is unclear
- Onset of action not as rapid as other agents
- Hydrocortisone (200–500 mg IV) or methylprednisolone (1 g IV) single dose.

EPINEPHRINE

- First-line agent in severe reactions and in anaphylaxis
- Give 0.1–1 mL of 1:10,000 concentration (10–100 mcg) IV (adult), titrated to response

KETAMINE

- Powerful bronchodilator
- Consider if there is a poor response to other agents

MAGNESIUM SULPHATE

- Not licensed for treatment of bronchospasm, but may be useful in refractory cases
- 1.2–2 g (4.8–8 mmol) IV over 20 minutes

VENTILATION

- Give 100% oxygen.
- Aim to reduce any risk of barotrauma and maintain oxygenation.
- Use a low frequency of ventilation with a long expiratory time (minimizes pulmonary distension).
- Use low tidal volumes to limit airway pressures.
- A degree of hypercapnia is acceptable, provided that oxygenation is maintained. The minute volume can be increased as the bronchospasm resolves.
- 'Educated-hand' manual ventilation may produce better oxygenation with higher minute

volumes and lower airway pressures than mechanical ventilation.
- In the most severe cases, expiration due to passive recoil of lung and thorax cannot occur – manual deflation of the chest may buy time (best with 2 persons):
  - Inflate with 100% oxygen.
  - Disconnect tracheal tube.
  - Squeeze antero-lateral aspects of chest for 10–15 s.
  - Repeat this cycle.
- Worst-case scenario: catastrophic bronchospasm with the risk of severe barotrauma; cardiac arrest is imminent. Consider cardiopulmonary bypass, if available.

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CROSS-REFERENCES

Asthma, Chapter 1
COPD, Chapter 1
Allergic reactions, Chapter 30

INTRAOPERATIVE HYPERTENSION

An episode of hypertension is generally defined as an elevation of blood pressure over 15% of the patient's baseline (the baseline being determined

by a series of recordings) or a systolic BP greater than 160 mmHg and/or a diastolic BP greater than 95 mmHg. Episodes of hypertension are relatively common during anaesthesia. Whether it is ultimately harmful to the patient depends on its degree, cause and duration, and on the condition of the patient. These factors also govern how actively it is treated.

Mean arterial (MAP) is determined by the systemic vascular resistance (SVR) and cardiac output (CO):

$$MAP = CO \times SVR$$

The most common cause of an increase in MAP is a raised SVR due to vasoconstriction. A raised BP does not imply a raised CO. Indeed, the increased afterload due to vasoconstriction often causes a reduced CO.

COMPLICATIONS OF INTRAOPERATIVE HYPERTENSION

- Myocardial ischaemia (especially subendocardial), myocardial infarction or heart failure.
- Haemorrhage from the operation site.
- Rupture of an existing aneurysm.
- Encephalopathy, cerebral oedema or cerebral haemorrhage.
- Severe hypertension may precipitate acute renal failure.

MANAGEMENT

If severe and life-threatening, (e.g. MAP >150 mmHg with signs of myocardial ischaemia), immediate therapy is warranted. Otherwise, seek the cause and treat that. If there is no likely cause, nonspecific therapy may need to be instituted.

CAUSES

PRE-EXISTING HYPERTENSION

- The AAGBI recommends controlling blood pressure prior to elective surgery to <160 mmHg systolic and <100 mmHg diastolic

measured in primary care (<180/110 measured in secondary care).
- Omission of regular antihypertensives during fasting may cause rebound hypertension.
- Assess preoperative therapy and administer appropriate drug, or use nonspecific therapy.

DRUG ERROR/SIDE EFFECT

- The wrong drug, dose or mode of administration (typically unintentional administration of vasopressors). Consider administration by surgeon (e.g. infiltration of adrenaline with local anaesthetic).
- Failure of administration of anaesthetic agent, e.g. due to equipment failure.
- Ketamine, ergometrine, desflurane anaesthesia (greater than 1.0 MAC) may cause hypertension.

Treatment

- Careful handling and labeling of all drugs.
- Use dedicated intravenous lines or locate connection close to patient to reduce risk of variable administration rate.
- Vasodilators may be indicated in cases of excessive/erroneous vasopressor administration.

INADEQUATE ANAESTHESIA/ ANALGESIA

- Usually accompanies a change in level of stimulation (e.g. movement of endotracheal tube) or a waning of drug effect. It is usually associated with tachycardia (bradycardia if vagal tone increased), lacrimation, tachypnoea, movement or laryngospasm.
- Treatment may include increasing anaesthesia and/or analgesia and temporarily reducing stimulation.

ANXIETY DURING REGIONAL ANAESTHETIC TECHNIQUES

- Reassure the patient and give sedation, if necessary.

INADEQUATE VENTILATION

- Carbon dioxide retention causing catecholamine release.

Treatment

- Check equipment and correct fault.
- Optimize airway/ventilation.
- Consider instituting IPPV.

DRUG INTERACTION

- For example, monoamine oxygenase inhibitors + vasopressors or opioids (especially pethidine).
- May require drug therapy (e.g. beta blockers or sodium nitroprusside).

ARTIFACT

- Use of the wrong size BP cuff (too small a cuff will result in over-estimation of BP).
- Resonance in the arterial catheter.
- Incorrect zero point.

Management

- Use appropriate cuff.
- Calibrate arterial line and compare to cuff BP.
- Use correct tubing or clamping device.
- Check zero point.

TOURNIQUET PAIN

- Slow onset, often after 1 h.
- Bilateral tourniquets with exsanguination may cause sufficient fluid shift to increase blood pressure.

Treatment

- Preoperative clonidine (3 mcg kg^{-1} IV) blunts hypertensive response.
- Consult with surgeon.
- May need drug therapy.

PRE-ECLAMPSIA

- Treat with magnesium sulphate and hypotensive agents.

PHAEOCHROMOCYTOMA

- Rare but important.
- Hypertensive crisis is associated with a high perioperative mortality (as high as 80%).

Treatment

- If suspected, a small bolus dose of phentolamine (1–5 mg) usually gives a significant fall in BP (if systolic BP falls more than 35 mmHg, phaeochromocytoma is likely).
- Give alpha blockers in addition to beta blockade (beta blockade alone may worsen vasoconstriction).
- Remifentanil may be useful.

RARE CAUSES:

- Fluid overload
- Aortic cross-clamping
- Hyperthyroid storm
- Malignant hyperthermia
- Raised intracranial pressure
- Interference with carotid body, brainstem or spinal cord
- Bladder distension (especially postop)
- Alcohol or addictive drug withdrawal
- Autonomic hyperreflexia

NONSPECIFIC TREATMENT

If the cause of hypertension cannot be removed or diagnosed, the following may be useful.

VASODILATORS

- *Anaesthetic agents* (e.g. isoflurane, sevoflurane, propofol) – Easy to titrate.
- *Hydralazine* – Arteriolar dilator, peak action after about 20 min following 5–10 mg slow IV.
- *Glyceryl trinitrate* – Arterial and venous dilator; dose 10–200 mcg min^{-1} by IV infusion.
- *Labetalol* – Combined alpha and beta blockade; dose 10–50 mg slow IV, repeated after 5 minutes if necessary, maximum dose 200 mg.

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- *Sodium nitroprusside* – Arteriolar dilator; very rapid response; administer by continuous IV infusion (0.5–1.5 mcg kg^{-1} min^{-1} starting dose, increased every 5 minutes in 500 ng kg^{-1} min^{-1} graduations, according to response); large doses may cause cyanide poisoning.

BETA BLOCKERS

- *Atenolol* – Cardioselective; dose 2.5 mg slow IV, repeated after 5 minutes if necessary.
- *Esmolol* – Rapid onset; short half-life (9 min); 50–200 mcg kg^{-1} min^{-1} infusion.

ALPHA BLOCKERS

- Phentolamine (1–5 mg IV)

POSTOPERATIVE CARE

- Continue to monitor patient.
- Provide adequate analgesia.
- Administer oxygen, titrated to SpO$_2$ of 94%–98% (reduce myocardial ischaemia).
- May need continuing therapy.
- May need investigations to exclude complications (e.g. myocardial infarctions) or to identify cause of hypertension.

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CROSS-REFERENCES

Pre-eclampsia, Chapter 12
Intra-operative arrhythmias, Chapter 30
Hypertension, Chapter 2
Phaeochromocytoma, Chapter 10

LOCAL ANAESTHETIC TOXICITY

Considering the large numbers of local anaesthetics administered, the frequency of toxic reactions is very small. The most important considerations are CNS and cardiac toxicity. Causes are related to elevated plasma drug levels. This is due to:

- Accidental (or misinformed) overdose.
- Inadvertent intravenous injection.

There is a general relationship between plasma levels of local anaesthetics and symptoms and signs of toxicity (Table 30.17).

Initial excitation is due to selective inhibition of inhibitory pathways in the CNS. With increasing blood levels there is an inhibition of both inhibitory and facilitatory pathways, leading to generalized CNS depression.

However, although a general relationship between blood levels and toxicity exists, the rate of injection (if intravenous) or uptake also influences the chance of toxicity; for example, a faster rate of injection produces signs of toxicity at lower venous plasma levels.

Table 30.17 The effect of increasing plasma drug levels

Symptom/sign	Drug level
Tingling in tongue and perioral region	Low
Dizziness	↓
Blurred vision	↓
Tinnitus	↓
Twitching and signs of CNS excitation	Intermediate
Loss of consciousness	↓
Convulsions	↓
Deep coma	↓
Respiratory and cardiac arrest	High

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METHODS OF REDUCING PLASMA LEVELS

Uptake is highest with concentrated solutions:

- Saturation of local binding sites
- Greater intrinsic vasodilating effects

Uptake is highest from highly vascular areas, such as the epidural and intercostal spaces and least from subcutaneous tissues. The principal technique for minimizing plasma levels is to reduce vascular uptake by the addition of adrenaline.

PHARMACOLOGY OF LOCAL ANAESTHETIC TOXICITY

- Potency varies directly with lipid solubility.
- Cardiac depression and CNS excitability varies directly with potency.

The mechanism for cardiac depression is unclear, but some experiments suggest that it may be related to decreased intracellular calcium. In addition, at high plasma levels, there will be generalized vasodilatation compounding the vascular collapse.

The relative potencies of bupivacaine and lidocaine are about 4:1, which is similar to their relative CNS toxicities. Both the blood levels required for cardiac toxicity and the ratio of the doses required for cardiac toxicity compared to doses required for CNS toxicity suggest that bupivacaine is considerably more cardiotoxic than lidocaine, ropivacaine or levobupivacaine.

INFLUENCE OF ACIDOSIS

- The convulsive threshold is decreased.
- An increase in $PaCO_2$ leads to an increase in CBF, thus allowing more drug to enter the brain.
- A decrease in intracellular pH will increase the amount of ionized drug; this limits diffusion and prevents drug leaving the cell.
- Decreased plasma protein binding results in more free drug.

Thus, acidosis increases the chances of developing CNS toxicity and also prolongs the toxicity.

CLINICAL ASPECTS OF LOCAL ANAESTHETIC TOXICITY

SENSIBLE PRECAUTIONS PRIOR TO COMMENCING REGIONAL ANAESTHESIA:

- All resuscitation facilities and drugs must be available.
- Monitor as per AAGBI guidelines.
- Access to circulation should be secured before starting.
- Trained assistance should be available.
- Maintain dialogue with patient during performance of block.

PREVENTION

- Careful technique.
- Aspirate before injection and intermittently aspirate during prolonged injection if large volumes are used.
- Appropriate choice of drug (e.g. avoid bupivacaine for intravenous regional anaesthesia.

Ester local anaesthetics are metabolized by plasma cholinesterase. Thus, if toxic plasma levels are achieved, the toxic reaction should be short-lived (except in the rare case of atypical cholinesterase).

RECOGNISING TOXICITY

INITIAL PRESENTATION

- *CNS* – Light-headedness, tinnitus, dizziness, metallic taste, circumoral tingling or numbness.
- *CVS* – Tachycardia, hypertension.

MAJOR EFFECTS

- *CNS* – Sudden alteration in mental state (agitation, loss of consciousness, convulsion).
- *CVS* – Cardiovascular collapse (arrhythmias include bradycardia, asystole, VF).

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TREATMENT

MINOR REACTIONS

- Stop the injection.
- Observe the patient.

MAJOR REACTIONS

- Stop the injection.
- Give 100% oxygen.
- Call for help.
- Resuscitate according to standard guidelines (ABC).
- Treat convulsions (e.g. thiopental, propofol, diazepam).
- IV fluids, inotropes and vasopressors as needed.
- If a cardiac arrest occurs then proceed to ALS.
- Prolonged CPR and resuscitation may be required.
- Give an immediate IV bolus of 20% lipid emulsion 1.5 mL/kg.
- Start an IV infusion of 20% lipid emulsion at 15 mL/kg/h.
- If cardiovascular instability continues the IV bolus of lipid emulsion may be repeated twice at 5-minute intervals. The infusion rate may be increased to 30 mL/kg/h.
- Do not exceed a maximum dose of 12 mL/kg of 20% lipid emulsion.

LIDOCAINE

- A potent antiarrhythmic agent.
- Arrhythmias are uncommon after overdosage.
- At high plasma levels, decreased cardiac conduction may be seen.

BUPIVACAINE

- S enantiomer is less toxic than the R enantiomer.
- May potentiate arrhythmias.
- Exact mechanism is unknown.
- Markedly depresses the rapid phase of depolarization of the cardiac action potential and prolongs the refractory period.

- May cause one-way block leading to re-entrant arrhythmias.
- Ventricular fibrillation is common in severe toxicity.
- Pregnant women are more sensitive to cardiotoxicity.

Bupivacaine seems to be significantly associated with cardiac toxicity compared to other local anaesthetics. CPR is very difficult in bupivacaine-induced cardiotoxicity because the drug binds to cardiac muscle (exacerbated by acidosis).

LEVOBUPIVACAINE

- Single S enantiomer of bupivacaine.
- Long acting.
- Intermediate in toxicity.
- Human volunteer studies suggest lesser cardiac depression with smaller changes in the indices of cardiac contractility compared to bupivacaine.
- Human studies suggest less CNS depression compared to bupivacaine.
- Lethal dose is higher than bupivacaine.

ROPIVACAINE

Intermediate in structure and potency to bupivacaine. It is represented as the S enantiomer rather than a racemic mixture.

- Larger doses are required to produce early features of CNS toxicity and cardiotoxicity when compared to bupivacaine.
- Animal studies show similar cardiotoxicity profile in both pregnant and non-pregnant state.

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CROSS-REFERENCE

Regional anaesthesia – general principles, Chapter 29

MAJOR TRAUMA

In the UK, trauma is the most common cause of death between the ages of 1 and 35 years. Road traffic accidents account for 38,000 admissions annually and trauma patients occupy more bed-days than cancer and cardiac patients combined. The cost to the nation is around 1% of the Gross National Product.

Blunt trauma is the most common in the UK. The incidence of life-threatening injuries to different systems is

- Head 50%
- Chest 20%
- Abdomen 10%
- Spine 5%

Up to 30% of emergency operating workload may be trauma related. All anaesthetists should have an understanding of the problems they may encounter and how to deal with them.

INITIAL MANAGEMENT

Use the ATLS principles using an AcBCDE approach (or CAcBCDE in the case of catastrophic haemorrhage). The trauma team leader (usually an emergency medicine consultant) will assign roles to the trauma team. The anaesthetist is likely to be involved in airway management, transfer to and from CT scan, and subsequent operative management.

PREOPERATIVE ASSESSMENT

Often limited due to the urgency of the situation or because the patient is unconscious. If possible, talk to paramedics, relatives, and/or the patient's GP to obtain an 'AMPLE' history:

- Allergies
- Medications (elderly in particular)
- Past medical and anaesthetic history
- Last meal
- Events (time, place and mechanism of injury)

Limit investigations to those influencing management:

- CT scan – most seriously injured patients should be scanned from head to mid-thigh level as soon as is safe to do so.
- Arterial blood gases.
- Urea, electrolytes, full blood count, clotting (including fibrinogen), blood glucose.
- ECG.
- Blood group and cross-match.
- Pregnancy test in women of child-bearing age.

THEATRE PREPARATION

- Appropriate personnel
- Anaesthetic machine, ventilator
- Intubation equipment, including cricothyroidotomy set
- Equipment for vascular access
- Fluid administration devices, e.g. Level One™ infuser
- Monitors (check function and correct calibration)
- Drugs (anaesthetic, resuscitation)
- Patient-warming devices
- Equipment for positioning

PERIOPERATIVE MANAGEMENT

Establish adequate venous access before surgery; intraosseous (IO) access may be used – the proximal humerus is the best site for rapid fluid infusion but avoid IO access in a fractured bone. Check existing catheters, drains, etc. for position, function and security.

GENERAL ANAESTHESIA

The technique used is determined by the physiological status of the patient, surgical plan, availability of equipment and drugs, and experience of the anaesthetist. This is the most commonly used technique:

- Regard all patients as having a full stomach.
- Use a rapid sequence induction, but modify as indicated by haemodyamic and airway/respiratory considerations.
- Drugs used are dictated by haemodynamic status (consider ketamine).
- Avoid nitrous oxide if pneumothorax, head injury or bowel obstruction is present or operative time may be prolonged.
- If possible, induce unstable patients in theatre to reduce movement, risk of intravenous lines being dislodged, and time to surgery.

REGIONAL ANAESTHESIA

- Sympathetic block may worsen hypotension.
- Difficulty in positioning injured patients for epidural/subarachnoid block.
- Delay in achieving adequate block.
- Inadequate when surgery in different body areas.
- May be appropriate for isolated peripheral surgery.

AIRWAY

An endotracheal tube may have already been inserted during resuscitation. Check:

- Position (listen, ETCO$_2$, chest X-ray)
- Cuff integrity
- Security
- Diameter and length (an uncut tube may be preferable with facial injuries or burns)
- Do not forget to protect the cervical spine

Anticipate difficult intubation if there is

- Trauma to soft tissues of face and neck
- Midface fractures
- Actual or potential injury to the cervical spine
- Upper airway burns
- Obvious pre-existing conditions

Consider:

- Inhalational induction
- Videolaryngoscopy
- Fibre-optic intubation; awake or postinduction (though bleeding in the airway may make this impossible)
- Surgical airway, cricothyroidotomy or tracheostomy
- Double-lumen tubes if thoracotomy planned

VENTILATION

IPPV in the presence of traumatic pneumothorax requires a chest drain to prevent a tension pneumothorax from developing.

- Check air entry bilaterally by listening in midaxillary lines
- Monitor ETCO$_2$ and O$_2$ saturation
- Measure expired tidal and minute volume, rate and pressure
- Adjust FiO$_2$, I/E ratio and PEEP to optimize oxygenation and provide 'lung protective' ventilation

Difficult ventilation may be due to:

- Gastric dilatation (pass a nasogastric or orogastric tube)
- Pneumothorax or haemothorax (insert chest drain)
- Diaphragmatic hernia
- A large air leak (e.g. bronchial tear) may require a double-lumen tube.
- Check arterial blood gases.
- Aim for normocapnia, but permissive hypercapnia may be appropriate (unless there is a head injury)

CIRCULATION

Maintenance of circulating volume is more important than normal haemoglobin.

- Intravenous access with short, wide cannulae.
- Secure all intravenous lines.
- Avoid intravenous access distal to limb fractures
- Warm all fluids.

- Hartman's solution is recommended for crystalloid infusions; excessive use of normal saline can lead to acidosis and worsen prognosis.
- In cases of life-threatening bleeding, initiate the major haemorrhage protocol; transfuse blood and blood products accordingly.
- Tranexamic acid 1 g IV over 10 minutes followed by a further 1 g over 8 hours was demonstrated to improve outcomes by the CRASH2 trial. It should be given as soon as possible after injury.
- Use cell salvage where appropriate.

Measure blood pressure directly in an upper limb. This is more accurate at low pressures, allows repeat sampling of arterial blood, and is less subject to interference.

In patients who present in haemorrhagic shock due to uncontrollable bleeding, do not attempt to resuscitate to normotension, accept systolic BP 70–80 mmHg. Administration of large volumes of fluid simply leads to increased blood loss. Delay aggressive fluid administration until operative control of haemorrhage.

In persistent hypotension:

- Ensure monitors are correctly calibrated.
- Check for occult haemorrhage.
- Adjust the ventilator to give the lowest mean intrathoracic pressure.

Then consider:

- Cardiac tamponade – Low BP, pulsus paradoxus, increased central venous pressure (CVP).
  - Use fluids and maintain heart rate to preserve cardiac output. Attempt pericardiocentesis if skills available. Maintain spontaneous ventilation if possible.
- Tension pneumothorax – Low BP, high inflation pressures, hyper-resonant percussion note, deviated trachea, increased CVP.
  - Needle thoracentesis followed by chest drain. Have a high index of suspicion with rib fractures or after central line insertion.
- Neurogenic shock – Low BP, bradycardia, vasodilatation.
  - Use volume expansion initially. Atropine and vasopressors may be required.

- Septic shock – Low BP, tachycardia, vasodilatation.
  - Uncommon early after injury and usually associated with abdominal injuries.
- Myocardial infarction – Low BP, arrhythmias, pulmonary oedema, chest pain (if conscious).

DISABILITY

Beware when moving or using fractured/injured limbs. Neurovascular injuries may be worsened or caused, especially around joints. Ensure adequate manpower for safe positioning of patients. Head injury is not a contraindication to general anaesthesia, but the principles of managing intracranial pressure should be applied. Adequately protect peripheral nerves and pressure areas, particularly the eyes, when prone.

EXPOSURE

The majority of trauma patients are hypothermic. This is worsened by cold fluids and exposure of body cavities (e.g. abdomen, chest). Temperature loss is most severe in the emergency department. Cooling predisposes to arrhythmias, decreases cardiac function, causes acidosis, adversely affects coagulation, causes left shift of the oxy-haemoglobin curve and enhances anaesthetic drugs. On recovery, shivering dramatically increases oxygen consumption. Therefore:

- Warm all fluids, especially blood.
- Monitor core and peripheral temperature.
- Warm and humidify all anaesthetic gases.
- Cover all exposed parts, including head.
- Raise theatre temperature when possible.
- Cover exposed bowel with dry towels or plastic sheet.
- Limit initial surgical therapy to life saving procedures (damage control).

Remember:

- Only blood loss kills early.
- GI injuries cause problems much later.
- Everything takes longer than you think.
- It is easy to miss an injury if you rush.
- Hypothermia, acidosis, and coagulopathy only lead to more of the same.

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- The best place for a sick patient is the ICU.
- Maintain normothermia in the head-injured patient and all trauma patients.

POSTOPERATIVE MANAGEMENT

On completion of surgery, ensure the following before extubation:

- Hypovolaemia corrected
- pH normal
- PaO$_2$ acceptable
- Temperature >34°C
- Airway reflexes intact
- Adequate analgesia

Any instability requires transfer to ICU/HDU or if there is a risk of the patient developing adult respiratory distress syndrome.

Beware of problems of transfer, even over short distances.

Remember the secondary survey if not already completed.

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CROSS-REFERENCES

Difficult airway – management, Chapter 26
Complications of position, Chapter 30
Massive blood transfusion, Chapter 30
Airway and aspiration risk, Chapter 26
Emergency surgery, Chapter 25

MALIGNANT HYPERTHERMIA

Malignant hyperthermia (MH) is a major potential anaesthetic hazard for the otherwise healthy individual. The mortality from MH has declined from >70% prior to 1980 to <4%. With the use of modern monitoring equipment and the mandatory availability of intravenous dantrolene, mortality from MH should be zero. The key to prevention of death from an MH reaction is early recognition followed by an appropriate management.

MH is a genetically heterogeneous disorder. More than 30 different mutations of the RYR1 (sarcoplasmic reticulum calcium release channel gene) have been reported. In many MH families, the precise molecular defect is yet to be determined. The result of each defect is the same – an uncontrollable rise in intracellular calcium ion concentration in skeletal muscle cells caused by a triggering drug. This rise in myoplasmic calcium ion concentration explains all the clinical and biochemical features.

The nature and course of MH reactions show considerable variation. The components can be very crudely divided into metabolic and muscular, both leading to rhabdomyolysis. The balance and severity of metabolic and muscle activity components varies according to which triggering drugs have been used. This is assumed to be a reflection of the dynamics of the rise in intracellular calcium ion concentration.

SUXAMETHONIUM

Suxamethonium is thought to produce a rapid and marked rise in intracellular calcium, but its duration of effect is limited. The predominant feature is thus increased muscle activity evident as rigidity. This is sometimes generalized, but may be limited

to the jaw muscles (masseter spasm). Homeostatic mechanisms normally restore intracellular calcium towards resting levels, usually within 10 min. The muscle activity leads to extrusion of potassium ions, creatine kinase and myoglobin. The hyperkalaemia due to suxamethonium alone usually is not life-threatening but the myoglobinaemia may be sufficient to cause acute renal failure. Postoperative renal failure has been the only presenting feature in some MH-susceptible patients. Serum creatine kinase is an indicator of the degree of muscle damage, reaching a maximum (often >20,000 units) after 24 h. Although metabolic processes will have been stimulated, the duration of the stimulus is so short that the resulting clinical features are mild and usually not noticed.

INHALATION ANAESTHETICS

The nature of the response suggests that they cause a steadily increasing intracellular calcium ion concentration. Calcium, at concentrations lower than those required to activate the contractile apparatus, has other important intracellular functions namely regulation of phosphorylation, and hence activity, of various enzymes, including those of the glycolytic pathway. As intracellular calcium rises, increased carbon dioxide and lactate production occur. In the spontaneously breathing patient there is an increase in respiratory rate and end-tidal carbon dioxide; if a circle system is in use, the soda lime will be rapidly exhausted. Simultaneously, or shortly following, a tachycardia develops secondary to the effects of acidaemia on the midbrain cardiovascular centre. The blood pressure may rise or fall, presumably due to a predominant effect of local metabolites on vascular smooth muscle. An increase in oxygen consumption occurs leading to a fall in the SaO_2. Arterial blood gases show acidaemia, hypercarbia, a base deficit and usually mild hypoxaemia.

The name malignant hyperthermia was originally coined because the patient became excessively hot and then died. Hyperthermia, however, is a relatively late manifestation. When doubt exists as to whether some metabolic signs are due to MH, the finding of a rapidly rising body temperature is very persuasive.

It is commonly believed that there is a stage of an MH reaction beyond which death is almost inevitable. In the early stages, mitochondria respond to increased pyruvate production by manufacturing more ATP. This helps to limit intracellular calcium ion concentration as ATP is required for the normal functioning of the calcium pumps of the sarcolemma and the sarcoplasmic reticulum. The mitochondria themselves also sequester calcium ions, the rate being entirely dependent on the myoplasmic calcium ion concentration. In the presence of continued release or influx of calcium into the myoplasm, the intramitochondrial calcium ion concentration continues to increase until the accumulated calcium disrupts the mitochondria. Glycolysis is stimulated and the only route for further metabolism of pyruvate is to lactate. Simultaneously, there is a rapid decline in ATP production, leading to a reduced rate of calcium removal from the myoplasm, and hence setting up a vicious cycle. Muscle rigidity appears and itself restricts microvascular perfusion of the tissue in which case dantrolene administered intravenously will not reach its site of action. The perfusion of the muscle will also be compromised as muscles swell within their fascial compartments as a result of the oedema associated with mitochondrial failure and cell death.

Calcium ions also stimulate activity of some intracellular phospholipases, leading to increased turnover of sarcolemmal phospholipid. When the demand for ATP exceeds the supply, the membrane permeability increases. This results in leakage of calcium into the cell and of potassium, creatine kinase and myoglobin out of the cell. The resulting hyperkalaemia may cause cardiac arrest while, if the acute reaction is survived, myoglobinaemia can result in renal failure.

A further feature of an advanced MH reaction is disseminated intravascular coagulation (DIC). Heat itself and/or procoagulant proteins that leak from dying muscle cells can cause DIC.

The response to an inhalation anaesthetics is more rapid following suxamethonium, possibly within 15 min. Halothane seems to be the most potent trigger of the inhalation anaesthetics, but enflurane, isoflurane, sevoflurane and desflurane are also implicated.

MANAGEMENT

MH is an emergency that requires a team approach not least because of the labour-intensiveness of rapidly mixing dantrolene for injection. All anaesthetists should be familiar with the location of the dantrolene.

INITIAL MANAGEMENT

- Stop trigger agents.
- Call for help and inform the surgeon that surgery must be abandoned or finished.
- Hyperventilate with 100% oxygen via a clean non-rebreathing circuit.
- Maintain anaesthesia with an intravenous agent.
- Muscle relaxation with a non-depolarising NMBD.
- Administer 2.5 mg kg^{-1} dantrolene. Repeat boluses of 1 mg kg^{-1} as required, up to a maximum dose of 10 mg kg^{-1}
- Initiate active cooling whilst avoiding peripheral vasoconstriction (e.g. ice packs to axillae and groins, cool IV fluids, bladder, gastric or peritoneal lavage with cool fluid).

SUPPORTIVE MANAGEMENT

- Hyperkalaemia may require administration of calcium chloride, sodium bicarbonate, and/or insulin with glucose.
- Treat arrhythmias with magnesium, amiodarone or beta-blockers. Avoid calcium channel blockers as they interact with dantrolene, potentiating myocardial depression.
- Disseminated intravascular coagulation may require blood product transfusion.
- Rhabdomyloysis may result in acute kidney injury; measure CK and institute forced alkaline diuresis.
- Transfer the patient to ICU once sufficiently stable.

SUBSEQUENT FOLLOW-UP

- The diagnosis of MH is confirmed by muscle biopsy and *in vivo* contracture testing at specialist centres.
- The patient and their family members must be followed-up and counselled regarding future anaesthetics.
- An alert should be documented in the patient notes and the patient should consider wearing an alert bracelet or carrying an alert card to notify medical staff of their condition in case of future emergencies.
- Contact the 24-hour MH helpline at St James' Hospital, Leeds (Phone 07947609601).

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CROSS-REFERENCE

Masseter spasm, Chapter 30

MASSETER MUSCLE SPASM (MMS)

A high incidence of subsequent malignant hyperthermia (MH) reactions was noted in patients whose mouths had been difficult to open following suxamethonium. This association was apparent in 70% of patients given suxamethonium who developed MH. Many patients who developed MMS were consequently referred for investigation of their MH status. Of those with clinically recognised MMS as the

only abnormal feature, 28% were susceptible to MH. This figure rises to 57% with accompanying metabolic features, or 76% if it was followed by features of muscle damage, such as myoglobinuria or severe incapacity from myalgia. It therefore appeared that patients developing MMS were at high risk from MH until proven otherwise.

Some increased tension normally develops in the jaw muscles following suxamethonium in virtually all children and in a large proportion of adults. This is usually of little importance. The term MMS is therefore restricted to severe and prolonged (>2 min) episodes of resistance to mouth opening following suxamethonium.

MANAGEMENT

The immediate concern is the maintenance of a patent airway and oxygenation in a paralysed patient. Fortunately, upper airway muscle tone seems to be maintained in MMS, making facemask ventilation a viable proposition. Naso-pharyngeal airway insertion or front of neck access are options if MMS persists and ventilation is difficult or impossible.

Further anaesthetic management depends on the urgency of the surgery and the feasibility of continuing nonurgent surgery without the use of volatile anaesthetic drugs. If the surgery is not urgent and continuation with a volatile-free technique is potentially compromising, the patient should be allowed to wake up. Should the surgery need to proceed, then facemask ventilation, via a 'clean' circuit without residual volatile, should continue until MMS has eased, when a nondepolarising NMBD can be given and intubation subsequently achieved. Nondepolarising NMBDs do not resolve ongoing MMS. Maintain anaesthesia with intravenous agents.

Regardless of the decision to wake or proceed, help should be summoned and preparations made to administer dantrolene if the patient develops signs of MH.

DIFFERENTIAL DIAGNOSIS

Establish that the correct dose of suxamethonium has been given intravenously; check the ampoule, syringe and injection site. It is unusual for mouth opening to be problematic even when no neuromuscular blocking drug has been given, unless the dose of induction agent was also inadequate: this could occur if the IV cannula had 'tissued' during induction.

RECORDING OF DIAGNOSTIC PREDICTORS

Although the patient who develops MMS must be considered to be potentially susceptible to MH until proven otherwise, evidence of metabolic stimulation and other indicators of increased muscle activity increase the likelihood of MH. Therefore, the patient should be immediately observed for the presence of generalized muscle rigidity and the duration of MMS should be recorded. An accurate chart of heart rate, blood pressure, pulse oximeter, capnography and central temperature readings should be made. Blood for arterial blood gas and serum potassium analysis should be sent. In the postoperative period, the first voided urine should be analysed for the presence of myoglobin and the serum creatine kinase should be estimated at 12 and 24 h.

FURTHER INVESTIGATIONS

Refer the patient for MH testing. They should be advised that they and all members of the family should be treated as potentially susceptible to MH until proven otherwise. In the interim, the reactor should undergo electromyographic studies to exclude congenital myotonia, some variants of which can be asymptomatic.

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CROSS-REFERENCE

Malignant hyperthermia, Chapter 30

NEUROLEPTIC MALIGNANT SYNDROME (NMS)

First described in 1960, it is a rare, potentially fatal condition caused by either treatment with dopamine receptor antagonists or by withdrawal of dopamine receptor agonists.

PATHOGENESIS

There is an acute blockade of dopaminergic transmission in the:

- *Nigrostriatum* – Which produces rigidity.
- *Hypothalamus* – Which produces hyperthermia.
- *Corticolimbic system* – Which produces an altered mental state.

The clinical similarities between NMS and malignant hyperthermia (MH) suggest a common pathophysiological element. *In vitro* halothane–caffeine contracture tests have not, however, supported any intracellular association.

The pathophysiological mechanism underlying the skeletal muscle rigidity in NMS is controversial. The current view leans towards this being central in origin. This is supported by the observation that neuromuscular blocking drugs produce flaccid paralysis in NMS, whereas in MH they have no effect.

There is now believed to be a relative glutaminergic transmission excess as a consequence of a dopaminergic block and it may be that drugs which antagonize glutamate have beneficial effects. The incidence is approximately 0.2% of neuroleptic users.

CLINICAL FEATURES AND DIAGNOSIS

Early diagnosis is important. NMS usually develops over a period of 24–72 h following exposure to neuroleptic agents. This exposure can have been over a period of days or months and may even follow a low dose of a neuroleptic agent. The features may continue for up to 10 days, even after stopping the triggering agent (Table 30.18). Complications are outlined in Table 30.19.

MORTALITY

Mortality from NMS is due to respiratory failure (most common), renal failure secondary to myoglobinuria (a strong predictor of mortality) and infections following pulmonary aspiration and/or prolonged ICU admission.

Figures of 8%–30% are frequently quoted, but the number of deaths has declined in recent years. A recent observational study in the United States found that the mortality rate was 5.6%, between 2002 and 2011, with a decreasing trend in recent years.

Table 30.18 Criteria for diagnosis of NMS

Major criteria	Minor criteria
High fever	Tachycardia
Muscular rigidity	Raised blood pressure
Elevated serum creatine kinase[a]	Tachypnoea
	Altered consciousness level
	Sweating

[a] No specific laboratory markers exist. Creatine kinase may be mildly or grossly elevated.

Table 30.19 Complications

- Respiratory
  - Secondary infection
  - Aspiration pneumonia
- Cardiovascular
  - Cardiac arrhythmias
  - Pulmonary embolism
- Musculoskeletal
  - Peripheral neuropathy
  - Rhabdomyolysis → Myoglobinuria

DIFFERENTIAL DIAGNOSIS

Early diagnosis and distinction from other conditions presenting in a similar fashion is crucial in order to prevent fatalities. Lethal catatonia is a very rare acute psychiatric syndrome. Severe psychosis in the early stages makes this diagnosis more probable. Rigidity is intermittent in lethal catatonia.

Compared to MH, NMS demonstrates:

- Slow onset
- Rigidity of central origin (controversial)
- Latency of effect with dantrolene
- Uneventful anaesthesia with MH triggering agents
- Lack of familial tendency (MH is autosomal dominant)

Ethanol withdrawal, sedative hypnotic withdrawal, cocaine and amphetamine intoxication or monoamine oxidase overdoses must be excluded before NMS is diagnosed. Some of these agents may also release central serotonin resulting in the central serotonin syndrome.

Neuroleptic-induced heat-stroke is a rare condition in which heat production exceeds heat loss. Neuroleptics make this more probable due to their anticholinergic actions (inhibiting sweating) and inhibition of hypothalamic thermoregulation. Flaccid muscle tone is the major distinguishing feature.

MANAGEMENT

- Withdraw trigger agent.
- Institute basic resuscitation measures, using an ABCDE approach.
- Cool the patient.

Both dantrolene and dopamine agonists such as bromocriptine appear to reduce the mortality rate and duration of symptoms when compared to supportive measures and they can be used as sole agents or in combination. The time to clinical effect with dantrolene is slow (several days). In high doses, dantrolene may cause hepatotoxicity, and should be used with caution in patients with biochemical evidence of hepatic injury.

Bromocriptine is more rapid-acting than dantrolene, with a reduction in rigidity observed within hours of administration. It is a vaso- and venodilator and may induce profound hypotension.

Success has been reported with ECT in cases of failure of pharmacotherapy. It is possible that ECT may only treat psychosis following neuroleptic withdrawal rather than NMS.

Withdrawal of a neuroleptic agent, when treatment is required for severely psychotic patients, is obviously hazardous. Mortality following reintroduction is variable. Risk may be reduced by using low potency neuroleptic agents, cautious titration against symptoms or using non-neuroleptic alternatives, e.g. ECT.

ANAESTHESIA

Anaesthetists need to be aware of this syndrome in the context of anaesthesia for ECT. It is important to note that the technique of anaesthesia must not aggravate the muscle disorder or produce the complications of NMS.

Avoid suxamethonium in the presence of active muscle disease, as it may release potassium and induce rhabdomyolysis. Mivacurium and rocuronium with sugammadex reversal have been reported as alternatives.

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CROSS-REFERENCE

Malignant hyperthermia, Chapter 30

PERMANENT PACEMAKERS AND ANAESTHESIA

There are approximately 20,000 permanent pacemakers (PPMs) implanted annually in the UK. Indications include complete or symptomatic heart blocks, sinus node dysfunction, tachyarrhythmias (overdrive pacing), heart failure (cardiac resynchronisation) and bradycardia following cardiac transplant. Patients with pacemakers often have a history of heart disease or other associated conditions:

- Myocardial ischaemia
- Cardiomyopathy
- Congenital heart disease
- Following cardiac surgery
- Peripheral vascular disease
- Diabetes
- Hypertension

PREOPERATIVE ASSESSMENT

This should be undertaken in the context of the proposed surgical operation, especially site. The patient should be referred to the cardiology department to check the pacemaker function and type. The majority of patients with a PPM cannot undergo magnetic resonance imaging (MRI); however, 'MRI conditional' pacemakers are now available.

PACEMAKER ASSESSMENT (SEE THE PATIENT'S 'PACEMAKER CARD')

- Reason for insertion.
- Type of pacemaker.
- Pacemaker function (this is expressed as a five-letter code, see page 78).
- Date of implantation – possibility of electrode displacement if within 4 weeks; possibility of battery failure if a long time ago (the median battery life for a dual-chamber pacemaker is approximately 8 years).

- History of vertigo or syncope (possible battery failure).
- Irregular heart rate (possible competition with intrinsic heart rate).

ECG

- May indicate ischaemia or previous myocardial infarction.
- Confirm pacing capture (if pacing rate > intrinsic rate).
- No intrinsic rhythm (patient is pacemaker dependent).
- Only intrinsic rhythm seen – request cardiology department to test pacemaker function.

CHEST X-RAY

- Usual assessment of heart size and lung fields.
- Continuity of pacing leads; distal tips within cardiac cavity (especially in patients with chest trauma).

BIOCHEMISTRY

- Hyper- and hypo-kalaemia and acidosis can increase the pacing threshold. Urea and electrolytes should be analysed routinely, and blood gas analysis should be undertaken if acid-base disturbance is a possibility. Abnormalities should be corrected preoperatively.

PACEMAKER REPROGRAMMING

Sometimes it is appropriate to reprogram a pacemaker preoperatively, to avoid inappropriate responses to electromagnetic interference. An individualized approach should be adopted, depending on the proposed surgery, anaesthetic technique, the degree of pacemaker dependence, and the existing programming of the pacemaker. Consult the cardiology team responsible for the implantation and care of the PPM. In many circumstances when the risk of electromagnetic interference can be minimized reprogramming is not required.

ADDITIONAL PREPARATION

A magnet may convert the pacemaker to a fixed rate if necessary. Modern pacemakers can have different responses to the placement of a magnet over the pacing box and the cardiology department should be consulted in order to confirm if the device is magnet-responsive, and what mode is activated by magnet placement.

PERIOPERATIVE MANAGEMENT

MONITORING

- Routine minimal monitoring.
- Invasive monitoring if indicated for operation.
- Caution: possibility of entanglement with pacing wires if inserting central venous catheters. Consider the femoral vein if central access is essential.
- Nerve stimulators can interfere with pacing (caution when using for brachial plexus blocks).

ANAESTHETIC TECHNIQUE

- Consider local anaesthesia.
- Vasodilatation may be poorly tolerated with fixed heart rates.
- Volatile anaesthetics may increase atrioventricular delay and pacing threshold; total intravenous anaesthesia may be preferable.
- Caution with suxamethonium:
  - Acute release of potassium may increase pacing threshold.
  - Myopotentials during fasciculation may be abnormally sensed.
- Avoid hypovolaemia.

DIATHERMY

- Use bipolar diathermy if possible.
- If unipolar diathermy must be used, attach plate as far away from chest as possible.
- Ensure that any current flow does not traverse the chest.
- May need to convert pacing state to fixed mode if diathermy around chest.

- There is a possibility of 'phantom reprogramming' due to electromagnetic induction.
- Alternative pacing methods (e.g. pacing defibrillator) should be immediately available if diathermy near to the pacing box is unavoidable.

CHRONOTROPIC DRUGS

- Atropine (0.5–3 mg) – may not be effective.
- Isoprenaline (1–10 mcg min^{-1} infusion) – note isoprenaline can decrease systemic vascular resistance.
- Ephedrine (3–6 mg boluses) – α and β effects.
- Adrenaline (1:100 000; 0.5–1 mL boluses).

POSTOPERATIVE MANAGEMENT

- Continue with ECG monitoring.
- Check that pacemaker programme is correct.

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Cardiomyopathy, Chapter 2
Coronary artery disease, Chapter 2
Patients with permanent pacemakers, Chapter 2
Preoperative assessment of cardiovascular risk in noncardiac surgery, Chapter 25

POSTOPERATIVE PAIN MANAGEMENT

The aim is to provide safe, effective, individualised pain relief to enable quick return to appropriate functional activity.

EFFECTS OF UNCONTROLLED PAIN

Pain after surgery is natural but not inevitable or harmless. Uncontrolled pain increases sympathetic activity and the stress response. It has multisystem consequences which may include hyperglycaemia, immunosuppression and an increased risk of myocardial ischaemia. Pain after abdominal or thoracic surgery can lead to splinting of the diaphragm and chest wall causing decreased lung volumes, atelectasis, poor cough, sputum retention, infection and hypoxia. Pain can also reduce mobility and increase risk of thromboembolism. The psychological impact of sustained acute pain can include anxiety and a feeling of helplessness, hampering recovery. Poorly controlled postoperative pain predisposes to the subsequent development of chronic pain. The very young and very old are at greater risk from the adverse effects of uncontrolled pain after surgery.

ASSESSMENT OF PAIN

The first step in management, assessment should be at regular, frequent intervals and after every intervention to treat pain. The frequency should be determined by the severity of pain and the effectiveness of the analgesic regimen. Pain should be recorded as the fifth vital sign along with blood pressure, heart rate, respiratory rate and temperature.

A thorough assessment should include:

- Site, circumstances associated with onset (details of surgery).
- Character (helpful in identifying visceral and neuropathic components to postoperative pain).
- Intensity (both at rest and on movement).
- Associated symptoms (e.g. nausea).
- Effect on activity and sleep.

- Relevant medical history (history of pain conditions and treatments, as well as other medical conditions).
- Other factors influencing the patient's treatment (belief concerning the causes of pain, expectations of pain relief and reduction required to resume reasonable activity).
- Current and previous medications and pain-management strategies.
- Relevant past medical history.

Measuring the severity of pain reliably can be difficult as pain is a subjective, physical and emotional response to potential or actual tissue damage. The main tools available fall into two categories:

Unidimensional scales are numeric (e.g. numeric rating scales, visual analogue scale) and categorical (verbal descriptor scale). These can be used as pain relief scales where '0/none' is no relief and '10/complete' is complete pain relief. They measure one aspect of pain, usually intensity.

Multidimensional scales (e.g. McGill Pain Questionnaire) are less useful in acute postoperative pain. However, unidimensional scales are unhelpful in identifying neuropathic pain and scales such as the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS) can be used postoperatively to identify those at risk of developing chronic neuropathic pain.

Pain assessment in special circumstances such as children and cognitively impaired patients may need pictorial scales and behavioural scales (e.g. FLACC scale, Abbey pain scale). The scale used should be easy to use and one that the patient understands and provides consistent responses.

BASIC PRINCIPLES OF ACUTE POSTOPERATIVE PAIN MANAGEMENT

Good analgesia begins before surgery. Planning an analgesic strategy forms part of the preoperative anaesthetic assessment.

The use of multimodal analgesia, preventive analgesic strategies, providing regular baseline analgesia, making provision for breakthrough/activity induced pain and prescribing for side-effects of analgesia

Table 30.20 Pain management strategy

Anticipated pain	Mild	Moderate	Severe
Pain score (rest/ movement)	1–3	4–7	7–10
	Step I	Step II	Step III
Regular	**Simple analgesics**	**Add**	**Add**
(At timed intervals)	Paracetamol	NSAIDs	Strong opiate
	and/or	±	**Replace**
	Low dose weak opiates	Weak opiates	Weak opiates with strong
	Use	**Use**	opiates
	Local infiltration or regional	Local infiltration or regional	**Consider**
	blocks whenever possible	blocks whenever possible	Epidural/PCA
As required	NSAIDs	Short-acting opiates	Short-acting opiates
(prn)	or	(e.g. morphine oral/IV/IM)	(e.g. morphine oral/IV/IM)
	Weak opiates		PCA/PCEA

form the basis of a good postoperative analgesic strategy.

The choice of analgesia can be guided by the principles outlined in Table 30.20 (modification of the WHO pain ladder). Start at the step in the ladder appropriate to the anticipated pain intensity. Use the oral route whenever possible.

If regular analgesia from one section is insufficient:

- Exclude a surgical or medical complication.
- Check that prescribed analgesia has been administered correctly.
- Consider moving to next step.
- Increase dose of step III agent at regular intervals if pain remains severe.

REGIONAL TECHNIQUES

ADVANTAGES OF REGIONAL TECHNIQUES IN POSTOPERATIVE PAIN

- Better postoperative analgesia.
- Decrease opiate use leading to a decrease in
  - PONV
  - Itching
  - Sedation and respiratory depression
  - Unplanned admission after ambulatory surgery
- Better patient satisfaction.
- Better dynamic pain relief compared to opiate analgesia.
- Reduction in length of stay in some cases.

Regional techniques for analgesia fall into three main categories: neuraxial blocks, peripheral nerve blocks and local infiltration. These can be single or continuous blocks. Timing may have some bearing on the quality of postoperative analgesia. Instituting the blocks before the stimulus has been shown to improve postoperative analgesia in some settings and may reduce anaesthetic and analgesic requirements intraoperatively.

Intrathecal and epidural opiates can be a useful adjuvant to postoperative analgesia. Extended release epidural morphine preparations were shown to provide analgesia for up to 48 h after surgery but are no longer available. Issues with neuraxial opiates include delayed respiratory depression, urinary retention, pruritis and sedation. Care needs to be exercised to avoid other sedating agents when neuraxial opiates are used. Areas caring for these patients should have clear and specific guidelines for monitoring and for management of complications and side effects.

Peripheral nerve blocks can be used for intraoperative and postoperative analgesia. Benefits need to be balanced against the risk of nerve injury, and the possibility of motor block and loss of function, which may delay onset of active physiotherapy. Some of the problems can be offset by using low dose local anaesthetics combined with adjuvants.

TAP blocks have been shown to improve analgesia, decrease opiate use and improve function in laparoscopic cholecystectomy, total abdominal

hysterectomy, caesarean section and laparotomy for bowel surgery. However, not all studies are in agreement and the place of TAP blocks in anaesthetic practice is a matter of current debate. Ultrasound-guided placement is advised due to the relative frequency of incorrect needle placement using the landmark technique. Likewise, the reported outcomes of rectus sheath blocks are mixed.

Continuous wound infiltration with local anaesthetic through a multi-hole catheter placed at the end of surgery provides similar analgesic outcomes to epidural analgesia for abdominal surgery in terms of 24-hour pain scores, with less risk of urinary retention.

ADJUNCTIVE ANALGESIC MEDICATIONS

There is increasing interest in the role of opioid-sparing adjunctive analgesic medications (e.g. ketamine, pregabalin, gabapentin, alpha-2 agonists). In addition to reducing opioid consumption (and, hence, side effects) theoretical benefits include the prevention of chronic postsurgical pain. Though there is growing evidence for these medications, their administration for the treatment or prevention of acute postoperative pain is off-license. Dosing regimens are determined locally.

PAIN IN SPECIAL CIRCUMSTANCES

OPIOID-DEPENDANT PATIENTS

These patients belong to one of three groups, namely, those who are prescribed long-term opioids for chronic pain, those who are prescribed opioids for cancer pain and those who have been or are currently using opioids recreationally. Management of pain after surgery is difficult in this group of patients. All patients on long-term opiates will be tolerant but not necessarily addicted. The aim of management should be to manage patient expectations, provide adequate analgesia and prevent or manage withdrawal symptoms. This can be achieved by involving the multidisciplinary team (local drug team, acute pain team, psychologist and pharmacist) early in the process. Start with a thorough preoperative evaluation and set

achievable targets. It is also useful in current users of recreational drugs to contact local drug teams to gauge baseline use from street values of drugs. Patients require continuation of their baseline opioid (or equivalent) to avoid withdrawal, with supplementation to provide analgesia for acute pain. Due to its opioid-sparing effect, regional anaesthesia should be used whenever possible.

ACUTE NEUROPATHIC PAIN AFTER SURGERY

The incidence of neuropathic pain after surgery is highly variable, depending on the surgery performed, e.g. approximately 85% of patients develop neuropathic pain following limb amputation. Mechanisms are poorly understood but pre-emptive analgesia such as regional techniques, ketamine and administration before the start of surgery may be useful. Diagnosis is facilitated by maintaining a high index of suspicion in patients who undergo surgical procedures known to be associated with the development of neuropathic pain (e.g. limb amputation, thoracic surgery, breast surgery), and the use of multidimensional pain assessment tools (e.g. LANSS).

Most of the evidence for the treatment of acute postoperative neuropathic pain is extrapolated from chronic neuropathic pain states. Current guidelines suggest that tricyclic antidepressants, ketamine, anticonvulsants, lidocaine and tramadol may have a role.

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CROSS-REFERENCES

Regional anaesthetic techniques, Chapter 29
Epidural, spinal and CSE, Chapter 29

PSEUDOCHOLINESTERASE DEFICIENCY

Pseudocholinesterase (PChE) is also known as plasma cholinesterase and butyrylcholinesterase. It is an enzyme predominantly synthesised in the liver which acts in the plasma to metabolise a number of choline-based esters.

The importance for anaesthesia is that decreased PChE activity may cause a reduction in the rate of hydrolysis and hence a prolonged duration of action of succinylcholine and mivacurium. Note that acetylcholine and non-choline-based esters such as remifentanil are not broken down by this pathway and

Table 30.21 Aetiology of decreased PChE activity

Physiological variation	
Gender	Males > females
Age	Newborns 50% of adults
Pregnancy	70%–80% of pre-pregnancy level until 6–8 weeks after delivery
Disease	
Liver failure	40%–70% decrease
Renal failure	10%–50% decrease
Malignancy	25%–50% decrease, depending on the localization
Severe burns	Lowest value 5–6 days after the injury Depending on the degree of injury, the reduction may exceed 80%

Table 30.22 Iatrogenic factors which decrease PChE activity

Factor	Decrease in PChE activity (%)
Glucocorticoids, oestrogen	30–50
Cytotoxics	35–70
Neostigmine	5–100
Ecothiopate eye drops	70–100
Bambuterol	30–90
Organophosphates	100
Plasmapheresis (removes PChe)	60–100

are thus unaffected by PChE deficiency. Decreased PChE activity may be inherited or acquired (Tables 30.21 and 30.22).

GENETICALLY DETERMINED CHANGES IN PChe ACTIVITY

More than 60 different mutations of PChE are known. Only a few of these have any clinical significance due to the production of poorly functioning mutant enzyme or a lack of PChE production. The cholinesterase activity of the different phenotypes differs qualitatively as well as quantitatively. It is not possible to estimate the clinical significance from measuring PChE levels alone. The phenotype (or genotype) must also be identified. (See Table 30.23.)

Table 30.23 Frequency of biochemical characteristics using benzoylcholine as a substrate of the clinically most important PChe phenotypes in populations of European descent

Phenotype	Frequency (%)	Dibucaine number	Duration of muscle relaxation
UU	98.7	>70	≈5 minutes
UA	1.23	31–70	10–30 minutes
AA	0.07	14–27	2–8 hours

Note: Some populations, e.g. those of Inuit or Persian Jewish descent, have a higher incidence of PChe deficiency.
Abbreviations: U = usual, A = abnormal.

DETERMINATION OF PChE PHENOTYPE

Analysis of PChE deficiency can be undertaken by measuring the rate of hydrolysis of a substrate catalysed by PChE (e.g. benzoylcholine). It is described in terms of the percentage inhibition of the hydrolysis by an inhibitor substance, usually dibucaine. The percentage inhibition is equivalent to the 'dibucaine number'. Less active, mutant forms of PChE are resistant to dibucaine inhibition and hence are assigned a lower dibucane number than wild-type PChE. Individuals with normal PChe activity have a dibucaine number of 75–85.

CLINICAL CONSEQUENCES

The clinical implications of decreased PChE activity are principally in relation to suxamethonium. Normally 90% of an injected dose is hydrolysed in plasma and only a small amount reaches the neuromuscular junction. If PChE activity is decreased, more succinylcholine reaches the receptor causing a profound block with a prolonged duration of action.

When there is a reduced activity in phenotypically normal patients, the duration of action may be moderately prolonged (20–45 min).

In patients that are heterozygous for the normal and the atypical genes, (without any other factors which might reduce PChE activity) the duration of action may be slightly prolonged (10–30 min). More prolonged paralysis and a phase 2 block may be seen if the PChE activity is further decreased by other reasons (Tables 30.21 and 30.22).

In patients homozygous for two abnormal genes, a very prolonged response (120 min or even longer) and a phase 2 block are always seen.

MANAGEMENT OF PROLONGED RESPONSE TO SUCCINYLCHOLINE OR MIVACURIUM

Management depends on the PChE activity and the phenotype. Often the patient's phenotype is unknown. Therefore, it is necessary to keep the patient sedated and ventilated and regularly evaluate the response to peripheral nerve stimulation.

Transfusion of fresh frozen plasma may antagonize the block due to the administration of allogenic PChE. However, because of the risks associated with its use, transfusion cannot be recommended as a routine measure.

Neostigmine can be used to antagonise the neuromuscular blockade of mivacurium but as it inhibits PChE it will further prolong its metabolism. A peripheral nerve stimulator must therefore be used in order to ensure the return of at least two twitches on the train-of-four before administration.

PATIENT FOLLOW-UP

The patient should be informed about the prolonged block. Enquiries should be made to determine if there have been any episodes of awareness during attempts to wake. If so, explanation, apology and psychological support should be offered. Blood samples should be drawn for determination of PChE activity and phenotype after at least 24 hours (suxamethonium itself inhibits PChE). Warning cards should be issued to the patient and the patient's general practitioner should be informed. Because of the genetic component, testing of the patient's family members may also be appropriate.

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RAISED INTRACRANIAL PRESSURE/CEREBRAL BLOOD FLOW CONTROL

The components within the skull are brain, blood and cerebrospinal fluid (CSF). The Monroe Kellie doctrine states that an increase in the volume of one component must be accompanied by an equal reduction in the volume of the others in order to maintain the same intracranial pressure (ICP) (Figure 30.8).

The initial compensatory mechanism for an increase in the volume of brain matter or blood is the extrusion of CSF into the spinal sac. When this mechanism is exhausted, further volume increases result in a sudden large increase in ICP. Raised ICP eventually impedes cerebral perfusion which if unchecked initiates a 'vicious cycle' of hypoperfusion, ischaemia and cerebral oedema.

Further brain swelling causes:

- Herniation of the temporal lobe through the tentorium and of the cerebellar tonsils through the foramen magnum.
- Brainstem torsion with reduced cerebral blood flow (CBF) and sudden obstruction of CSF flow with acute hydrocephalus.
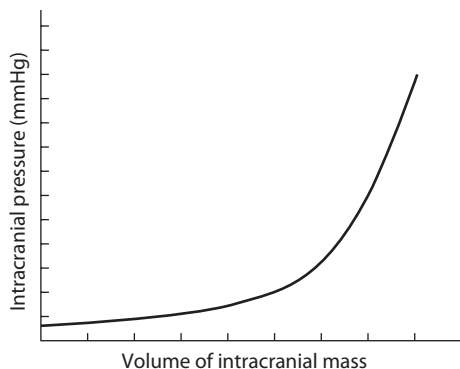
Figure 30.8 Effect of increasing intracranial mass on ICP. Small changes in ICP, initially, become much greater beyond a critical intracranial mass.

CLINICAL SIGNS AND SYMPTOMS OF RAISED ICP

- Headache, worse after waking, which is exacerbated by lying down.
- Nausea and vomiting.
- Papilloedema.
- Decreased conscious level.
- Hypertension and bradycardia.
- Abnormal respiratory pattern.

CAUSES OF RAISED ICP

- Traumatic brain injury
- Space-occupying lesions (e.g. tumour, abscess)
- Haemorrhage
- Venous thrombosis
- Infection (e.g. meningitis, encephalitis, abscess)
- Hydrocephalus
- Metabolic encephalopathy (e.g. hypoxia, hypercapnoea, hypoglycaemia, hepatic)
- Status epilepticus

AGGRAVATING FACTORS

- Venous obstruction (e.g. from poor neck positioning, tube ties, etc.)
- Raised intrathoracic pressure.
- Fibre-optic bronchoscopy increases the ICP significantly but transiently. This is not completely abolished by sedation, analgesia or muscle relaxation.

AMELIORATING FACTORS

- A head-up tilt of 30° gives maximal benefit from venous drainage, whilst minimizing the reduction in cerebral arterial pressure due to the hydrostatic pressure difference between the heart and brain level.

CEREBRAL PERFUSION PRESSURE (CPP)

CPP is defined as the difference between the systemic MAP and the ICP. In health, it is typically 70–85 mmHg. In patients with traumatic brain

injury, guidelines typically advise maintaining a CPP of 50–70 mmHg.

CEREBRAL BLOOD FLOW

Normal cerebral blood flow (CBF) is approximately 50 mL min^{-1} per 100 g of brain tissue. Of the three intracranial components described by the Monroe Kellie doctrine, blood is the only one that can be manipulated nonsurgically. The principles of managing CBF are therefore fundamental to the initial management of the patient with raised ICP. Autoregulation of cerebral blood flow is maintained between a mean arterial pressure (MAP) of 50 and 150 mmHg; however autoregulation can be impaired in brain injury and with the use of various medications, including volatile anaesthetic agents.

REGULATING FACTORS

- Cerebral metabolic rate for oxygen ($CMRO_2$) – CBF increases proportionally with increasing $CMRO_2$. This is increased by pyrexia, pain, agitation and seizures.
- PaO_2 – there is little change in CBF above a PaO_2 of 6.7 kPa. Below this, CBF increases.
- $PaCO_2$ – there is a near-linear relationship between CBF and $PaCO_2$ between 2.7 kPa (maximal vasoconstriction) and 10.6 kPa (maximal vasodilation). Hyperventilation reduces CBF only temporarily, with a return to normal values after approximately 5 hours.
- Other (e.g. autonomic innervation, local factors).

PHARMACOLOGICAL AGENTS THAT AFFECT CBF

Inhalational agents

Isoflurane has an indirect vasoconstricting effect secondary to a reduction in $CMRO_2$ and a direct vasodilating effect. Isoflurane provides cerebral protection and ischaemic changes do not develop until CBF is reduced to 8–10 mL min^{-1} 100g^{-1}. At >1.5 MAC or in the damaged brain the vasodilating effect predominates.

Sevoflurane has similar effects to isoflurane. Autoregulation of cerebral blood flow is preserved up to 1.5 MAC.

Desflurane produces complete abolition of autoregulation at 1.5 MAC and is a more potent cerebral vasodilator than isoflurane or sevoflurane at equivalent MAC dose. It is frequently used in neuroanaesthetic practice at a low dose in combination with remifentanil.

Nitrous oxide causes significant global increase in CBF by direct vasodilatation.

Hypnotics

Propofol reduces ICP, but may also reduce CPP due to a fall in MAP. In patients with intracerebral tumours, there is less cerebral swelling after opening the dura than when volatiles are used. Propofol without narcotic does not prevent the rise in ICP on intubation.

Barbiturates and midazolam produce a dose-dependent reduction in $CMRO_2$ and cerebral blood flow.

Muscle Relaxants

Suxamethonium may cause a small, transient increase in ICP. However, it is commonly used in rapid sequence induction for patients with traumatic brain injury. Non-depolarising muscle relaxants do not have any effect on ICP.

Opioids

In the absence of controlled ventilation, opioids will increase ICP secondary to hypercapnia from respiratory depression. In addition, some studies suggest that opioids may increase CBF, though the mechanism is poorly understood.

Osmotic agents

The initial effect of a bolus of mannitol is haemodynamic, augmenting intravascular volume and MAP and CPP. With intact autoregulation, cerebral vasoconstriction and a decreased metabolic rate reduce ICP. In addition, plasma expansion reduces blood viscosity, improving cerebral microvascular perfusion. The osmotic effect occurs after approximately 15 minutes, and removes water from the brain tissue down an osmotic gradient. This osmotic effect relies on the presence of an intact blood–brain barrier

and both the haemodynamic and osmotic effects are less effective in the damaged brain. Mannitol subsequently induces an osmotic diuresis, which may reduce MAP and CPP.

Hypertonic saline is an alternative to mannitol and is becoming more frequently used in clinical practice. A number of trials suggest that hypertonic saline is more effective than mannitol in reducing ICP, and presents an advantage in that it does not induce an osmotic diuresis.

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ROBOTIC SURGERY

Potential benefits of using robotic telemanipulators for minimally invasive surgery include reduced surgical trauma, shorter hospital stay and increased surgical precision.

The most common commercially available robot is the da Vinci system, which comprises three units: a console, an instrument tower, and the operating robot. The surgeon sits at the console, controlling the robot and viewing a 3-dimensional image of the operative field streamed from a stereoscopic videoscope mounted on the robot via the instrument tower. The robot itself has four arms (earlier versions had three). One holds the video scope and the other three perform procedures with a variety of interchangeable instruments. The benefits over other forms of minimally invasive surgery (e.g. laparoscopy) are principally the 3-dimensional nature of the image which affords depth perception, the electronic filtering of hand tremor, and motion scaling in which a large movement by the surgeon is translated to a small movement by the robot, increasing precision. Though the surgeon is remote from the patient, scrubbed assistance is required.

Robotic surgery has thus far been used in general surgery, ENT, cardiothoracic, gynaecology, neurosurgery and urology. Robotic-assisted prostatectomy is currently the most common robotic surgical procedure.

ANAESTHETIC CONSIDERATIONS

PATIENT ACCESS

- The large size of the operating robot, plus the presence of the other units, restricts access to the patient and mobility around the operating theatre. Depending on the operative site, the patient's airway access may be limited (e.g. in ENT or thoracic surgery) and removing the robot from the patient is slow when compared to a human surgeon stepping back from the operative field. Tracheal intubation and large-bore IV access are recommended and must be thoroughly secured.

POSITIONING

- Unusual patient positions may be required (e.g. steep Trendelenburg in robot-assisted prostatectomy). Preoperative assessment should be undertaken in the context of the anticipated patient position. Padding and special equipment may be used to prevent pressure injuries and aid positioning (e.g. shoulder braces). Once the procedure has commenced it is impossible to reposition the patient without resetting the robot, so meticulous attention should be paid to safe and secure positioning. Steep Trendelenburg

has been associated with cerebral and airway oedema, and reflux of gastric secretions. IV fluids should be administered with caution, and antacid premedication should be considered.

PNEUMOPERITONEUM/ CAPNOTHORAX

- In pelvic or abdominal surgeries a pneumo-peritoneum is required. This, combined with the steep Trendelenburg position, may displace the carina, causing endobronchial intubation and absorption of CO_2 may further increase intraocular and intracranial pressure (ICP). Tube position should be checked after positioning and again after insufflation of the abdomen. Patients with glaucoma or raised ICP are unlikely to be suitable for such procedures. Thoracic surgery avoids the positioning problems of steep Trendelenburg but hypercapnoea remains a potential concern.

DURATION

- Robotic surgery can be very prolonged in comparison with equivalent non-robotic procedures. Pressure areas must be appropriately protected, and thromboprophylaxis administered as appropriate.

COMMUNICATION

- Because the surgeon is remote from the patient and views the operative site on a shrouded monitor, communication can be difficult. Strategies such as closed-loop communication may be of particular relevance in this circumstance.

ANALGESIA

- One of the benefits of robotic surgery is reduced surgical trauma and analgesic requirements are likely to be less than in the equivalent non-robotic procedure. In the rare event that epidural is sited, it should not be topped-up while the patient is in steep Trendelenburg to avoid cranial spread.

It is likely that as technology progresses and healthcare workers become more familiar with robotic surgery some of these potential issues will diminish in their significance. However, it is also likely that the use of robotic surgery will expand into new surgical specialties and procedures, ensuring that the learning curve is maintained for some years to come.

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CROSS-REFERENCES

Complications of position, Chapter 30
Airway and aspiration risk, Chapter 26
Prolonged anaesthesia, Chapter 28

THROMBOSIS AND EMBOLISM

A thrombus is a blood clot that forms within a blood vessel. If a fragment breaks off and is carried along to a distant site, thromboembolism results. The effect of the embolus depends on where it ultimately lodges. If it originates from the systemic venous side (venous thromboembolism, VTE), it will lodge in a pulmonary vessel (pulmonary embolus, PE). If it originates from the systemic arterial side, it will lodge in a peripheral artery. The resultant symptoms and signs depend on the vessel occluded and the size of the embolus. Thromboembolic disease is a major cause of morbidity and mortality, much of which can be prevented with simple prophylactic measures.

Factors contributing to thrombus formation may be considered in terms of Virchow's triad:

- Abnormality of the endothelium of the blood vessel (e.g. trauma, shear stress, inflammation).
- Slowing or other disturbances of blood flow (e.g. prolonged immobility, varicose veins).
- Hypercoagulability of the blood (e.g. due to hyperviscosity, clotting factor mutations, or secondary to pregnancy or malignancy).

DEEP VEIN THROMBOSIS

Deep vein thrombosis (DVT) is a common event in hospital patients. Patients most at risk are listed in Table 30.24.

Table 30.24 Risk factors for venous thromboembolism

Patient factors
- Age
- Obesity
- Varicose veins with phlebitis
- Immobility (bed rest >3 days)
- Pregnancy and puerperium
- Oestrogen therapy (hormone replacement therapy or oral contraceptive pill)
- Previous DVT or pulmonary embolism in patient or first degree relative
- Thrombophilias

Factors relating to disease or procedure
- Surgery or trauma, especially to the pelvis, hip or lower limb
- Long surgical procedures (>90 minutes or 60 minutes if surgery involves pelvis or lower limb)
- Malignancy, especially pelvic, abdominal or metastatic
- Heart failure
- Recent myocardial infarction
- Lower limb paralysis
- Infection
- Inflammatory conditions
- Polycythaemia
- Dehydration
- Critical care admission

DIAGNOSIS OF DVT

NICE recommends the use of a 2-level Wells Score (Table 30.25). If the Wells Score indicates that DVT is likely, an ultrasound scan of the proximal leg veins should be performed within 4 hours and if this is negative, a D-dimer should be performed. If an ultrasound scan is not available, a D-dimer test is undertaken and a dose of anticoagulant administered whilst awaiting ultrasound imaging. Patients with a positive D-dimer and a negative ultrasound should have their ultrasound scan repeated after 6–8 days.

If the Wells Score indicates that DVT is unlikely, a D-dimer test should be sent and if the result is positive the patient should be referred for an ultrasound scan which should be performed within 4 hours. If an ultrasound scan is not available, a dose of anticoagulant should be administered whilst awaiting ultrasound imaging.

Table 30.25 The 2-level Wells Score for DVT

Clinical feature	Score
Active cancer (treatment ongoing, within 6 months, or palliative).	1
Paralysis, paresis or recent plaster immobilisation of the lower extremities.	1
Recently bedridden for 3 days or more or major surgery within 12 weeks requiring general or regional anaesthesia.	1
Localised tenderness along the distribution of the deep venous system.	1
Entire leg swollen.	1
Calf swelling at least 3 cm larger than the asymptomatic side.	1
Pitting oedema confined to the symptomatic leg.	1
Collateral superficial veins (non-varicose)	1
Previously documented DVT.	1
An alternative diagnosis is at least as likely as a DVT.	–2

Source: Reproduced from National Institute for Health and Clinical Excellence. (2012). *Venous Thromboembolic Diseases: Diagnosis, Management and Thrombophilia Testing*. NICE has not checked the use of its content in this publication to confirm that it accurately reflects the NICE publication from which it is taken.

Note: A DVT is deemed to be likely if the 2-level Wells score is 2 or more.

In the postsurgical patient, D-dimer testing is unreliable as fibrin breakdown products will be detectable due to the lysis of haemostatic clots. In this instance, a lower threshold for imaging may be reasonably adopted.

TREATMENT OF DVT

Anticoagulate with either low-molecular-weight heparin (LMWH) or fondaparinux, depending on local protocols and patient factors. Unfractionated heparin (UFH) is reserved for patients with a high risk of bleeding or severely impaired renal function. Warfarin is commonly used for longer-term treatment. It may be started on the first day, but additional anticoagulants should be continued until the warfarin becomes effective.

VTE PROPHYLAXIS

The prophylactic strategy should be guided by the balance of the risks of thrombosis and bleeding. The risk of VTE should be assessed on admission, after 24 hours, and whenever the clinical situation changes.

Patients at low risk of VTE should be encouraged to mobilise and prevent dehydration. Surgical patients should be provided with mechanical prophylaxis (e.g. graduated compression stockings) unless contraindicated.

Patients at increased risk of VTE should be offered additional prophylaxis including pharmacological measures (e.g. LMWH, fondaparinux, rivaroxaban, dabigatran), as indicated by local policies, and mechanical measures such as graduated compression stockings or intermittent pneumatic compression devices.

PULMONARY EMBOLISM

Mortality from PE has decreased in recent years and is believed to account for approximately 2% of in-hospital deaths. The majority of patients who die from PE do not have a premortem diagnosis of DVT.

DIAGNOSIS AND TREATMENT OF PULMONARY EMBOLISM

Massive acute PE presents with cardiovascular collapse or cardiac arrest. The clinical features of minor PE are often nonspecific making diagnosis difficult. They depend partly on the degree of pulmonary arterial obstruction and may include tachypnoea, pleuritic or dull chest pain, tachycardia, cyanosis, raised CVP and gallop rhythm.

NICE recommends the use of the 2-level Wells Score for PE (Table 30.26) to guide investigations and management. If the Wells Score indicates that PE is likely, a CT pulmonary angiogram (CTPA) should be performed immediately. If a CTPA cannot be performed immediately, a dose of anticoagulant is administered whilst awaiting imaging.

If the Wells Score indicates that PE is unlikely, a D-dimer test should be sent and if the result is positive the patient should be referred for an immediate CTPA. If a CTPA cannot be performed immediately, a dose of anticoagulant is administered whilst awaiting imaging.

Ventilation/perfusion (VQ) scans should be reserved for patients with an allergy to contrast,

Table 30.26 The 2-level Wells Score for PE

Clinical feature	Score
Clinical signs and symptoms of DVT (minimum of leg swelling and pain with palpation of the deep veins).	3
An alternative diagnosis is less likely than PE	3
Heart rate >100 beats per minute	1.5
Immobilisation for more than 3 days or surgery in the previous 4 weeks.	1.5
Previous DVT/PE	1.5
Haemoptysis	1
Malignancy (on treatment, treated in the last 6 months, or palliative)	1

Source: Reproduced from National Institute for Health and Clinical Excellence. (2012). *Venous Thromboembolic Diseases: Diagnosis, Management and Thrombophilia Testing*. NICE has not checked the use of its content in this publication to confirm that it accurately reflects the NICE publication from which it is taken.

Note: A PE is deemed to be likely if the 2-level Wells score is 4 or more.

renal impairment or who are at high risk from irradiation (e.g. pregnant patients).

Other investigations include: ECG (common changes include tachycardia, right bundle branch block or S1:Q3:T3 in 25% of patients); chest X-ray which may show pulmonary oligaemia or wedge-shaped opacity; and blood gases, which may show hypoxaemia. Echocardiography may show an acutely dilated right ventricle.

Initial treatment is supportive with oxygen, fluids and analgesia. Specific treatment options include anticoagulation, thrombolysis and pulmonary embolectomy.

Minor pulmonary embolism with no haemodynamic disturbance can be treated with anticoagulation.

Major pulmonary embolism presenting with haemodynamic instability will initially require resuscitation with an ABC approach. This should be followed by anticoagulation with UHF and thrombolysis with streptokinase, urokinase or recombinant tissue plasminogen activator (rtPA). Note that the UHF infusion should be stopped during administration of streptokinase or urokinase and continued for several hours after thrombolysis. Subsequent anticoagulation is with LMWH or fondaparinux.

Pulmonary embolectomy is occasionally attempted in specialist centres for patients in whom thrombolysis is contraindicated or has failed.

ARTERIAL THROMBOEMBOLISM

The source of arterial thromboembolism is often the heart. The majority of emboli of cardiac origin are the result of atrial fibrillation, particularly when this is associated with mitral stenosis or thyrotoxicosis. Other predisposing conditions include prosthetic heart valves, recent myocardial infarction with mural thrombus formation and low cardiac output states. Cardiac emboli may travel peripherally, or more commonly to the cerebral circulation, producing a stroke. NICE guidelines recommend that the majority of patients will benefit from anticoagulation (similarly to VTE, the risk of bleeding and the risk of stroke must be assessed and considered). Heparin is used initially, followed by long-term treatment with apixaban, dabigatran, rivaroxaban or warfarin.

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TOTAL SPINAL ANAESTHESIA

DEFINITION

- Total spinal anaesthesia (TSA) is spread of local anaesthetic to block all of the spinal nerves and the brain stem.
- Usually results in unconsciousness, respiratory failure and severe hypotension which may progress to cardiovascular collapse and cardiac arrest
- 'High' spinal anaesthesia is the blockade of nerve roots above T4, without intracranial spread.

PHYSIOLOGICAL EFFECTS

See Figure 30.9.

CARDIOVASCULAR COLLAPSE

- Total preganglionic efferent sympathetic block.
- Peripheral sympathetic (T1–L2) block leads to loss of vasoconstrictor tone with profound
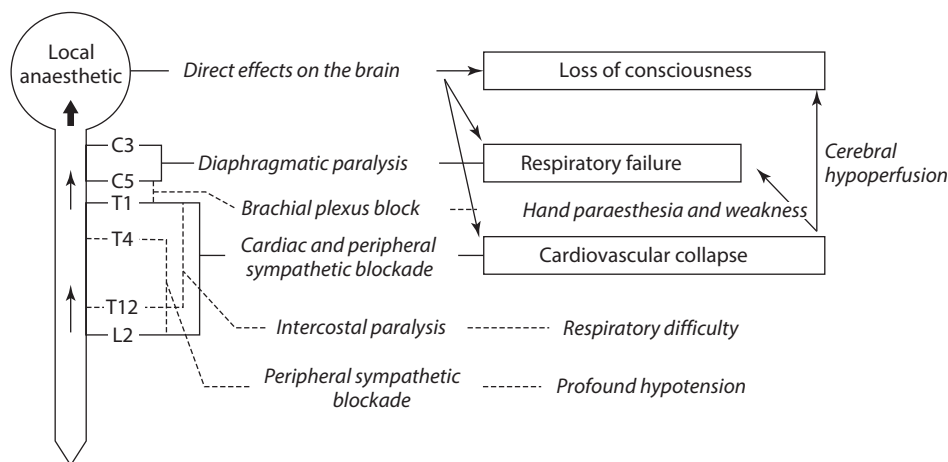
Figure 30.9 Physiological effects of total spinal anaesthesia.

reduction in systemic vascular resistance and venous return.

- Block of cardiac sympathetic fibres (T1–T4) with unopposed vagal nerve supply results in severe bradycardia.

RESPIRATORY FAILURE

- Progressive intercostal muscles paralysis (rising from T12 to T1).
- Diaphragmatic paralysis (C3–C5).
- Inhibition of respiratory centre due to direct effect of local anaesthetic or secondary to cerebral hypoperfusion.

LOSS OF CONSCIOUSNESS

- Direct action of local anaesthetic on the brain.
- Secondary to cerebral hypoperfusion due to severe hypotension.

AETIOLOGY

INTENTIONAL

- Intentional TSA with respiratory and cardiovascular support, has been used in chronic pain management to provide transient relief of intractable pain. The reported regimen is 20–30 mL of 1.5% lidocaine.

ACCIDENTAL

TSA occurs as a complication of neuraxial or peripheral nerve block.

- Neuraxial block:
  - Aspiration of an epidural catheter and test-dose administration can be falsely negative.
  - Increased risk of TSA when topping-up an epidural following dural tap during insertion.
  - Inadvertent subdural placement can result in unpredictable block.
  - Catheter migration or multi-compartment block in which a multi-hole catheter straddles the epidural, subdural and subarachnoid spaces, can lead to delayed collapse following previously normal top-ups.
  - Caudal epidurals are theoretically susceptible to all of the above problems.
  - Spinal anaesthesia following recent epidural top-up may result in an unpredictably high block as the volume of the space is reduced by compression due to fluid in the epidural space.
- Peripheral block. Local anaesthetic spreads to the subarachnoid space along the radicular dural cuff or via the perineural space in case of intraneural injection. Cases have been reported after:
  - Retrobulbar block
  - Cervical plexus block

- Stellate ganglion block
- Brachial plexus block (interscalene approach)
- Intercostal block.
- Paravertebral block
- Lumbar plexus block

PREVENTION

- Attention to detail when performing blocks close to the spinal cord.
- Factors which may lead to a high block during spinal anaesthesia are
  - Larger volumes and doses of local anaesthetic
  - Head-down patient position
  - Higher level of injection
  - Pregnancy (engorgement of epidural veins)
- Precautions that should be taken when using an epidural catheter are
  - Aspirate without a filter following catheter insertion.
  - Aspirate before each top-up.
  - Use a test dose sufficient to produce a reliable subarachnoid block, e.g. 10 mg bupivacaine.
  - Test for S1 motor block 5 minutes after test dose.
  - Titrate epidural local anaesthetic in incremental doses.
  - It is essential to assess frequently both sensory and motor block.
- Precautions to be taken with other blocks are
  - Use the shortest practicable needle.
  - Maintain an in-plane view of the needle with ultrasound guidance.
  - Careful aspiration prior to injection.

EARLY RECOGNITION AND TREATMENT OF POTENTIAL TSA

- Resuscitation equipment and anaesthetic assistance should be available before any regional block.
- Signs and symptoms:
  - Respiratory difficulty (weak voice, inability to cough).

- Upper limb paraesthesia and weakness.
- Horner's syndrome.
- Cerebral hypoperfusion (restlessness, nausea, vomiting, headache).
- Management directed at restricting spread of local anaesthetic:
  - Hyperbaric solution (spinal), reverse Trendelenberg.
  - Solutions intended for non-subarachnoid use are likely to be isobaric or hypobaric within CSF; the patient should be kept still; a slight head-down tilt will encourage caudal spread of hypobaric local anaesthetic and help to maintain venous return.
  - Obstetric patients should be managed in a left lateral tilt to avoid aortocaval compression.

TREATMENT

- Adopt an ABC approach.
- Stop the injection/infusion if ongoing.
- Cardio-respiratory support until the effects recede.
- Give 100% oxygen.
- Tracheal intubation and assisted ventilation – may require general anaesthesia, sedation or muscular relaxation.
- Cardiovascular support:
  - Maintain venous return – elevation of legs; left lateral tilt (obstetric patient); intravenous fluid resuscitation.
  - Give atropine and vasopressor drugs if required.

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CROSS-REFERENCES

Spinal epidural and CSE, Chapter 29
Local anaesthetic toxicity, Chapter 30
Cardiopulmonary resuscitation, Chapter 30

TRANSPORT OF THE CRITICALLY ILL

Lack of an available ICU bed is statistically the most common reason for transferring critically ill patients. The second is referral for specialist services.

The demand for critical care services has constantly outstripped the supply. As the demand increases relentlessly critically ill patients are increasingly being transferred from one hospital to another. This situation is particularly clear in the UK where there are fewer ICU beds per person compared with many other European countries.

Transfers of the critically ill are not benign. Adverse events occur in between 4 and 70% of transfers. These are predominantly related to equipment failure, but approximately one-third involve physiological deterioration. Up to 91% of these incidents may be preventable, highlighting the need for thorough preparation and anticipation of potential problems. A series of regional bed bureaux in the UK coordinate bed availability. These bed services operate systems that are based on daily telephone surveys of ICU beds.

In the UK, local and regional planning for ICU transfers is mandatory. This has led to the development of critical care networks which are responsible for, amongst other things, the development of transfer services within defined groups of regional hospitals.

Each network has a lead clinician and manager who are involved in transfer process mapping, protocols and quality assurance programs. They are also responsible for the availability of appropriate equipment, training and resources to allow the safe and coordinated transfer of the critically ill.

PRACTICAL CONDUCT OF TRANSFERS

ORGANISATION

Transfers for capacity reasons should be kept to a minimum. It is often preferable to establish a temporary ICU area elsewhere in the hospital while capacity is generated on the ICU. When a transfer is undertaken, it should be to an appropriate local hospital within the designated network.

There must be a consultant in an acute speciality responsible for transfers 24 hours a day, usually the ICU consultant. Each patient should be accompanied by two healthcare professionals. One should be medically qualified and have relevant experience and competencies in transport medicine. They should be able to reintubate and resuscitate the patient and be competent in the use of the transfer equipment.

Dedicated retrieval teams have been shown to improve patient outcomes in terms of physiological parameters. Whether these services are utilised depends on local arrangements, though in the paediatric setting in the UK this represents the agreed standard of care. If a retrieval team is unavoidably delayed, or in the case of a time-critical emergency (e.g. intracranial haemorrhage), a local transfer team should be used instead.

VEHICLES

Road transport has many advantages. Ambulances with trained paramedic staff are easy and quick to mobilise and are rarely affected by weather. Dedicated retrieval ambulances have many potential advantages including standardisation of gas and power supplies and installation of permanent equipment. However, substantial workload has to exist to justify such a resource.

There are some situations when air transport may be preferable. The obvious advantage of speed over long distances must be balanced against other considerations. Aeromedical transfers by either helicopter or fixed wing aircraft have the following disadvantages:

- Organisational delays.
- Transfer considerations at either end of the journey.

- Staff involved must have previous aeromedical training.
- An unfamiliar, noisy and often cramped environment may make monitoring and assessing the patient challenging.
- A fall in atmospheric pressure may lead to an increased FiO_2 requirement. Any gas-containing cavity will tend to expand, e.g. pneumothorax, pneumoperitoneum, intracranial air.
- Altitude is also accompanied by an increased risk of hypothermia.

MONITORING

Minimal monitoring standards for transfers as defined by the intensive care society are

- Continuous ECG.
- Noninvasive blood pressure (often unreliable in a moving vehicle and an arterial line should generally be used).
- Oxygen saturation.
- End tidal carbon dioxide in ventilated patients.
- Temperature, core or peripheral.
- Presence of appropriately trained staff.

PREPARATION

- Resuscitation of the patient should be complete before the start of the transfer in most cases. If it is not, the risk–benefit ratio of the transfer should be re-examined.
- Persistent hypotension after resuscitation should trigger a further search for continuing blood loss and/or systemic inflammatory response syndrome.
- If there is any doubt regarding airway patency, tracheal intubation should be carried out prior to transfer.
- Generally, all ventilated patients should be paralysed and stabilised on the transfer ventilator prior to departure.
- Chest drains should not be clamped. Leaflet valves can, if necessary, replace underwater seals.
- At least two IV cannulae should be sited and secured thoroughly.

- All long bone fractures should be splinted. This will reduce blood loss, pain and risk of nerve damage.
- Patients must be safely secured to the transfer trolley (e.g. with a 5-point harness) and equipment should be secured so it does not become a missile in the case of rapid deceleration.
- Hypothermia is common on transfers. Ensure that the patient is appropriately insulated.

HANDOVER AND DOCUMENTATION

In many circumstances the transfer personnel have not been involved in the initial treatment of the patient. It is imperative that they make an independent assessment of the patient's condition prior to the transfer. This will include collecting and reviewing relevant X-rays, scans and laboratory results. Formal handovers between medical and nursing staff should occur prior to departure and on arrival at the receiving hospital. Particular care should be taken to avoid the erroneous administration of blood products during or following the transfer.

Transfer documentation may be standardised within networks or regions. The information recorded should include: patient's diagnosis, reason for transfer, responsible consultants, vital signs, treatment modalities used and drugs given during the transfer.

SAFETY OF PERSONNEL AND INSURANCE

Motor vehicle collisions involving ambulances are rare but not unknown. Risks can be minimised by avoiding high-speed driving, which is unnecessary in the majority of cases, ensuring that safety-belts are worn and equipment is appropriately secured. In the event of injury or death to transfer personnel, the insurance situation is unclear. NHS Trusts would provide employer's liability benefits but it is possible that negligence on the part of the employer may have to be proven first. In the UK, AAGBI or Intensive Care Society membership provides automatic and fully comprehensive transfer insurance. In other European countries, the situation should be checked in advance of the possible need to go on a transfer.

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CROSS-REFERENCE

Trauma, Chapter 22

TRANSURETHRAL RESECTION OF THE PROSTATE SYNDROME

The profound alteration in the functioning of the cardiovascular and nervous systems produced by absorption of large volumes of electrolyte-free irriga-tion fluid during transurethral resection of the pros-tate (TURP) is described as the TURP syndrome. It has also been described in connection with percuta-neous ultrasonic lithotripsy, vesical ultrasonic litho-tripsy and intrauterine endoscopy.

Fluid absorption occurs principally through the venous sinuses of the prostatic capsule. Distilled water was used as the irrigation fluid but the high incidence of haemolysis, with its ensuing compli-cations, limits its suitability. Irrigation by non-electrolyte solutions is necessary in order to reduce dispersion of current during electrocautery. Glycine 1.5% solution with an osmolarity of 220 mmol L^{-1}, is the most widely used irrigant for TURP and undergoes both renal excretion and metabolism to ammonia by the liver.

PATHOPHYSIOLOGY

Irrigation fluid is absorbed at 15–30 mL min^{-1} during TURP surgery. Excessive fluid absorption into the circulation results in fluid overload and dilution of both proteins and electrolytes. Reduction of oncotic pressure promotes fluid shifts from the vascular compartment into the interstitial compartments pro-ducing oedema in tissue beds.

Hyponatraemia is induced by dilution and natriuresis. The low serum sodium seen in TURP syndrome is often not associated with a change in serum osmolality. However, patients with both low sodium and low serum osmolality are more likely to be symptomatic as a reduction in plasma osmo-lality overwhelms compensatory mechanisms and cerebral oedema rapidly develops. A very low serum sodium (<120 mmol L^{-1}) is associated with more severe symptoms and a poorer prognosis

Transient elevation of the serum potassium has been observed in the absence of haemolysis and hyperkalaemia may be implicated in the produc-tion of cardiac arrest during uptake of irrigating fluid.

Glycine is an inhibitory neurotransmitter, and its metabolite ammonia is neurotoxic. High con-centrations of these substances in the CNS lead to neurological sequalae including confusion, coma or visual disturbances. These features may be com-pounded by the effects of hyponatraemia and cere-bral oedema.

In severe TURP syndrome, renal function may be impaired due to acute tubular necrosis, which may be the result of the reduction in renal blood flow pro-duced by hypotension or by renal swelling.

SIGNS AND SYMPTOMS

These are related to the volume of irrigant absorbed:

RESPIRATORY

Dyspnoea, cyanosis, pulmonary oedema.

CARDIOVASCULAR

Initial rise in blood pressure (hypervolaemia). If plasma sodium falls below 120 mmol L^{-1}, heart rate

falls and profound bradycardia may occur. Reduced myocardial contractility produces ECG abnormalities including loss of P waves, nodal rhythm, ventricular tachycardia, widened QRS complexes, depression of ST segments and T wave inversion. Chest pain may be reported by the conscious patient.

CNS

Under spinal or epidural blockade, the first signs of excess absorption are disorientation and restlessness, often preceded by severe apprehension. Other CNS effects include reduction of consciousness and grand mal seizures. Transient blindness has been described and has been attributed to a direct effect of glycine on ocular retinal potentials.

MANAGEMENT

- Adopt the ABC approach. Give 100% oxygen, intubate and ventilate if necessary.
- Conclude surgery as soon as is feasible and irrigate bladder with warm normal saline.
- Send blood for sodium measurement. Baseline laboratory investigations should include full blood count, electrolytes, arterial blood gases and clotting screen.
- Administer an intravenous diuretic (e.g. furosemide in initial dosage of 20 mg) together with an infusion of normal saline. As a diuresis commences, maintain fluid balance with normal saline. Further administration of diuretic should be based on assessment of initial diuresis.
- In most centres, administration of hypertonic saline (e.g. 3% NaCl) is restricted to the small group of patients demonstrating severe symptoms, e.g. seizures or severe cardiac dysfunction as a result of electrolyte imbalance. Infusion of 3% saline must be undertaken with caution, via a large proximal vein (or central line) and with appropriate cardiovascular monitoring. The aim is to increase the serum sodium by no more than 1 mmol L^{-1} h^{-1} in order to avoid further fluid overload or neurological complications.

- During the diuresis, the serum potassium should be monitored as hypokalaemia frequently occurs during this phase.

PREVENTION OF TURP SYNDROME

- Regional anaesthesia without sedation allows early detection of neurological symptoms.
- Appropriate preoperative preparation with monitoring of urea and electrolytes. Adequate hydration prior to surgery particularly as hypovolaemia reduces venous pressure allowing more irrigant to be absorbed.
- Limitation the duration of the surgical procedure. Where possible, resection time should be restricted to less than 1 hour.
- Do not place the bag of irrigation fluid higher than 70 cm above the patient.
- Keep bladder distension to a minimum with frequent bladder drainage.
- Some centres add 1% ethanol to the irrigation fluid and monitor expired ethanol.
- Use a technique that avoids diathermy (e.g. laser TURP). Irrigation fluids with physiological concentrations of electrolytes can be used for these cases.

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